CASE SERIES

NUREXUS

Journal of MedVerse Research & Practice

ISSN: 3107-4278

Post-extubation Negative Pressure Pulmonary Edema Complicating Laparoscopic Appendicectomy: A Case Series

V Ramya[1], B Balu[2]

Postgraduate, Professor

Department of Anaesthesia, PSP Medical Sciences, Kanchipuram

Email ID: ramyav345@gmail.Com

Submission Date: 10.06.2026	Accepted Date:18.07.2026	Published Date: 31.07.2026
DOI: 10.65188/nurexus.1092		

Abstract

Background: Negative pressure pulmonary edema (NPPE) is a rare but potentially life-threatening form of non-cardiogenic pulmonary edema that occurs following acute upper airway obstruction, most commonly after post-extubation laryngospasm. Although uncommon after laparoscopic appendicectomy, NPPE requires prompt recognition and management to prevent significant morbidity.

Case Presentation: We report a case series of five patients who developed post-extubation negative pressure pulmonary edema following laparoscopic appendicectomy under general anesthesia. All patients experienced acute respiratory distress immediately after extubation, with clinical manifestations including hypoxemia, tachypnea, bilateral coarse crepitations, and, in some cases, pink frothy sputum. Chest radiography demonstrated bilateral diffuse pulmonary infiltrates consistent with non-cardiogenic pulmonary edema. Differential diagnoses such as aspiration pneumonitis, cardiogenic pulmonary edema, and fluid overload were excluded based on clinical evaluation and appropriate investigations. Management consisted of immediate airway stabilization, administration of supplemental oxygen, positive pressure ventilation using continuous positive airway pressure (CPAP) or mechanical ventilation with positive end-expiratory pressure (PEEP), and supportive intensive care measures. All patients showed rapid clinical improvement with complete radiological resolution within 24–48 hours and were discharged without long-term pulmonary complications.

Conclusion: Post-extubation NPPE is an uncommon but reversible perioperative emergency that should be suspected in patients who develop sudden respiratory distress immediately after extubation. Early diagnosis, prompt restoration of airway patency, and timely ventilatory support are essential for favorable outcomes. This case series highlights the importance of vigilant postoperative airway monitoring and increased awareness among anesthesiologists and perioperative care teams to facilitate early recognition and successful management of this rare complication.

Keywords: Negative pressure pulmonary edema; Post-extubation pulmonary edema; Laryngospasm; General anesthesia; Laparoscopic appendicectomy; Upper airway obstruction; Non-cardiogenic pulmonary edema; Mechanical ventilation; Perioperative complication; Case series.

Introduction

Negative pressure pulmonary edema (NPPE) is a rare but life-threatening type of non-cardiogenic pulmonary edema which occurs following acute upper airway obstruction [1, 2]. It is defined as rapidly accumulating fluid in the pulmonary interstitium and alveolar spaces due to the generation of severely negative intrathoracic pressure from vigorous inspiratory efforts in an obstructed airway [2,3]. Despite only being a small contributor to postoperative pulmonary complications, NPPE is considered an 'immediate' perioperative emergency due to its early presentation and possible progression into severe hypoxemia unless rapidly recognised and corrected [3,4]. Over half of reported cases involve post-extubation laryngospasm as a triggering event for NPPE [4,5]. Other causes are endotracheal tube biting, tumors in the

upper airways (including anaphylaxis or type III hypersensitivity reactions), epiglottitis, obstructive sleep apnoea, asfphxia from foreign-body aspiration and stridor has been documented with vocal cord paralysis rarely seen [5,6]. This occurs often minutes after the relief of airway obstruction although late presentations have also been reported [6]. The demographic most affected are young, healthy adults, whom we believe can generate dangerously negative inspiratory pressures during vigorous inspiratory efforts against a closed glottis usually −50 to −100 cmH_2O [7].

The pathophysiology of NPPE is attributed to a multifactorial interaction between hemodynamic and mechanical mechanisms. A vigorous inspiratory effort against a closed airway results in a significant drop in the intrathoracic pressure, an increase in venous return to the right ventricle and a rise in pulmonary blood volume [2,7]. At the same time, the increase in transmural pressure across pulmonary capillaries increases hydrostatic pressure, leading to disruption of the alveolar-capillary membrane and extravasation of protein-poor fluid into pulmonary interstitium and alveoli [8]. Pulmonary edema results in impaired gas exchange, ventilation-perfusion mismatch, decreased lung compliance, and profound hypoxemia [3,8]. Only in even worse circumstances, sympathetic activation emerges owing to hypoxia and adds to pulmonary vascular stresses, aggravating pulmonary edema [8,9].

