# Acute Development of Negative Pressure Pulmonary Hemorrhage Secondary to Endotracheal Tube Obstruction: A Case Report

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## Abstract

Negative pressure pulmonary edema (NPPE) immediately after general anesthesia is a rare yet life threatening complication. It is caused by an increased fluid in the interstitial spaces and alveoli due to forced inspiratory efforts against tightly closed glottis and was described since 1977. The resulting pulmonary edema can appear within a few minutes after airway obstruction or in a deferred way after several hours, but rarely have frank pulmonary hemorrhage such as in this case. The clinical manifestations are potentially serious, but normally respond well to treatment with supplemental oxygen, positive pressure mechanical ventilation and diuretics. We report a clinical case with acute negative pressure pulmonary edema and exsanguinations of pulmonary bleeding after deep cervical lymph node biopsy under general anesthesia.

**Keywords**: negative pressure pulmonary edema, postoperative complication, airway obstruction, pulmonary hemorrhage, postobstructive pulmonary edema.

# ภาวะเลือดออกเฉียบพลันจากแรงดูดในทางเดินหายใจเนื่องจากท่อช่วยหายใจอุดตัน: รายงานผู้ป่วย

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## บทคัดย่อ

ภาวะ negative pressure pulmonary edema (NPPE) ที่พบหลังจากการดมยาสลบ เป็นภาวะที่พบได้ค่อนข้างยากแต่เป็นอันตรายมากทำให้ผู้ป่วยถึงแก่ชีวิตได้ เมื่อปี ค.ศ. 1977 ได้เริ่มมีการศึกษาและพยายามอธิบายพยาธิสภาพของอาการนี้ว่าเกิดขึ้นเนื่องจากการใช้แรงหายใจอย่างมากในขณะที่ทางเดินหายใจถูกปิดกั้นทำให้มีของเหลวเข้ามาอยู่ในเนื้อเยื่อระหว่างถุงลมในปอด ก่อให้เกิดภาวะน้ำท่วมปอด และในผู้ป่วยบางรายซึ่งพบจำนวนน้อยกว่าแต่อาจมีอาการรุนแรงกว่าคือพบว่ามีเลือดออกอย่างรุนแรงในทางเดินหายใจเช่นในผู้ป่วยรายนี้ อาการที่พบในผู้ป่วยกลุ่มนี้ค่อนข้างรุนแรงและอันตราย แต่ตอบสนองต่อการรักษาดี โดยการให้ออกซิเจน การใช้เครื่องช่วยหายใจแบบมีความดันเป็นบวกและการให้ยาช่วยขับปัสสาวะ

รายงานฉบับนี้เป็นรายงานกรณีผู้ป่วยที่มีเลือดออกอย่างรุนแรงในทางเดินหายใจ (negative pressure pulmonary hemorrhage) เนื่องจากผู้ป่วยกัดท่อช่วยหายใจทำให้ท่อช่วยหายใจอุดตัน ภายหลังการดมยาสลบเพื่อผ่าตัดต่อมน้ำเหลืองบริเวณคอส่วนลึกเพื่อการวินิจฉัยโรค

## Introduction

The negative pressure pulmonary edema (NPPE) or postobstructive pulmonary edema (POPE), is an unusual complication found during surgery under general anesthesia.[1,2] The incidence of NPPE has been reported at only 0.05% to 0.1% of all anesthetic practices.[2] Nevertheless, over the past 15 years, increasing number of such cases has been reported in literature. According to an Australian study of 4,000 incidences of laryngospasm in postanesthesia, this complication occurred up to 4% of total cases.[3] The NPPE can be classified into two types: Type I or Type II.[4] Type I NPPE develops immediately after onset of acute airway obstruction, whereas Type II develops after the relief of chronic upper airway obstruction. Although causes of upper airway obstruction that lead to NPPE have been varied but postoperative laryngospasm is on the top of chart.[5] Other recognized causes include tumor, strangulation, hanging, bilateral vocal cord paralysis, foreign body aspiration, pseudomembranous formation in pharynx, saber sheath trachea, endotracheal tube obstruction and severe episode of obstructive sleep apnea. The negative pressure pulmonary hemorrhage is an extremely rare complication that could be found in post airway obstruction case.

We report a case of a 20-year-old female patient with clinical presentation of a prolonged fever of unknown origin over the past 5 months. The patient developed acute upper airway obstruction after tightly clenched on endotracheal tube towards the end of deep left cervical lymph node biopsy procedure. Vigorous inspiratory efforts against a completely obstruction airway led to the development of acute negative pressure pulmonary hemorrhage.

