

Post-laryngospasm pulmonary edema. A case report

Edema pulmonar poslaringoespasmo. Presentación de un caso

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Summary

Negative Pressure Pulmonary Edema (NPPE) is a potentially fatal form of non-cardiogenic pulmonary edema that generally affects young individuals without comorbidities. The pathophysiology involves the development of negative intrathoracic pressure due to intense inspiratory effort and upper airway obstruction. In adults, the most common etiology is post-extubation laryngospasm in the immediate postoperative period, affecting less than 0.1% of healthy patients undergoing general anesthesia. We present the case of a 22-year-old male patient with no comorbidities who was admitted to the emergency department with abdominal pain. Acute appendicitis was diagnosed, and an open appendectomy was performed without complications. The patient was extubated 50 minutes after the start of surgery. Two hours later, he developed laryngospasm, with oxygen saturation levels of 88–90% without dyspnea. Despite an increase in oxygen flow, hypoxemia persisted, and a chest X-ray revealed multiple ground-glass opacities, suggesting pulmonary edema. The patient was treated with furosemide and respiratory therapy. This reported case highlights the need to suspect a diagnosis of NPPE in young, healthy patients who develop acute hypoxemia in the immediate postoperative period, as this condition can be fatal if not promptly identified and treated.

Key words (MeSH)

Pulmonary edema
Postoperative period
Hypoxia

Palabras clave (DeCS)

Edema pulmonar
Periodo posoperatorio
Hipoxia

Resumen

El edema pulmonar por presión negativa (EPPN) es una forma de edema pulmonar no cardiogénico, potencialmente fatal, que generalmente afecta a personas jóvenes y sin comorbilidades. La fisiopatología implica el desarrollo de la presión negativa intratorácica debido al esfuerzo de inspiración intenso y a la obstrucción de las vías aéreas superiores. En adultos, la etiología más común es el laringoespasmo posextubación en el periodo posoperatorio inmediato, que afecta a menos del 0,1% de pacientes sanos sometidos a anestesia general. Se presenta el caso de un paciente de 22 años de edad, sin comorbilidades, que fue ingresado en urgencias por dolor abdominal. Se encontró apendicitis aguda y se realizó una apendicectomía abierta sin complicaciones. El paciente fue extubado 50 minutos después del inicio de la cirugía. Dos horas más tarde, desarrolló el laringoespasmo, con una saturación de oxígeno del 88-90 %, sin disnea. A pesar del aumento en el flujo de oxígeno, la hipoxemia persistió, y una radiografía de tórax mostró múltiples opacidades en vidrio esmerilado, que sugirieron edema pulmonar. El paciente fue tratado con furosemida y terapia respiratoria. Este caso subraya la necesidad de sospechar un diagnóstico de EPPN en pacientes jóvenes y sanos que desarrollan hipoxemia aguda durante el periodo posoperatorio inmediato, ya que esta condición puede ser fatal si no se identifica y se trata a tiempo.

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Introduction

Negative pressure pulmonary edema (NPPE) is a potentially life-threatening emergency (1) that can develop as a result of acute or chronic disease (2). This condition is characterized by the presence of bilateral pulmonary interstitial edema (3,4). There are two types of NPPE: the first occurs after extubation and accounts for 50% of cases attributable to laryngospasm, while the second occurs after the removal of a chronic upper airway obstruction (1).

Laryngospasm leads to acute upper airway obstruction (3), resulting in the development of hypoxia within minutes (1-90 minutes) (4). It has been proposed that healthy patients can generate high levels of negative inspiratory pressure reaching up to -140 cmH20, which is transmitted to the intrapleural spaces; subsequently, there is an increase in venous return to the right heart and pulmonary venous pressures. During this process, a drop in perivascular hydrostatic pressure has been demonstrated, causing the displacement of intracapillary fluid to the pulmonary interstitial space or increasing the permeability of the capillaries and alveolar epithelium. Another accepted theory is the hyperadrenergic state, which can cause peripheral vasoconstriction and

increased venous return, further increasing pulmonary blood flow (3). The first clinical case was described in 1973 (5). This pathology is rare, and there is little epidemiological data available. Its incidence varies from 0.1% to 12% (6,7) and in surgical patients under general anesthesia it is less than 0.1% (6). This article presents a case of pulmonary edema following an episode of laryngospasm.

Case description

A 22-year-old man with no relevant medical history was admitted to the emergency department with a 12-hour history of abdominal pain in the flank and right iliac fossa, accompanied by nausea and subjective fever. Physical examination revealed a distended abdomen, positive Blumberg's sign, hemodynamic stability, no fever, and SatO2 of 97% at room temperature, with moderate leukocytosis and CRP of 52.26 mg/L.

