As a Rare Reason of Alveolar Consolidation, Negative Pressure Pulmonary Edema: Case Report

Bu yazıda negatif basınçlı akciğer ödemi (NPPE) olan iki olgu tanımlanmıştır. Tikalı bir hava yoluna karşı, aşırı negatif hava yolu basıncının alveollerde ve akciğerde ödeme neden olduğu bildirilmiştir. Hastalar genellikle herhangi bir tıbbi problemi olmayan genç yetişkinlerdir. Bu durum, her iki akciğerin santral interstisyel bölgesini içeren ve nonnvasive mekanik ventilasyon ve kortikosteroidlerle tedavi edilir.

Anahtar kelimeler: Akciğer ödemi, bilgisayarlı tomografi, alveolar konsolidasyon

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INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a rare reason of acute respiratory failure and alveolar consolidation. In patients with NPPE, there are patchy non-segmental alveolar consolidations in central distribution on CT. In alveolar pattern, filling of the alveolar spaces with pus, edema, hemorrhage, inflammation or tumor cells are seen. NPPE is one of the illnesses which induces an alveolar pattern. In this paper, we aimed to present two patients who developed acute respiratory failure due to NPPE after general anesthesia.

CASE 1

A 25-year-old man was admitted to our hospital due to perianal abscess and operated. His BMI was 27 kg/m\(^2\) and he had ASA II score due to anemia. Preoperatively, his chest examination results were normal, and a chest radiograph indicated clear lung fields. Standard monitorization of the patient was performed with electrocardiogram, noninvasive arterial blood pressure measurement and pulse oximetry. According to anesthesia induction protocol, 1.5 μg/kg fentanyl, 2 mg/kg propofol and 1 mg/kg rocuronium bromide were used after satisfactory muscular relaxation an orotracheal intubation was performed with an 8-mm-thick cuffed tube. The patient was ventilated with a 550 ml tidal volume in order to ensure 14 respirations per minute in a volume control mode. The respiratory parameters were set in order to have a CO\(_2\) end-tidal level between 35-40 mmHg. Anesthesia was maintained by providing fresh gas of 1-2% minimally alveolar consolidation (MAC) sevoflurane and 50% O\(_2\)-air with 3 L/min. The surgery lasted for about 60 minutes. In the operation, 800 mL crystalloid (0.9% NaCl) infusion was made. The procedure was terminated without any complications. When enough respiratory depth was reached, severe agitation, laryngospasm and inspiratory effort developed following extubation. Negative pressure pulmonary edema was suspected due to the presence of foamy, pink colored, bloody secretion, auscultating common rales in both lungs, and oxygen-free saturation values of 86 percent. The patient was given oxygen and taken to intensive care unit for postoperative follow-up. In the first blood gas sample taken in the intensive care unit, pH was measured as 7.32, pO\(_2\): 53 mmHg, pCO\(_2\): 33 mmHg, HCO\(_3^-\): 18 mmol/L Lactate: 1.2 mmol/L. The patient did not need intubation and we performed NIMV with pressure mode and PEEP (positive end-expiratory pressure). In computed tomography of the patient, there were ground-glass opacifications and alveolar consolidations which were denser in the central interstitial area of both lungs. Noninvasive

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**Figure 1.** A 25-year-old man with NPPE. In computed tomography alveolar consolidations which were denser in the central interstitial area of both lungs are seen (A). Following the treatment, decreased consolidations are seen (B).

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mechanical ventilation and 8 mg dexamethasone and 20 mg furosemide were given to the patient taken to intensive care unit. Following the treatments, both symptomatic and radiological improvement was seen (Figure 1).