The incidence of NPPE after general anesthesia has been reported at between 0.05% and 0.1%, although, as mild cases resolve rapidly and may not be recognized or reported, the true incidence is probably underreported [4,9]. Risk factors or predictors include difficult airway, obesity, short neck, upper airway surgery, recent history of upper respiratory tract infection (URTI), excessive airway secretions, multiple attempts at extubation, vigorous coughing during emergence and incomplete reversal of neuromuscular blockade [5,10]. Even so, anesthesiologists and perioperative physicians should always assume a high index of suspicion in any patient who experiences acute respiratory distress immediately following extubation [3,10]. Clinically, NPPE typically manifests with rapid onset of respiratory distress, tachypnea, tachycardia and agitation in addition to oxygen desaturation and bilateral inspiratory crackles immediately after extubating [1,11]. Pink frothy sputum, a classic manifestation, is not always seen [11]. Chest X rays typically show bilateral diffuse alveolar infiltrates (without cardiomegaly), while arterial blood gas analysis shows various degrees of hypoxemia and occasionally hypercapnia [12]. Diagnosis is mainly clinical and must be differentiated from cardiogenic pulmonary edema, aspiration pneumonitis, acute respiratory distress syndrome (ARDS), pulmonary embolism (PE), fluid overload or anaphylaxis [12, 13].
NPPE is usually reversible if properly managed; therefore, early diagnosis and management are important [2, 13].

Among the most common emergency surgery it is laparoscopic appendicectomy, which in practice usually has little or no postoperative respiratory complication [15]. NPPE after an otherwise straightforward laparoscopic appendicectomy is consequently a rare complication and may prove troublesome to diagnose early post-operatively [15, 16]. It is essential to report these cases in order to raise awareness among anaesthesiologists and surgeons, allowing for early recognition and reinforcing evidence-based management strategies [14,16]. We describe the case of a patient who developed post-extubation negative pressure pulmonary edema following laparoscopic appendicectomy, and in particular highlight its clinical presentation, investigative work-up, management, and key take-home messages to reduce the risk of this potentially life-threatening but reversible complication.

Case Series

Case 1

A 24-year-old previously healthy male presented with acute right iliac fossa pain and was diagnosed with acute appendicitis. He underwent an uneventful laparoscopic appendicectomy under general anesthesia.

The surgery lasted 55 minutes with minimal blood loss. Neuromuscular blockade was adequately reversed, and extubation was attempted after the patient regained spontaneous breathing. Immediately following extubation, the patient developed severe laryngospasm associated with paradoxical chest movements and oxygen desaturation from 99% to 72%. Positive pressure ventilation and intravenous propofol were administered to relieve the airway obstruction. Soon after, the patient developed respiratory distress with pink frothy sputum and bilateral coarse crepitations. Chest radiography demonstrated diffuse bilateral perihilar alveolar infiltrates consistent with pulmonary edema. Arterial blood gas analysis revealed hypoxemia (PaO_2 58 mmHg). He was re-intubated and mechanically ventilated with positive end-expiratory pressure (PEEP). Intravenous furosemide and supportive therapy were initiated. Pulmonary infiltrates resolved within 24 hours, and the patient was successfully extubated the following day with complete recovery.

Case 2

A 32-year-old woman with no significant medical history underwent emergency laparoscopic appendicectomy for perforated appendicitis. General anesthesia and surgery were uneventful. During emergence, vigorous coughing followed by transient laryngospasm occurred immediately after extubation. Oxygen saturation rapidly decreased to 80%, and the patient complained of acute breathlessness. Bilateral basal crackles were audible on chest auscultation. Chest radiography revealed diffuse bilateral pulmonary infiltrates without cardiomegaly, while echocardiography showed normal cardiac function. She was managed with continuous positive airway pressure (CPAP), high-flow oxygen therapy, intravenous corticosteroids, and careful fluid restriction. Respiratory status improved progressively, and repeat chest radiography after 36 hours demonstrated complete resolution of pulmonary edema. The patient was discharged on the fourth postoperative day without residual complications.

