Negative Pressure Pulmonary Edema: Early Diagnosis ve Early Treatment

Negatif Basınçlı Akciğer Ödemi: Erken Tanı ve Erken Tedavi

Hüseyin Çetik, Gönül Ölmez Kavak, Zeynep Baysal Yıldırım, Mahir Kuyumcu

Abstract

Negative pressure pulmonary edema is a rare complication following general anesthesia, potentially requiring intensive care monitoring and mechanical ventilatory support. Prompt clinical suspicion and early diagnosis, along with appropriate management and respiratory support, can support rapid and complete recovery with favorable outcomes. Particular care should be taken in young, adult and male patients who develop laryngospasm during extubation, including close monitoring, and negative pressure pulmonary edema should be considered in the differential diagnosis if pulmonary edema develops. We present here the diagnostic, monitoring and therapeutic approach applied to a young male patient who developed negative pressure pulmonary edema following extubation after undergoing appendectomy.

Keywords: *Negative Pressure, Pulmonary Edema, Mechanical Ventilation, Laryngospasm, Appendectomyac.*

Öz

Negatif basınçlı akciğer ödemi, genel anestezi sonrasında görülebilen nadir bir komplikasyon olup, yoğun bakım izlemi ve mekanik ventilasyon desteğini gerektirebilen bir akciğer ödemi tablosudur. Klinik şüphe duyup erken teşhis edilmesi, doğru yönetim ve solunum desteği tedavisi, hızlı ve tam iyileşme sağlar ve sonuçlar yüz güldürücüdür. Özellikle ekstübasyon işlemi sırasında laringospazm gelişen genç, erişkin ve erkek hastalarda dikkatli olunmalı, yakın takip edilmeli ve akciğer ödemi bulguları gelişirse ayırıcı tanıda mutlaka negatif basınçlı akciğer ödemi tanısının akılda tutulması gerekmektedir. Bu olgumuzda apendektomi operasyonu geçiren genç, erkek hastada, ekstübasyon sonrası gelişen negatif basınçlı akciğer ödeminin tanı, takip ve tedavi yaklaşımları sunulmuştur.

Anahtar Kelimeler: *Negatif Basınç, Akciğer Ödemi, Mekanik Ventilasyon, Laringospazm, Apendektomi.*

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Negative pressure pulmonary edema (NPPE) is an uncommon complication of general anesthesia that can develop after extubation or in the postoperative period, and that may necessitate intensive care monitoring and mechanical ventilatory support. The reported incidence of NPPE in anesthesia practice ranges between 0.05% and 0.1%, although it has been suggested that the condition develops more frequently than documented (1). Although rare, its clinical manifestations are striking, and early recognition followed by immediate initiation of treatment can prevent significant morbidity and mortality associated with NPPE. Clinicians should thus be familiar with the condition, promptly recognize it and avoid delays in management (1,2). We present this case report to the literature to serve as guidance to colleagues in the management of this rare complication. For this purpose, the evaluation, monitoring and treatment course of a patient who developed NPPE are presented.

CASE

A 29-year-old male patient weighing 75 kg and 180 cm in height was scheduled for surgery in the general surgery department with a diagnosis of acute appendicitis. The patient had no known comorbidities or history of allergies, and laboratory results and chest radiography were within normal limits (Figure 1). The patient had no history of smoking or alcohol consumption and was classified as ASA Physical Status 1E before surgery. Standard ASA monitoring was conducted. After an 8-hour fasting period, anesthesia induction was performed with intravenous midazolam (0.05 mg/kg), propofol (2 mg/kg), lidocaine (1 mg/kg), fentanyl (2 μg/kg) and rocuronium (0.6 mg/kg).