A contrast-enhanced computed tomography (CT) scan of the abdomen was performed, revealing findings consistent with acute appendicitis without alterations in the lung parenchyma (Figure 1). The patient underwent open appendectomy under general anesthesia and was extubated 50 minutes after the start of surgery without complications.

The patient was admitted to the post-anesthesia care unit with low-flow supplemental oxygen and was hemodynamically stable. Two hours later, he developed laryngospasm with a SatO2 of 88-90% with a nasal cannula at 2 liters, BP 117/70 mmHg, HR 78 bpm, RR 20 bpm, without dyspnea. On physical examination, he was alert, with no signs of respiratory distress and no pathological breath sounds. The oxygen flow was increased to 3 liters, and observation and respiratory therapy were indicated.

Five hours later, the patient continued to have borderline oximetry readings (SatO2 of 88-92%) and was unable to be weaned off oxygen. A chest X-ray was performed, showing multiple ground-glass opacities diffusely occupying both lung fields, predominantly in the upper lobes (Figure 2). Treatment with a single 40 mg dose of furosemide was indicated. On examination, right apical crackles were auscultated without dyspnea. Follow-up laboratory tests showed decreasing leukocytosis with marked neutrophilia and CRP that doubled its previous value, a finding expected in different types of pulmonary edema. A chest CT scan (Figure 3) showed multiple areas of ground glass opacity, diffusely affecting both lung fields with a tendency toward consolidation of the anterior segment of the right upper lobe.

On the second day of hospitalization, he required ventilatory support with a nasal cannula and presented with epistaxis and hemoptysis. Tests for autoimmune pathology were requested, with negative results. Control paraclinical tests showed a significant decrease in CRP and a blood count without leukocytosis. The patient had no further episodes of respiratory distress. No respiratory function tests were performed; however, given the adequate clinical and paraclinical evolution, discharge was indicated.

Discussion

NPPE is a non-cardiogenic disease that can be triggered by acute or chronic diseases (2). It is classified into types I and II. Type I occurs due to acute obstruction of the upper airway, such as vocal cord paralysis, foreign bodies, hanging (1), and post-extubation laryngospasm, which accounts for approximately 50% of cases (2), while type II occurs after the release of a chronic obstruction (1) in patients with various factors such as laryngeal tumors, goiter, enlarged tonsils, obesity, and obstructive sleep apnea (1,2). It is usually more common in healthy people or athletes, but it can also occur in children (8). In this case, it involves a young man with no risk factors, in the postoperative period of an open appendectomy. The patient was hemodynamically stable before and during the procedure, but presented with type I NPPE following a possible laryngospasm.

The diagnosis of NPPE is based on clinical history, imaging tests, and identified triggers. Symptoms range from mild respiratory distress to hypoxemia and metabolic acidosis, depending on the duration and severity of the obstruction (2). Laryngospasm leads to the development of hypoxia within minutes (1-90 minutes) (4). In this case, it occurred 50 minutes after extubation, manifesting as a decrease in oxygen saturation despite the use of low-flow supplemental oxygen, with no physical examination findings suggesting another diagnosis.

According to the literature, in cases of post-extubation laryngospasm, chest X-rays show pulmonary edema and a normal heart (1), as was the case with this patient. The chest CT scan showed bilateral ground-glass opacities with evidence of consolidation in the basal segment of the right upper lobe.

Differential diagnosis is essential to establish the appropriate treatment, considering pathologies such as fat embolism (4, 8), posterior pneumonia, acute respiratory distress syndrome (ARDS), heart failure, and COVID-19 (2). ARDS differs from NPPE in that it is triggered by other causes such as sepsis, severe pneumonia, or trauma. Furthermore, NPPE commonly resolves within 1 to 2 days if treated appropriately. Radiologically, ARDS is characterized by diffuse bilateral pulmonary infiltrates with an alveolar, ground-glass appearance, heterogeneous distribution, areas of consolidation, and relatively preserved areas (9). On the other hand, NPPE is characterized by a perihilar distribution with a more symmetrical pattern, butterfly wings, and decreased vascular clarity in areas of opacity (10).

The prevention of NPPE has not been fully proven; however, it is currently based on reducing risk factors such as laryngeal irritation caused by the use of devices such as endotracheal or supraglottic tubes and injuries from repeated laryngoscopies. For this reason, the use of topical laryngotracheal anesthesia and stage 1 extubation is recommended, since stage 2 extubation carries a higher risk of laryngospasm (11).