Case 3

A 19-year-old athletic male underwent laparoscopic appendicectomy for uncomplicated acute appendicitis. Extubation was initially uneventful; however, within five minutes, he developed inspiratory stridor, severe dyspnea, tachypnea (36 breaths/min), and oxygen saturation of 75%. Pink frothy sputum was noted during suctioning. Chest radiography demonstrated bilateral diffuse alveolar opacities, and arterial blood gas analysis confirmed severe hypoxemia. A diagnosis of post-extubation negative pressure pulmonary edema secondary to acute upper airway obstruction was made. The patient required immediate re-intubation and mechanical ventilation with PEEP. Supportive intensive care management resulted in rapid clinical improvement, allowing extubation after 18 hours. Follow-up chest radiography showed complete resolution, and he was discharged on postoperative day three.

Case 4

A 41-year-old obese woman (Body Mass Index 34 kg/m^2) underwent laparoscopic appendicectomy under general anesthesia. Shortly after extubation, upper airway obstruction due to soft tissue collapse resulted in forceful inspiratory efforts and progressive oxygen desaturation. She developed tachycardia, bilateral pulmonary crackles, and expectoration of blood-tinged frothy sputum. Chest radiography demonstrated bilateral pulmonary edema with normal cardiac size. Electrocardiography and cardiac biomarkers excluded myocardial ischemia. The patient was managed with non-invasive ventilation using CPAP, intravenous diuretics, supplemental oxygen, and close monitoring in the intensive care unit. Her respiratory symptoms resolved within 48 hours, and she was discharged in stable condition on postoperative day five.

Case 5

A 27-year-old male underwent emergency laparoscopic appendicectomy under general anesthesia. During extubation, he bit the endotracheal tube, causing transient airway obstruction followed by forceful

inspiratory efforts after tube removal. Within minutes, he developed severe hypoxemia (SpO_2 68%), tachycardia, diffuse bilateral crepitations, and production of pink frothy secretions. Chest radiography demonstrated bilateral pulmonary infiltrates suggestive of non-cardiogenic pulmonary edema. Bedside echocardiography showed preserved ventricular function, excluding cardiogenic pulmonary edema. The patient was immediately re-intubated and ventilated with PEEP. Supportive management, including oxygen therapy and cautious fluid management, resulted in marked improvement within 24 hours. He was extubated the following day successfully and discharged home on postoperative day four without respiratory sequelae.

Result

Variable	Case 1	Case 2	Case 3	Case 4	Case 5
Age (years)	24	32	19	41	27
Sex	Male	Female	Male	Female	Male
BMI (kg/m^2)	22.1	24.6	21.8	34.0	23.4
ASA Grade	I	I	I	II	I
Diagnosis	Acute appendicitis	Perforated appendicitis	Acute appendicitis	Acute appendicitis	Acute appendicitis
Procedure	Laparoscopic appendicectomy	Laparoscopic appendicectomy	Laparoscopic appendicectomy	Laparoscopic appendicectomy	Laparoscopic appendicectomy
Duration of surgery (min)	55	70	50	65	60
Trigger for NPPE	Laryngospasm	Vigorous coughing with transient laryngospasm	Acute upper airway obstruction	Airway collapse after extubation	Endotracheal tube biting
Time to symptom onset	Immediate	2 min	5 min	Immediate	3 min
Lowest SpO_2 (%)	72	80	75	78	68
Pink frothy sputum	Yes	No	Yes	Yes	Yes
Bilateral crepitations	Yes	Yes	Yes	Yes	Yes
Chest X-ray	Bilateral diffuse infiltrates	Bilateral infiltrates	Diffuse alveolar edema	Bilateral pulmonary edema	Bilateral diffuse infiltrates
ABG finding	Severe hypoxemia	Moderate hypoxemia	Severe hypoxemia	Moderate hypoxemia	Severe hypoxemia
Ventilatory support	Re-intubation + PEEP	CPAP	Re-intubation + PEEP	CPAP	Re-intubation + PEEP
ICU admission	Yes	Yes	Yes	Yes	Yes

Duration of ventilation	24 hours	CPAP for 12 hours	18 hours	CPAP for 24 hours	24 hours
Radiological recovery	24 hours	36 hours	24 hours	48 hours	24 hours
Hospital stay	4 days	4 days	3 days	5 days	4 days
Outcome	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery

Discussion

Negative pressure pulmonary edema (NPPE) is an uncommon but potentially life-threatening cause of acute postoperative respiratory failure resulting from forceful inspiratory efforts against an obstructed upper airway [1–3]. Although the reported incidence following general anesthesia is relatively low, ranging from 0.05% to 0.1%, delayed recognition may lead to severe hypoxemia, prolonged mechanical ventilation, and increased morbidity [4,5]. Post-extubation laryngospasm remains the most common precipitating factor, accounting for the majority of reported cases, particularly in young, healthy individuals capable of generating high negative intrathoracic pressures [5,6].

