Case Report/ Olgu Sunumu

# Late period negative-pressure pulmonary edema after laparoscopic appendectomy in a young patient

## Genç bir hastada laparoskopik apendektomi sonrası geç dönem negatif basınçlı akciğer ödemi

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**ABSTRACT**

This case report aimed to present the diagnosis and treatment process of a young patient who developed negative-pressure pulmonary edema (NPPE) in the late period of laparoscopic appendectomy. A 25-year-old man was admitted with abdominal pain, vomiting, and a lack of appetite. An emergency appendectomy due to a diagnosis of acute appendicitis was performed via laparoscopy. The operation, which lasted one hour, was completed without any complications. In the 3rd hour of the patient's ward follow-up, respiratory distress started, and the saturation values decreased to 60% in room air. The computed tomography scan revealed bilateral infiltration areas at diffuse ground glass density. The patient was followed up at the intensive care unit (ICU) with the diagnosis of NPPE. Non-Invasive-Mechanic-Ventilation (NIMV) was started with steroid and furosemide treatment. After treatment, dramatic improvement was observed on the patient's chest radiograph after 24 hours. NIMV was applied 8 hours/day, and the patient was treated for two days at ICU. The patient, whose vital signs were stable and whose chest X-ray was normal, was discharged on the 5th postoperative day.

Keywords: Appendectomies, edema, non-invasive ventilation, pulmonary.

**ÖZ**

Bu olgu sunumunda laparoskopik apendektomi sonrası geç dönemde negatif basınçlı akciğer ödemi (NBAÖ) gelişen genç bir hastanın tanı ve tedavi sürecini sunmayı amaçladık. 25 yaşında erkek hasta karın ağrısı, kusma ve iştahsızlık şikayetleri ile başvurdu. Akut apandisit ön tanısıyla laparoskopi ile acil apendektomi uygulandı. Bir saat süren operasyon komplikasyonsuz olarak tamamlandı. Hastanın servis takibinin 3. saatinde solunum sıkıntısı başladı ve oda havasında satürasyon değerleri %60'a kadar düştü. Bilgisayarlı tomografi taraması, yaygın buzlu cam yoğunluğunda iki taraflı infiltrasyon alanları gösterdi. Hasta NBAÖ tanısı ile yoğun bakım ünitesinde (YBÜ) takibe alındı. Steroid ve furosemid tedavisi ile Non-İnvaziv-Mekanik-Ventilasyon (NİMV) başlandı. Tedavi sonrası 24 saat sonra hastanın akciğer grafisinde dramatik düzelme görüldü. NİMV 8 saat/gün uygulandı ve hasta iki gün yoğun bakımda tedavi edildi. Vital bulguları stabil, akciğer grafisi normal olan hasta ameliyat sonrası 5. günde taburcu edildi.

Anahtar Kelimeler: Apendektomiler, ödem, non-invaziv ventilasyon, pulmoner.

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## Introduction

Appendectomy is the most common emergency surgical procedure encountered by surgeons. In a five-year longitudinal observational study, the morbidity rate was 18.61% after an appendectomy.[1] Another study showed an overall complication rate of 8.2% to 31.4% following an appendectomy.[2] Complications discussed in the literature are surgical procedure and surgical site-related complications. However, anaesthesia-related complications can also be seen very rarely.

Negative pressure pulmonary edema (NPPE) is a rare but severe complication that can develop after acute obstruction in the upper respiratory tract.[3] In the pathophysiology of NPPE, it has been reported that forced-strong inspiratory effort due to upper airway obstruction causes an increase in intrathoracic pressure, leading to fluid passage into the pulmonary interstitial space.[4] Although the exact incidence of NPPE is unknown, and it is estimated to be up to 12% in patients who develop acute upper airway obstruction.[5] NPPE is temporary and reversible if recognised and treated in the early period. In cases where NPPEs are noticed late, the severity of desaturation increases, and hypoxia develops. Bradycardia and systolic arrest may develop due to severe hypoxia.[6]

This case report aimed to present the diagnosis and treatment process of a young patient who developed NPPE in the late period after laparoscopic appendectomy.

## Case Report

A 25-year-old man was admitted to our hospital's general surgery outpatient clinic with abdominal pain, vomiting, and a lack of appetite for about two days. Abdominal pain was first started in the peri-umbilical area and changed location to the right lower quadrant. He had a history of Behçet disease requiring colchicine treatment of 3 tablets per day. He had no other disease or surgery history. In addition, he had a history of working in a clothing workshop containing excessive dust for about two years.