CASE 2

A 28-year-old, obese (BMI: 35.6), ASA score II man was admitted to hospital due to cholelithiasis and operated. Preoperatively, his chest examination results were normal, and a chest radiogram indicated clear lung fields. The patient was ventilated with a 500 ml tidal volume in order to maintain 15 respirations per minute in a volume control mode. The operation lasted for about 90 minutes. In the operation, 1200 mL crystalloid (0.9% NaCl) infusion was made. The procedure was concluded without any complications. Following extubation, severe agitation, bronchospasm and inspiratory effort developed in the patient. In recovery room, negative pressure pulmonary edema was suspected due to auscultation of common rales in both lungs and with 2-4 L/min oxygen saturation values of 91% at postoperative tenth minutes. In the first blood gas sample pH: 7.31, pO$_2$: 52 mmHg, pCO$_2$: 31 mmHg, HCO$_3$: 19 mmol/L Lactate: 1.5 mmol/L in recovery room. Together with oxygen support, non-contrast computed tomography was obtained. There were ground-glass opacifications and alveolar consolidations which were denser in the central interstitial area of both lungs (Figure 2). The patient was given oxygen and taken to intensive care unit for postoperative follow-up. Noninvasive mechanical ventilation and 8 mg dexamethasone and 20 mg furosemide treatments were given to the patient taken to the intensive care unit. Following the treatments, both symptomatic and radiological improvement was seen. The patient did not need intubation and we applied NIMV for positive pressure ventilation strategy. The patient was discharged from the intensive care unit after two days of intermittent NIMV application.

DISCUSSION

Although NPPE is a rare complication with a rate of 0.094% in general anesthesia protocol, this rate increases over 11% in patients with postoperative acute upper airway obstruction. In its pathogenesis, it has been reported that intense inspiratory effort against an obstructed airway leads to pulmonary edema (Figure 3)$^{1,2}$. The range of normal inspiratory pleural pressure varies between (-2) to (-5) cmH$_2$O; however, inspiratory pleural nega-

Figure 2. A 28-year-old man with NPPE. Alveolar consolidations are prominent in the central interstitial area of both lungs.

Figure 3. Intense inspiratory effort against an obstructed airway causes pulmonary edema.
tive pressure may increase up to about 100 cm-
H₂O during the difficult inspiratory effort against
obstruction. Venous return to right heart in-
creases, left ventricular output decreases, right
ventricular dilatation, shifting to the left in the
interventricular septum and left ventricular dia-
stolic dysfunction develop owing to the increased
pressure. It has been reported that, in this case,
microvascular circulation fails, capillary insuf-
iciency develops, alveolar fluid flow increases,
and, eventually pulmonary edema develops. Adrenergic discharge, depending on anxiety with
hypoxia, makes the case even worse. In its eti-
ology, croup, epiglottitis, previous oropharynge-
al surgery, short neck, obesity, foreign materials,
tumors, non-viscous tracheal secretions are also
taken into consideration.

However, the most frequently known cause of
NPPE in adults is laryngospasm after extubation. It
has been reported that NPPE is more common
in healthy young adults who have well-developed
inspiratory muscles and can generate this pressure
easily. We think that our cases were also healthy
young patients, post-extubation laryngospasm and hypoxia contributed to this situation.

Cascade et al. reported that NPPE has to be sus-
ppected postoperatively in young patients with
bilateral central pulmonary edema, increased vas-
cularity, and normal cardiothoracic ratio. For these
patients, it is important to make a proper differ-
ential diagnosis among massive fluid transfu-
sion, cardiogenic pulmonary edema, anaphylaxis,
neurogenic pulmonary edema, and acute respira-
ory distress syndrome in terms of treatment
choice. There was no history of allergy in our
patients. Postoperative cardiovascular system ex-
aminations were normal. Since posterior anterior
(PA) chest X-ray revealed bilateral central inter-
stitial infiltrations and normal cardiothoracic ratio,
there were alveolar type consolidations in upper
lobes of bilateral lung parenchyma, lack of mas-
vie liquid transfusion during surgery and a rapid
recovery pattern with corticosteroid treatment
confirmed the diagnosis of NPPE. The treatment is
the breakage of the vicious cycle by correcting of
hypoxia. Proper diagnosis provides the choice
of appropriate treatment and rapid recovery.

In conclusion, NPPE is a rare complication of
general anesthesia affecting young adults. In patients
with sudden onset of respiratory distress in the
postoperative period, alveolar consolidations,
which are more prominent in the upper lobes of
both lungs, should suggest the diagnosis of NPPE
in order to select the appropriate treatment.

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