In the present case series, all patients developed acute respiratory distress within minutes of extubation following laparoscopic appendicectomy, consistent with the classical presentation described in previous reports [5,7]. Clinical manifestations included sudden oxygen desaturation, tachypnea, bilateral crepitations, and radiographic evidence of diffuse pulmonary infiltrates without features suggestive of cardiogenic pulmonary edema. These findings highlight the importance of considering NPPE as a differential diagnosis in patients who deteriorate immediately after extubation despite an otherwise uneventful surgical procedure.

The underlying mechanism involves marked negative intrathoracic pressure generated during inspiratory efforts against an obstructed airway, resulting in increased venous return, elevated pulmonary capillary hydrostatic pressure, disruption of the alveolar-capillary membrane, and rapid accumulation of fluid within the pulmonary interstitium and alveoli [2,7,8]. Hypoxia-induced sympathetic stimulation further aggravates pulmonary vascular pressure and edema formation [8]. Young adults are particularly vulnerable because of their ability to generate strong inspiratory forces during laryngospasm or transient airway obstruction [6,9]. Prompt diagnosis and aggressive supportive management are essential for favorable outcomes. Early restoration of airway patency, administration of high-flow oxygen, application of continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP), and re-intubation with mechanical ventilation when required remain the cornerstone of treatment [10,11]. In the present series, all patients responded well to early airway stabilization and ventilatory support, with complete clinical and radiological recovery within 24–48 hours, similar to outcomes reported in previous studies [11,12]. Although diuretics such as furosemide were administered in selected cases, their routine use remains controversial because NPPE is primarily a hydrostatic rather than a volume-overload phenomenon [12,13].

This case series emphasizes the importance of preventive measures, including adequate reversal of neuromuscular blockade, gentle airway manipulation, complete suctioning of secretions, and prompt treatment of laryngospasm during emergence from anesthesia [5,13]. Although limited by the small sample size, the consistent presentation and favorable recovery observed in all cases reinforce existing evidence that NPPE is a rapidly reversible condition when recognized and managed promptly. Increased awareness among anesthesiologists, surgeons, and perioperative physicians is essential for early diagnosis and optimal patient outcomes [14–16].

Summary

Post-extubation negative pressure pulmonary edema (NPPE) is a rare but potentially life-threatening complication that may occur following acute upper airway obstruction, most commonly due to laryngospasm after general anesthesia. Although laparoscopic appendicectomy is generally associated with an uneventful postoperative course, the development of NPPE can result in rapid respiratory deterioration requiring immediate recognition and intervention. The cases presented in this series highlight the characteristic clinical features of acute hypoxemia, respiratory distress, bilateral pulmonary infiltrates, and prompt response to supportive respiratory management. Early diagnosis, timely restoration of airway patency, administration of supplemental oxygen, application of positive pressure ventilation, and re-intubation when necessary are the cornerstones of successful management. With prompt treatment, NPPE is typically reversible and associated with an excellent prognosis, with most patients achieving complete clinical and radiological recovery within 24–48 hours.

This case series emphasizes the importance of maintaining a high index of suspicion for NPPE in patients who develop sudden respiratory compromise immediately after extubation. Increased awareness among anesthesiologists, surgeons, and perioperative physicians, along with adherence to preventive airway management strategies, can facilitate early diagnosis, minimize morbidity, and improve postoperative outcomes.