On first evaluation, the vital findings of the patient were as follows: blood pressure (BP): 124/75 mm Hg, heart rate (HR): 102 beats per minute, oxygen saturation on room air: 95%, and body temperature: 37.8°Celsius. On physical examination of the abdomen, there was tenderness and rebound in the right lower quadrant. There was no laboratory pathology except for C-reactive protein (CRP) elevation (56 mg/L) and leukocyte count elevation ($17x10^3/mm^3$). Ultrasonography showed an appendix with a diameter of 12 mm. There was no pathology at preoperative chest radiography. An emergency appendectomy was performed via laparoscopy.

Preoperatively, the patient was evaluated as ASA IIE (due to smoking), and he had a common cold five days ago and tested for COVID-19 (negative). Lung sounds were normal via auscultation. In the induction of anaesthesia, 1µg $kg^{-1}$ fentanyl, 2.5 mg $kg^{-1}$ propofol and 0.6 mg $kg^{-1}$ rocuronium bromide were used. The patient was intubated with an 8.0 mm endotracheal tube without any problems. In the maintenance of anaesthesia, 2% sevoflurane was administered in a mixture of 60% medical air-40% $O_2$. The operation, which lasted one hour, was completed without any complications. After the end of the operation, anaesthesia was terminated, and the patient was extubated on the operating table. Extubation was also complication free, there wasn't any obstruction at the airway tract, and laryngospasm didn't occur. Nevertheless, the patient had some secretion. It wasn't pink or frothy. He had crackle sounds in his right lung, which was thought to be due to the common cold and smoking. He had some mild coughs, but nothing more happened to be noted. The patient was moved to the post-anaesthetic care unit. After 20 minutes, GCS:15, $SPO_2$:98% at room air, BP: 124/66 mmHg, HR:78/min, the patient was sent to the inpatient bed.

In the 3rd hour of the patient's service follow-up, respiratory distress started. The vital findings of the patient were as follows: blood pressure: 113/68 mm Hg, heart rate: 124 beats per minute, oxygen saturation on room air: 60%, and body temperature: 37.5° Celsius. There were bilateral widespread coarse crackles on listening without no bilateral pretibial edema. The computed tomography scan showed bilateral infiltration areas at diffuse ground glass density (Figure 1). Pulmonary embolism was also excluded. An incidental pulmonary artery aneurysm was also noted at CT. The patient was followed up at the intensive care unit (ICU) with the diagnosis of NPPE.

At ICU, the patient was treated with Non-Invasive-Mechanic-Ventilation (NIMV) with PEEP:8 cm $H_2O$, $FiO_2$:75%, and Psup:10 cm $H_2O$. Furosemide 40 mg intravenously and a single daily dose of 40 mg methylprednisolone sodium succinate were applied. Dramatic improvement was observed.. $SPO_2$ was 100% after 5 minutes, and respiration rate had regressed from 30/min to 17/min. A significant improvement was determined in the patient's chest radiograph after 24 hours (Figure 2). NIMV was applied 8 hours/day, and the patient was treated for two days at ICU. After two days of NIMV, the patient's vital signs were as follows: blood pressure 118/72 mm Hg, heart rate: 94 beats per minute, oxygen saturation on room air: 93-95%, and body temperature: 37.2° Celsius. There was also progressive improvement in the chest X-ray (Figure 3). Therefore, the patient was transferred back to the ward. The patient was discharged on the 5th postoperative day, as the vital signs of the patient were stable, and his chest X-ray was normal (Figure 4).

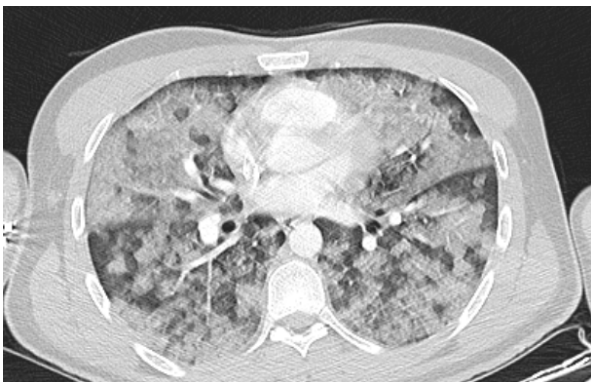

Figure 1 - A computed tomography scan showed bilateral infiltration areas at diffuse ground glass density.