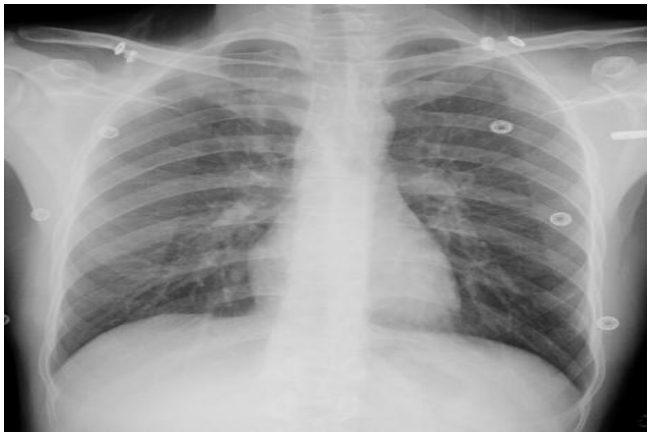

Figure 1: Preoperative chest radiograph of the case

Orotracheal intubation was achieved with a 7.5 mm cuffed endotracheal tube. Anesthesia was maintained with sevoflurane (1–2%) in a mixture of 50% air and 50% oxygen at a fresh gas flow rate of 2 L/min. The surgical procedure lasted approximately 100 minutes, during which no respiratory or hemodynamic instability occurred. The patient received 1000 mL of crystalloid fluid intraoperatively. At the conclusion of the surgery, the neuromuscular blockade was reversed with sugammadex (200 mg). Extubation was performed once spontaneous breathing and response to verbal stimuli were confirmed. Five minutes after being transferred to the postoperative recovery room, the patient developed inspiratory effort, agitation and laryngospasm. Abdominal breathing and intercostal retractions were observed, and oxygen saturation decreased to 60%. Airway maneuvers and mask ventilation with 100% oxygen were applied, improving saturation to 85%. Electrocardiography (ECG), arterial blood gas analysis and other laboratory investigations were obtained. The ECG showed only mild sinus tachycardia, while the arterial blood gas analysis revealed pH: 7.24, pO_2: 58.1 mmHg, pCO_2: 54.6 mmHg and SpO_2: 78.9%. Biochemistry, complete blood count, cardiac markers, D-dimer and pro-BNP were within normal limits. The patient produced several episodes of pink, frothy sputum, and bilateral diffuse rale was auscultated, and was administered intravenous methylprednisolone (80 mg), pantoprazole (40 mg) and furosemide (20 mg). Echocardiography showed normal cardiac function and chamber dimensions. The patient was subsequently transferred to the intensive care unit due to persistent dyspnea and tachypnea. In the ICU, noninvasive blood pressure was 118/65 mmHg, heart rate 106 bpm and oxygen saturation 87%. Chest radiography revealed bilateral infiltrates consistent with pulmonary edema, and chest computed tomography revealed diffuse consolidations, septal thickening and ground-glass opacities. Considering the development of respiratory distress after laryngospasm, the absence of pre-existing cardiac disease, bilateral crackles on auscultation, laboratory findings and the presence of pink, frothy sputum, the patient was diagnosed with NPPE and the appropriate treatment was initiated (Figures 2 and 3).

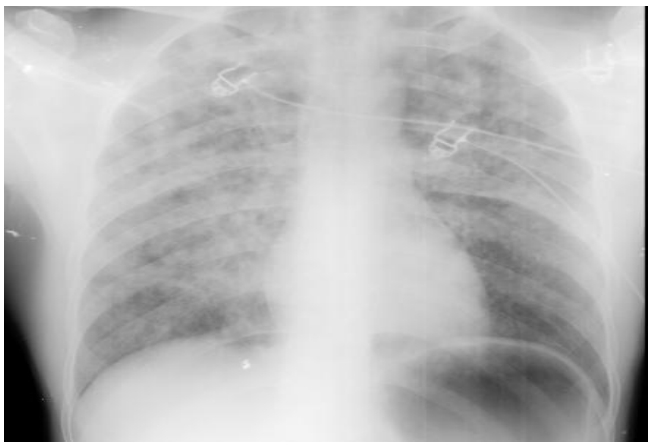

Figure 2: Postoperative chest radiograph showing infiltrative areas consistent with bilateral pulmonary edema

Fluid restriction was applied, and limited intravenous fluid administration, salbutamol and budesonide nebulization were provided. Noninvasive mechanical ventilation (NIMV) was started with an oronasal mask in CPAP mode, with PEEP 10 cm H_2O, pressure support 15 cm H_2O and FiO_2 60%, and after one hour of NIMV, clinical improvement and resolution of hypoxemia were observed, with SpO_2 rising to 95% on 6 L/min oxygen. The results of an arterial blood gas analysis at the fourth hour were pH: 7.27, pO_2: 92.8 mmHg, pCO_2: 49.1 mmHg and SpO_2: 93.9%. Oxygen therapy was continued via a nasal cannula. The tachypnea and dyspnea regressed, and a follow-up blood gas analysis at 12 hours showed pH: 7.39, pO_2: 181 mmHg, pCO_2: 40.5 mmHg and SpO_2: 99.4%. Chest radiography confirmed the resolution of the pulmonary edema findings (Figure 4). After 24 hours of monitoring and treatment, the patient no longer required supplemental oxygen and remained stable in room air. Chest radiography on postoperative day 2 revealed a marked regression of pulmonary edema (Figure 5). The patient, who had been monitored and treated in the ICU for one day, was transferred to the general surgery ward and discharged in good condition after an additional day of observation. The blood gas analyses and hemodynamic results recorded during the process are presented in Table 1.