Treatment of NPPE includes maintaining a patent airway. The administration of positive end-expiratory pressure (PEEP) is recommended, as it allows for the management of hypoxemia and avoids intubation (11). Mechanical ventilation should only be reserved for critically ill patients who do not respond to noninvasive mechanical ventilation. Another pillar of treatment is the use of diuretics such as furosemide; however, the use of diuretics has not been proven effective in studies and may exacerbate hypoperfusion and decrease blood volume (3).

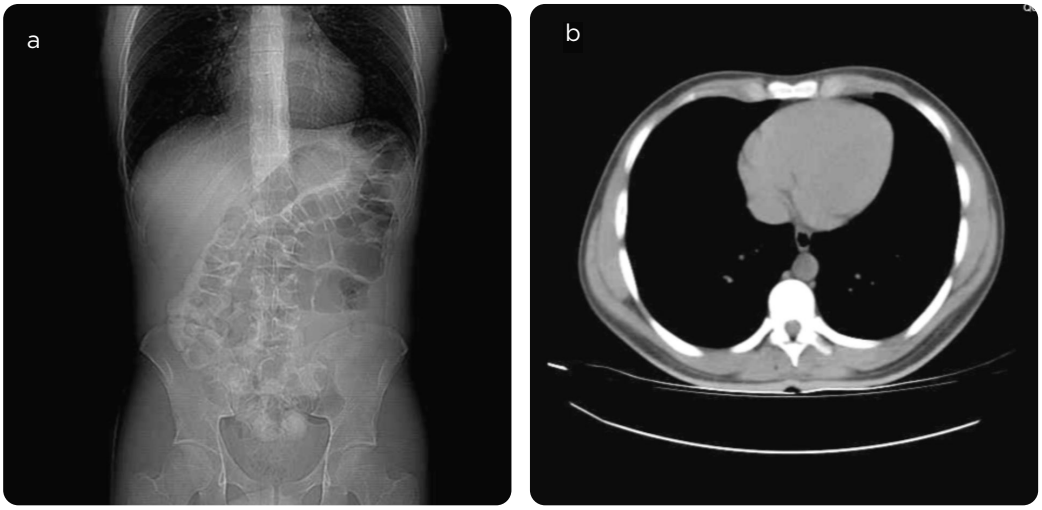


Figure 1. Abdominal CT scan prior to appendectomy. No opacities, consolidations, or pleural effusions are visible in the lung bases.

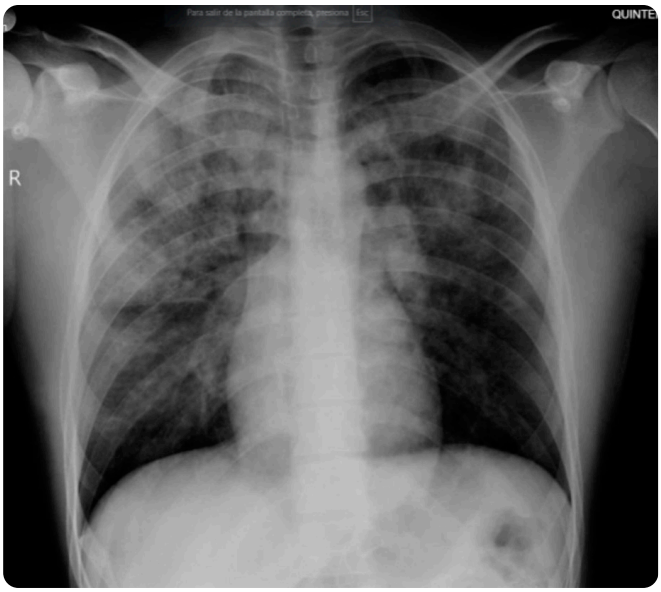


Figure 2. Post-appendectomy chest X-ray. Anteroposterior, single projection. Alveolar opacities with a tendency to coalesce, obscuring the silhouette of the blood vessels. Distribution in the lung apices and middle lobe.