## Case Report

A 20-year-old female, weighing 50 kilograms, presented with a prolonged fever of unknown origin over the past 5 months. The physical examination showed enlargement of the deep left cervical lymph node in posterior triangle of neck. The plain x-ray and computer tomography of the chest revealed an anterior mediastinal mass. (Figure 1 and 2) The patient was scheduled for a deep left cervical lymph node biopsy.

Anesthesia was induced with propofol 120 mg with fentanyl 75 µg intravenously and endotracheal intubation was easily facilitated by atracurium 30 mg intravenously. Anesthesia was

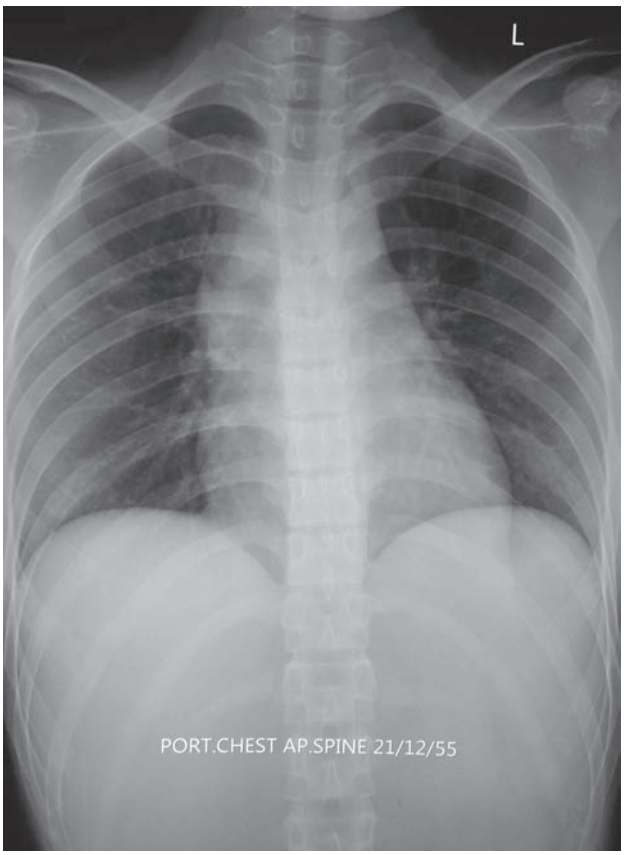


**Figure 1:** Chest X-ray showed slightly widening mediastinum.

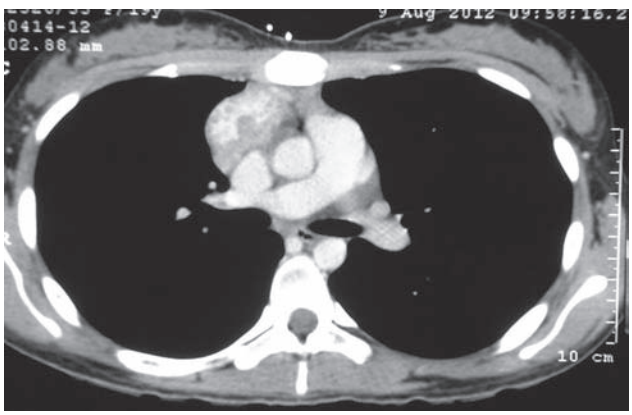


**Figure 2:** CT scan showed anterior mediastinal mass.

maintained with 1-2% sevoflurane in nitrous oxide and oxygen mixture. The operation was smooth and completed easily. At the end of procedure, the patient was given 1.2 mg of atropine with 2.5 mg of *neostigmine* for reversal of muscle relaxant.

After recovery of the muscle power, patient tightly clenched on the endotracheal tube and began struggling to take breath against the obstructed tube. The patient then developed hypoxemia, with oxygen saturation on pulse oxygenator declining to 25%. Patient blood pressure was around 141/93 mmHg, with heart rate raising to 160 beats per minute.

Rescued maneuvers was carried out with 100 mg of thiopental and 200 mg of propofol given. Approximately 1-2 minute later, the patient started to develop hemoptysis with massive amount of frank blood flowing into the tube. Blood suction was performed with a total of more than 600 ml of frank blood collected in the fluid suction collector over the entire resuscitation time.

The satisfactory oxygenation level was achieved with 100% oxygenation and high positive end-expiratory pressure (PEEP), continuous endotracheal tube suction, and consistent intravenous infusion of propofol 20 ml per hour given. Moreover, 20 mg of intravenous furosemide and 8 mg of intravenous dexamethasone were provided.