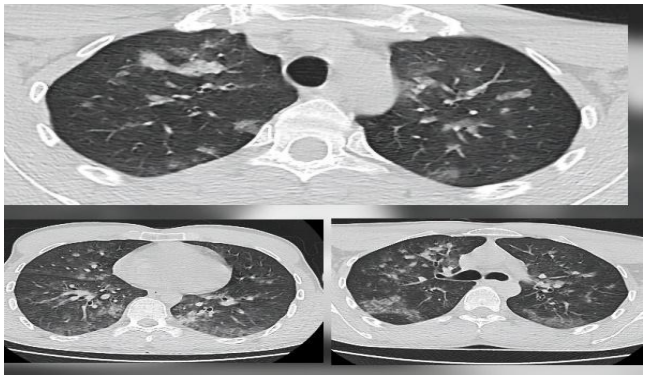

Figure 3: Thoracic computed tomography revealing ground-glass opacities and areas of consolidation

DISCUSSION

The incidence of NPPE is higher among male patients and those with ASA physical status I or II (3), which is likely attributable to the ability of otherwise healthy adults to generate markedly elevated intrathoracic negative pressures. Other risk factors for NPPE include procedures involving the upper airway, obesity, smoking, short neck anatomy, difficult intubation, upper respiratory tract infection and obstructive sleep apnea (4). In the presented case, it is thought that the healthy status and young age of the patient, and the development of laryngospasm following extubation contributed to the onset of NPPE. During forceful inspiration, the increase in negative intrathoracic pressure augments venous return to the right heart and elevates pulmonary venous pressure. This disrupts pulmonary microvascular circulation, and the resulting increase in pulmonary capillary permeability allows transudation of fluid from the pulmonary capillaries into the alveolar spaces, culminating in pulmonary edema. Pulmonary perfusion is further compromised by fluid accumulation, leading to hypoxemia. Hypoxemia and acidosis due to inadequate ventilation exacerbate pulmonary vascular resistance via alveolar-capillary membrane injury, which intensifies the hyperadrenergic response and may result in diffuse alveolar hemorrhage as well as cardiac complications (2,5). While the symptoms of NPPE typically manifest immediately after extubation, they may develop at any time during the postoperative period. Clinically, initial manifestations include oxygen desaturation, pink frothy sputum production and radiographic abnormalities consistent with pulmonary edema, all of which evolve in a characteristic sequence. The occurrence of laryngospasm during extubation, followed by respiratory distress and hypoxemia, strongly supports the diagnosis (6). Signs of laryngospasm include suprasternal retractions, stridor, use of accessory inspiratory muscles and agitation, and bilateral diffuse crackles and rhonchi may be present on lung auscultation. Radiologic findings are critical for both diagnosis and follow-up, while thoracic CT and chest radiography in NPPE patients may reveal bilateral pulmonary infiltrates and interstitial edema (1).

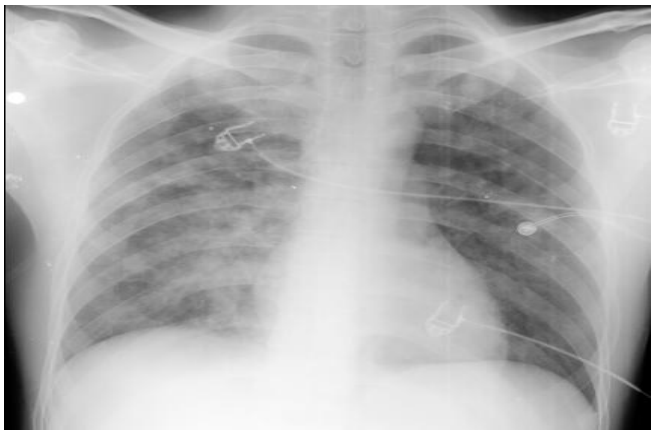

Figure 4: Chest radiograph taken approximately 12 hours after treatment showing regression in the infiltrative areas