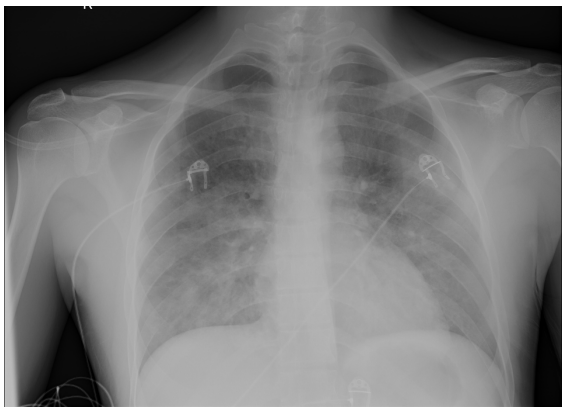

Figure 2 - Dramatic improvement in the patient's chest X-ray after 24 hours of medical treatment.

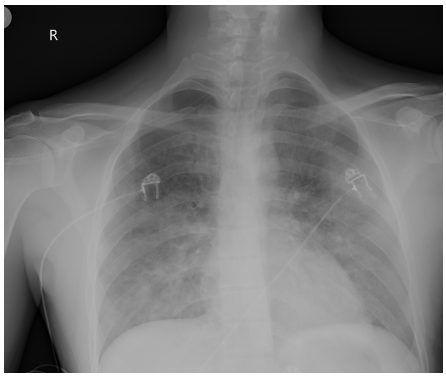


Figure 3- Chest X-ray control of the patient after two days of treatment at the intensive care unit.

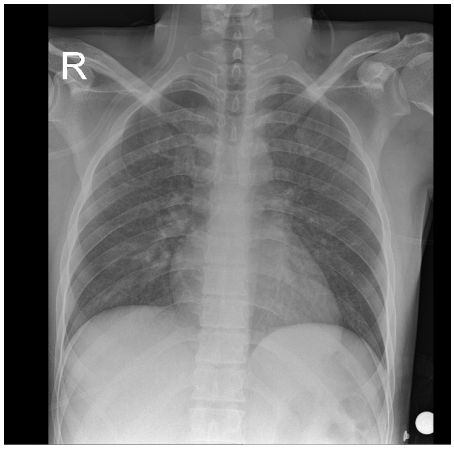


Figure 4- Chest X-ray control of the patient after two days of treatment at the intensive care unit.

## Discussion

Negative pressure pulmonary edema (NPPE) is an infrequent life-threatening complication developed immediately after upper airway obstruction. Although NPPE is more common in children (9.4%), its overall incidence is about 0.1% in all general anaesthesia practices.[7] Laryngospasm is the most common cause of NPPE and often presents with hypoxia. Increased negative intrathoracic pressure increases venous return to the heart. It decreases blood drainage into the left atrium, thus increasing hydrostatic pressure in the pulmonary bed and transudation to the alveoli. As a result of all these effects, although pulmonary edema can develop immediately, it has been reported that it can occur within 6 hours or even later.[8] In this case, we present the diagnosis and treatment process of a young patient who developed NPPE late after laparoscopic appendectomy.

The first goal in treating NPPE is to correct hypoxia, which is also blamed on the etiology, and to break the vicious circle.[9] Depending on the obstruction's severity and hypoxia, invasive or non-invasive mechanical ventilation (NIMV) may be preferred. Patient compliance is critical here, and the most appropriate method for the patient should be preferred. Although approximately 50% of cases require positive pressure invasive mechanical ventilation with PEEP[10], cases often require at least physiological PEEP.[11] On the other hand, diuretic treatment was initiated by close hemodynamic, electrolyte and urine monitoring, considering that it would contribute to removing fluid in the alveoli. However, the use of diuretics in the treatment of NPPE is still controversial. Although many studies suggest waiting with fluid restriction, there are also studies reporting that diuretic therapy may contribute to the removal of increased fluid in the alveoli.[12,13] In addition, steroid therapy is recommended in NPPE since increased negative intrathoracic pressure causes damage to the alveoli in the etiology of NPPE, and the effectiveness of steroids in treating alveolar damage has been proven. In the present patient, we started treatment with NPPV in the first hours with a slightly higher dose than the physiological dose. PEEP continued at physiological levels after the treatment response. In addition, diuretic treatment with intravenous furosemide 40 mg/4 mL vial every 24 hours and steroid treatment with a single daily dose of 40 mg methylprednisolone sodium succinate were started.

## Conclusion

NPPE is a rare but severe complication that can develop after acute obstruction in the upper respiratory tract. Laryngospasm is the most common cause of NPPE and often presents with hypoxia. As a result of laryngospasm and hypoxia, pulmonary edema develops. In the first-line treatment depending on the severity of the obstruction and the state of hypoxia, invasive mechanical ventilation or non-invasive mechanical ventilation (NIMV) with diuretic and steroid treatment may be preferred.

## Etik Kurul Kararı

Olgu sunumu oluğundan etik kurul onamına gerek olmayıp; olgunun sunulması için hastanın yazılı onamı alınmıştır.

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## Yazar Katkıları

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