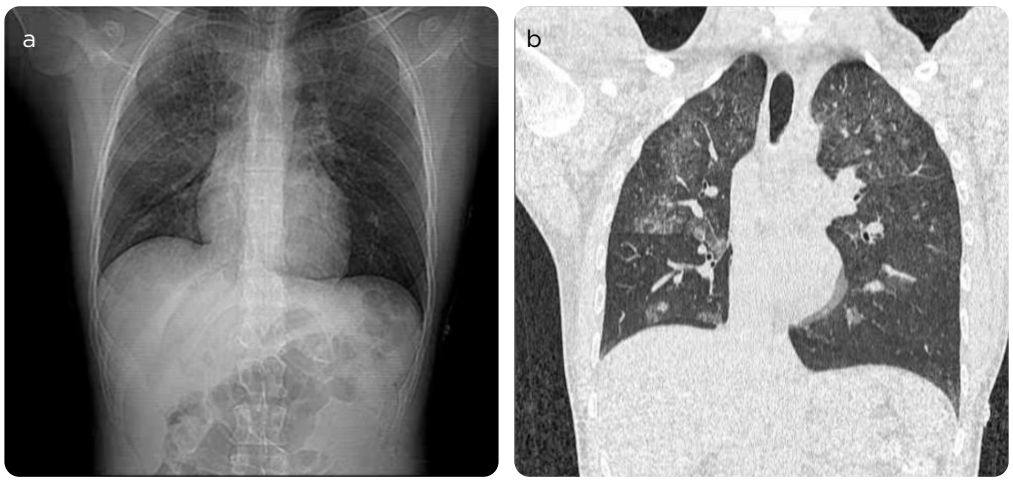


Figure 3. Post-surgical chest CT scan. Window for lung parenchyma. Coronal reconstructions: bilateral asymmetric perihilar ground-glass opacities mainly affecting the upper lobes and middle lobe.

In the case documented here, the patient presented with laryngospasm minutes after extubation, which was initially treated with increased supplemental oxygen flow. Given the persistence and findings of pulmonary edema on chest X-ray, it was decided to add anti-edema management.

This is a case of pulmonary edema following post-extubation laryngospasm in the postoperative period of an open appendectomy under general anesthesia in a young patient with no apparent risk factors and no previous clinical or imaging findings of pulmonary pathology. Therefore, it is important to suspect and quickly identify this condition, as it is a potentially life-threatening emergency.

Conclusion

Post-laryngospasm NPPE is a rare condition, but it is crucial to consider it in young, healthy patients who experience hypoxemia within minutes without a known cause, as it represents an emergency that can be fatal if not diagnosed and managed promptly.

References

1. Tebay A, Bouti K, Tebay N. Œdème pulmonaire à pression négative après une cholécystectomie – à propos d'un cas. Revue de Pneumologie Clinique. 2017;73(5):267-71. https://doi.org/10.1016/j.pneumo.2017.08.006
2. Ma J, Liu T, Wang Q, Xia X, Guo Z, Feng Q, et al. Negative pressure pulmonary edema (Review). Exp Ther Med. 2023;26(3):455. https://doi.org/10.3892/etm.2023.12154
3. Lemyze M, Mallat J. Understanding negative pressure pulmonary edema. Intensive Care Med. 2014;40(8):1140-3. https://doi.org/10.1007/s00134-014-3307-7
4. Carels K, Herpels V, Cardoen L, Lecluyse C, Traen S, Verschakelen J. Postextubation pulmonary edema: an unusual cause of transient pulmonary edema. JBR-BTR. 2013;96(1):17-8. https://doi.org/10.5334/jbr-btr.165
5. Capitanio MA, Kirkpatrick JA. Obstructions of the upper airway in children as reflected on the chest radiograph. Radiology. 1973;107:159-61.
6. Deepika K, Kenaan CA, Barrocas AM, Fonseca JJ, Bikazi GB. Negative pressure pulmonary edema after acute upper airway obstruction. J Clin Anesth. 1997;9:403-8.
7. Tami TA, Chu F, Wildes TO, Kaplan M. Pulmonary edema and acute upper airway obstruction. Laryngoscope. 1986;96(5):506-9. https://doi.org/10.1288/00005537-198605000-00007
8. Ben-David Y, Bentur L, Gur M, Ilivitzki A, Toukan Y, Nir V, et al. Pediatric negative pressure pulmonary edema: Case series and review of the literature. Pediatr Pulmonol. 2023;58(12):3596-9. https://doi.org/10.1002/ppul.26700
9. Goodman LR, Fumagalli R, Tagliabue P, Tagliabue M, Ferrario M, Gattinoni L, et al. Adult respiratory distress syndrome due to pulmonaray and extrapulmonary causes: CT, clinical, and functional correlations. Radiology. 1999;213(2):545-52. https://doi.org/10.1148/radiology.213.2.r99nv42545
10. Karaman I, Ozkaya S. Differential diagnosis of negative pressure pulmonary edema during COVID-19 pandemic. J Craniofac Surg. 2021;32(5):e421-3. https://doi.org/10.1097/SCS.0000000000007226
11. Bhaskar B, Fraser JF. Negative pressure pulmonary edema revisited: Pathophysiology and review of management. Saudi J Anaesth. 2011;5(3):308-13. https://doi.org/10.4103/1658-354X.84108

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