In cases where clinical suspicion necessitates differentiation between cardiogenic and non-cardiogenic pulmonary edema, echocardiography (ECHO) may be employed for the assessment of right and left ventricular dimensions, left ventricular ejection fraction, valvular function and wall motion abnormalities (7). Differential diagnoses for NPPE include fluid overload, cardiogenic pulmonary edema, anaphylaxis and Mendelson's syndrome, and excluding these is crucial for management guidance (1,5). No evidence of anaphylaxis was

observed perioperatively in the presented case, and there was no known history of allergy. The use of a cuffed endotracheal tube, absence of vomiting during or after extubation, and lack of perioperative oxygen desaturation ruled out Mendelson's syndrome, and the absence of underlying cardiac disease, the normal postoperative ECG and echocardiography results, and the timing of pulmonary findings after laryngospasm argued against cardiogenic pulmonary edema. Furthermore, the administration of 1000 mL of isotonic solution during surgery was not considered sufficient to cause fluid overload. The primary goal in NPPE management is to ensure adequate oxygenation and airway patency, with early application of positive pressure ventilation when necessary. In mild cases, oxygen supplementation via a face mask may be sufficient (8). However, if there is no clinical or oxygenation improvement, non-invasive mechanical ventilation (NIMV) should be initiated. NIMV reduces venous return and pulmonary preload, thereby limiting the progression of pulmonary edema, and if alveolar fluid is already present, it facilitates its redistribution into the interstitial space. In most cases, NPPE resolves with NIMV without the need for invasive mechanical ventilation (3,7). Nonetheless, there are reports in the literature of cases requiring reintubation and invasive ventilation (3,4). In the presented case, oxygen supplementation via a face mask was insufficient, and the hypoxemia was dramatically corrected following NIMV. The available pharmacologic treatments include diuretics, although their use remains controversial (7). Even in the absence of fluid overload in the pathophysiology, diuretics may be administered to enhance alveolar fluid clearance with careful monitoring of urine output, electrolytes and hemodynamics, for which a few favorable results have been reported (9). While the use of steroids remains a subject of debate, Chuang et al. (10) reports that steroids may be beneficial in cases of alveolar injury, reducing respiratory distress and accelerating recovery. Beta-2 agonists have also been recommended, as bronchodilator therapy may alleviate pulmonary edema symptoms (7). The presented case was administered diuretics, steroids and beta-2 agonists, leading to clinical improvement. In conclusion, NPPE is a rare complication of general anesthesia, for which prompt clinical suspicion, appropriate management and timely respiratory support typically ensure rapid and complete recovery and favorable outcomes. Particular caution should be exercised in young, healthy male patients who develop laryngospasm during extubation as a higher risk group. Such patients should be closely monitored, and NPPE should be considered in the differential diagnosis when pulmonary edema findings emerge.

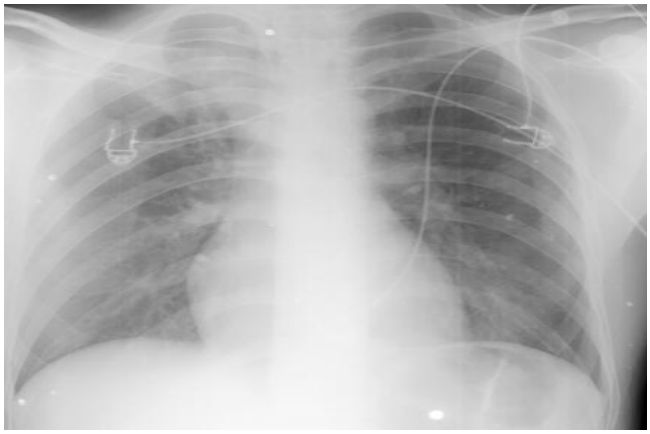

Figure 5: Chest radiograph prior to discharge

Table 1: Blood gas and hemodynamic values of the patient during the treatment period

Parameter	Before extubation	After extubation	After extubation 5th minute	Upon ICU admission	ICU 4th hour	ICU 12th hour
BP (mmHg)	124/65	126/70	133/76	118/65	124/69	121/67
HR (beats/min)	76	85	115	106	87	70
SpO_2 (%)	99	97	60	91	94	99
pH			7.24		7.27	7.39
pO2 (mmHg)			58.1		92.8	181
pCO2 (mmHg)			54.6		49.1	40.5
Lactate ($mmol \cdot L^{-1}$)			2.2		1.5	2
BG-SpO_2 (%)			78.9		93.9	99.4

BP: Blood pressure, HR: Heart rate, SpO_2: Peripheral oxygen saturation, pO_2: Partial pressure of oxygen, pCO_2: Partial pressure of carbon dioxide, BG-SpO_2: Blood gas oxygen saturation, ICU: Intensive Care Unit

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - H.Ç.; Planning and Design - H.Ç., Z.B.Y., G.Ö.K.; Supervision - H.Ç., Z.B.Y.; Funding - ; Materials - ; Data Collection and/or Processing - H.Ç., Z.B.Y., G.Ö.K.; Analysis and/or Interpretation - ; Literature Review - H.Ç., Z.B.Y., G.Ö.K.; Writing - H.Ç., Z.B.Y., G.Ö.K.; Critical Review - H.Ç., Z.B.Y., G.Ö.K.

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