The patient was then rapidly transferred to surgical cardiac care unit (SCCU). The initial arterial blood gas, while being on ventilator with 100% oxygen and 10 $cmH_2O$ PEEP at SCCU, reported mixed respiratory acidosis and metabolic acidosis with normal oxygenation (pH 7.28, $pCO_2$ 46.7 mmHg, $pO_2$ 142.2 mmHg, BE -4.9 mmol/L, $HCO_3$ 21.5 mmol/L). Patient haematocrit dropped from 33% in pre-operative day to 28% and packed red blood cells were transfused. The immediate chest x-ray film showed butterfly appearance as shown in figure 3. The patient later received 24-hour sedation with regular intravenous infusion of 20-35 ml per hour of propofol.

On the postoperative day 1, patient's clinical conditions improved with satisfactory arterial blood gas and chest x-ray film as shown in figure 4. Patient's arterial blood gas was normal at 100%

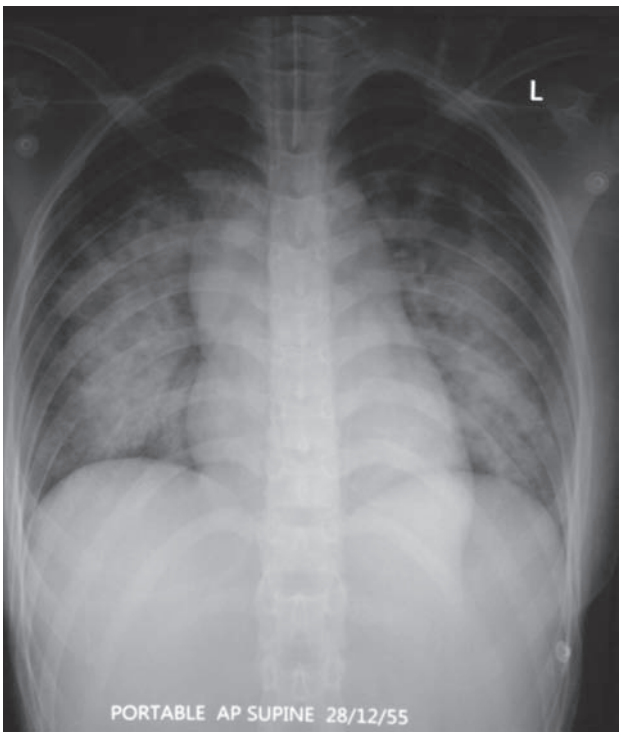


**Figure 3:** The chest x-ray film showed bilateral perihilar alveolar edema (butterfly appearance).

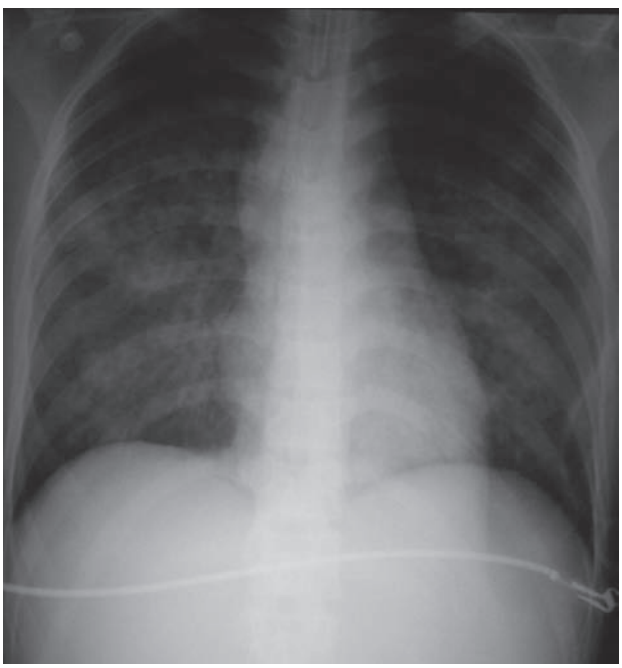

**Figure 4:** The chest x-ray film showed improvement of bilateral alveolar edema on post-operative day 1.

Oxygen and 10 $cmH_2O$ (pH 7.38, $pCO_2$ 37.5 mmHg, $pO_2$ 251.2 mmHg, BE -3.3 mmol/L, $HCO_3$ 22.4 mmol /L). Subsequently, the ventilator parameter was set to an appropriate level.

On the post-operative day 3, the patient successfully managed to extubate herself without any subsequent complication. Non-Hodgkin lymphoma was the definite diagnosis of pathological lymph node. After the first course of chemotherapy, the patient was discharged from the hospital on post-operative day 7.