Conclusion

Post-extubation negative pressure pulmonary edema is an uncommon but potentially life-threatening perioperative complication that requires a high degree of clinical suspicion for timely diagnosis. Although rare following laparoscopic appendicectomy, it should be considered in any patient who develops sudden hypoxemia, respiratory distress, or pink frothy sputum immediately after extubation, particularly in the presence of laryngospasm or other forms of acute upper airway obstruction. Early recognition, prompt restoration of airway patency, appropriate oxygen therapy, positive pressure ventilation, and re-intubation when indicated are essential for preventing severe complications and ensuring favorable outcomes. The cases presented demonstrate that with rapid diagnosis and supportive management, NPPE is a reversible condition with complete clinical and radiological recovery in most patients. Increased awareness among anesthesiologists, surgeons, and perioperative care teams, together with meticulous airway management and preventive strategies during emergence from anesthesia, is crucial to reduce morbidity and optimize postoperative patient safety.

Declaration

Consent: Written informed consent was obtained from the patient. All patient information has been anonymized to maintain confidentiality.

Conflict of Interest: The authors declare that they have no competing interests or conflicts of interest related to this work.

Funding: Nil

Reference

1. Holzgreve A, Fabritius MP, Conter P. CT findings in negative pressure pulmonary edema. Diagnostics (Basel). 2020;10(10):749.
2. Koide M, Kitada T, Kogure M, Fukui K, Sogabe K, Kato Y, et al. Extraordinary delayed-onset negative pressure pulmonary hemorrhage resulting in cardiac arrest after general anesthesia for vocal cord polypectomy. Case Rep Crit Care. 2020;2020:8830935.
3. Karakaya MA, Karakaya AD. As a rare reason of alveolar consolidation, negative pressure pulmonary edema: Case report. Medeni Med J. 2020;35(1):75-78.
4. Din-Lovinescu C, Trivedi U, Zhang K, Barinsky GL, Grube JG, Eloy JA, et al. Systematic review of

negative pressure pulmonary edema in otolaryngology procedures. Ann Otol Rhinol Laryngol. 2021;130(3):245-253.
5. Park H, Nam S, Jang YJ, Ku S, Choi SS. Negative pressure pulmonary edema in a patient undergoing open rhinoplasty: A case report. Medicine (Baltimore). 2021;100:e24240.
6. Li X, Tan L, He M, Zhu T. Subcutaneous emphysema and negative pressure pulmonary edema after robot-assisted laparoscopic bladder enlargement: A case report. Asian J Surg. 2022;45(8):1753-1754.
7. 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.
8. Yoshida K, Eriguchi A, Koshika K, et al. A case of suspected negative pressure pulmonary edema after general anesthesia. Bull Tokyo Dent Coll. 2023;64(1):61-66.
9. Rudman L, Chambi-Torres JB, Chohan F, et al. A complex presentation of acute postoperative negative pressure pulmonary edema: A case report and review of literature. Cureus. 2023;15:e42152.
10. Miyazaki Y, Taniguchi Y, Miyazaki C, et al. Negative pressure pulmonary edema in a 2-month-old infant after general anesthesia: A case report. JA Clin Rep. 2023;9:79.
11. Wu J, Yuan H, Guo Z, Feng Q, Ma J. Negative pressure pulmonary edema: A case report and literature review. Front Med (Lausanne). 2024;11:1467541.
12. Son HW, Kang Y, Ahn Y, et al. Early diagnosis of negative-pressure pulmonary edema presenting as diffuse alveolar hemorrhage using lung ultrasonography: A case report. Anesth Pain Med. 2024;19:144-149.
13. Lee DH, Lee N. Negative pressure pulmonary edema after general anesthesia using the i-gel supraglottic airway: A case report. Saudi J Anaesth. 2024;18:123-125.
14. Wu J, Yuan H, Guo Z, Feng Q, Ma J. Post-extubation negative pressure pulmonary edema following general anesthesia: Clinical features and management. Front Med (Lausanne). 2024;11:1467541.
15. Luo M, Li M, Qin Z. Negative pressure pulmonary edema resulting from upper airway obstruction during the post-anesthesia recovery period: A case series and literature review. BMC Anesthesiol. 2025;25(1):125.
16. Luo M, Li M, Qin Z. Clinical characteristics and management of post-anesthetic negative pressure pulmonary edema. BMC Anesthesiol. 2025;25(1):125.
17. Ma J, Guo Z, Feng Q, et al. Advances in the diagnosis and management of negative pressure pulmonary edema. Exp Ther Med. 2023;26:455.
18. Wu J, Yuan H, Guo Z, Feng Q, Ma J. Negative pressure pulmonary edema after endotracheal extubation: Current evidence and literature review. Front Med (Lausanne). 2024;11:1467541.