## Discussion

Many studies try to explain the pathophysiology of NPPE. In 1927, Moore, et al. had created experimental pathology of the NPPE in spontaneously breathing dogs with resistive load.[6] The clinical significance of this theory in three adult patients was reported by Oswalt, et al.[7] The pathophysiology of NPPE has been extensively reviewed by several studies. From many studies in pathophysiologic view, disturbances of pulmonary fluid homeostasis can be induced by four mechanisms: 1) increased hydrostatic pressure in the pulmonary capillary bed (or decrease of pressure in the interstitium); 2) decrease of osmotic pressure of plasma; 3) increase of permeability of the membrane; and 4) decrease of return of fluid to the circulation via lymphatics.[8]

The pathophysiologic sequence of NPPE can be described as follows: Inspiratory efforts against the blocked airway during episodes of upper airway obstruction may result in a highly negative pleural pressure (more than -100 $cmH_2O$) and a sudden increase of venous return of blood to the heart which consequently increase both end diastolic and end systolic ventricular volume. The sudden increase of pulmonary microvascular pressure, due to very low intrapulmonary pressure, high left ventricular end diastolic pressure (LVEDP) and low left ventricular compliance, lead to the pulmonary edema.

The highly negative intrathoracic pressure immediately increases systemic venous return to the heart and results in a simultaneous drop in cardiac output associated with the preload to the left atrium. Pulmonary capillary pressure increases, while intraalveolar pressure drops. Moreover, alveolar cell junctions are disrupted. Fluid flows rapidly into interstitial and alveolar spaces, after alveolar flooding or capillary failure occurs.[9]

The hypoxemia caused by upper airway obstruction increase the pulmonary vascular resistance and capillary pressure, and precipitate a hyperadrenergic state mimicking neurogenic pulmonary edema.[10]

The disorder can be classified as type I or type II (Table 1). Type I NPPE develops immediately after onset of acute airway obstruction, whereas type II develops after the relief of chronic upper airway obstruction. This case was the Type I NPPE, due to endotracheal tube obstruction.

Clinical presentation included decreased oxygenation with pink frothy sputum and chest radiographic abnormalities. Pulmonary edema caused both impaired diffusion of oxygen and ventilation/perfusion mismatching, leading to sudden and severe hypoxemia.

The pathophysiology of NPPE in this case involved airway obstruction causing severe hypoxemia, and vigorous inspiratory effort against obstructed endotracheal tube causing massive hemoptysis due to capillary failure.[11] The radiographic finding was compatible with perihilar alveolar edema.

The arterial blood gas showed mixed respiratory acidosis and metabolic acidosis. The alveolar-arterial gradient was equivalent to 524.1 mmHg. The shunt effect could be calculated from $PaO_2/PAO_2$ equation and $PaO_2/FiO_2$ equation. The $PaO_2/PAO_2$ equaled to 0.21 and $PaO_2/FiO_2$ equaled to 142.2. Thus, this case involved pulmonary shunt effect. These figures were in correspondence with this patient clinical presentations.

In general, most patients receive standard treatment that includes PEEP and diuretic. However, steroids administration is controversy in NPPE.[12,13]

Table 1:

Causes of negative pressure pulmonary edema[4]

| Type I NPPE | Type II NPPE |
|---|---|
| Postextubation laryngospasm | Posttonsillectomy/adenoidectomy |
| Epiglottitis | Postremoval of upper airway tumor |
| Croup | Choanal atresia |
| Choking/foreign body | Hypertrophic redundant uvula |
| Strangulation | |
| Hanging | |
| Endotracheal tube obstruction; eg.biting, secretions | |
| Laryngeal mask airway blockage; biting, displacement | |
| Laryngeal tumor | |
| Goiter | |
| Mononucleosis | |
| Postoperative vocal cord paralysis | |
| Migration of Foley catheter balloon used to tamponade epitaxis | |
| Near drowning | |
| Intraoperative direct suctioning of endobroncheal tube | |

There has been a case report of NPPE that progressed to adult respiratory distress syndrome (ARDS) and resulted in death[14], although most of the patients respond quickly without further sequelae.

## Conclusion

Most of the NPPE patients resolve generally without any further sequalae with prompt diagnosis and therapeutic action, despite mortality rates ranging from 11% to 40%[15]. The incidence of negative pressure pulmonary hemorrhage is less than that of NPPE. It can be found in young athletic patients that have strong thoracic muscular power.

There is no intervention proven to prevent NPPE. This case report places emphasis on the important of bite block/oropharyngeal airway used at the end of surgery in order to avoid NPPE. The period of patient emerging from anesthesia is critical and can lead to tube biting and endotracheal tube obstruction.

In conclusion, early detection and appropriate treatment are crucial to decrease morbidity and mortality in these patients.

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