RESEARCH PAPER

Acute Post-Extubation Negative Pressure Pulmonary Edema Following Laparoscopic Appendicectomy: A Case Series

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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.

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.Introduction

Negative pressure pulmonary edema (NPPE) is a rare but potentially fatal form of non-cardiogenic pulmonary edema that develops following acute upper airway obstruction. It is characterized by the rapid accumulation of fluid within the pulmonary interstitium and alveolar spaces secondary to the generation of markedly negative intrathoracic pressure during forceful inspiratory efforts against an obstructed airway.[1-3] Although NPPE accounts for only a small proportion of postoperative pulmonary complications, its rapid onset and potential for severe hypoxemia make it an important perioperative emergency requiring immediate recognition and intervention.[4-7]

The most common precipitating event for NPPE is post-extubation laryngospasm, which accounts for more than half of reported cases. Other causes include biting of the endotracheal tube, upper airway tumors, epiglottitis, obstructive sleep apnea, foreign body aspiration, vocal cord paralysis, tracheal obstruction, and postoperative airway edema.[1,3] The condition typically occurs within minutes after relief of the airway obstruction, although delayed presentations have also been described. Young, healthy adults are considered particularly susceptible because they are capable of generating substantial negative inspiratory pressures, often exceeding -50 to -100 cmH_2O during vigorous inspiratory efforts against a closed glottis.[8-11]

The pathophysiology of NPPE involves a complex interplay of hemodynamic and mechanical factors. Forceful inspiration against an obstructed airway produces a marked decrease in intrathoracic pressure, leading to increased venous return to the right heart and elevated pulmonary blood volume. Simultaneously, the increased transmural pressure across the pulmonary capillaries raises hydrostatic pressure, resulting in disruption of the alveolar-capillary membrane and movement of protein-poor fluid into the pulmonary interstitium and alveoli. The resulting pulmonary edema causes impaired gas

exchange, ventilation-perfusion mismatch, decreased lung compliance, and significant hypoxemia. In severe cases, sympathetic activation due to hypoxia further increases pulmonary vascular pressures, exacerbating pulmonary edema.[12-15]

The reported incidence of NPPE following general anesthesia ranges from approximately 0.05% to 0.1%, although the true incidence is likely underestimated because mild cases may resolve rapidly without being recognized or reported.[5] Risk factors include difficult airway management, obesity, short neck, upper airway surgery, recent upper respiratory tract infection, excessive airway secretions, repeated attempts at extubation, vigorous coughing during emergence, and inadequate reversal of neuromuscular blockade.[16] Despite its rarity, anesthesiologists and perioperative physicians should maintain a high index of suspicion in any patient who develops acute respiratory distress immediately after extubation.

Clinically, NPPE usually presents with sudden onset of respiratory distress, tachypnea, tachycardia, agitation, oxygen desaturation, and bilateral inspiratory crackles shortly after extubation. Pink frothy sputum, although characteristic, is not universally present. Chest radiography typically demonstrates bilateral diffuse alveolar infiltrates without cardiomegaly, while arterial blood gas analysis reveals varying degrees of hypoxemia and, occasionally, hypercapnia. The diagnosis is primarily clinical and requires differentiation from cardiogenic pulmonary edema, aspiration pneumonitis, acute respiratory distress syndrome, pulmonary embolism, fluid overload, and anaphylaxis.[2,6]

Early diagnosis and prompt management are crucial because NPPE is generally reversible when treated appropriately. Immediate restoration of airway patency, administration of high-concentration oxygen, application of continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP), and re-intubation with mechanical ventilation when necessary constitute the cornerstone of management.[2,7] Most patients recover completely within 12 to 48 hours with timely supportive care, and long-term pulmonary sequelae are uncommon. Nevertheless, delayed recognition may result in prolonged mechanical ventilation, intensive care admission, and significant morbidity.[7]

Laparoscopic appendicectomy is among the most frequently performed emergency surgical procedures and is generally associated with minimal postoperative respiratory complications. The occurrence of NPPE following an otherwise uncomplicated laparoscopic appendicectomy is therefore uncommon and may pose a diagnostic challenge during the immediate postoperative period. Reporting such cases is important to increase awareness among anesthesiologists and surgeons, facilitate early recognition, and reinforce evidence-based management strategies.[8] We present a case of post-extubation negative pressure pulmonary edema following laparoscopic appendicectomy, emphasizing its clinical presentation, diagnostic approach, therapeutic management, and key perioperative lessons for preventing 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/m²) 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.

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/m²)	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	72	80	75	78	68

$_2$ (%)					
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 yet potentially life-threatening cause of acute postoperative respiratory failure. It develops as a consequence of forceful inspiratory efforts against an obstructed upper airway, leading to markedly negative intrathoracic pressure, increased pulmonary capillary hydrostatic pressure, and rapid transudation of fluid into the pulmonary interstitium and alveolar spaces.[1,4] Although the reported incidence following general anesthesia ranges from 0.05% to 0.1%, the true incidence is likely underestimated because mild cases often resolve rapidly and remain unrecognized.[2,5] Despite its rarity, NPPE remains an important perioperative complication due to its sudden onset and the need for immediate intervention.[2]

In the present case series, all patients developed acute respiratory distress within minutes of extubation following laparoscopic appendicectomy, which is consistent with the typical clinical presentation described in the literature.[2,3] Post-extubation laryngospasm was the most common precipitating factor, while transient upper airway obstruction due to vigorous inspiratory efforts and endotracheal tube biting also contributed in individual cases. These observations support previous studies identifying upper airway obstruction as the primary initiating event in most cases of NPPE.[2,3]

The pathophysiological mechanism involves the generation of markedly negative intrapleural pressures, often reaching −50 to −100 cmH_2O, during attempts to inhale against an obstructed airway. This results in increased venous return, elevated pulmonary blood volume, increased left ventricular afterload, and disruption of the alveolar-capillary barrier, allowing fluid to accumulate within the alveolar spaces. In addition, hypoxia-induced sympathetic activation further elevates pulmonary vascular pressures, thereby aggravating pulmonary edema.[1,4] The rapid progression from airway obstruction to severe hypoxemia emphasizes the importance of continuous monitoring during emergence from anesthesia.

The diagnosis of NPPE is primarily clinical and should be suspected in patients who develop sudden hypoxemia, tachypnea, bilateral crackles, and pink frothy sputum immediately after extubation.[2,6] Chest radiography typically reveals bilateral diffuse alveolar infiltrates without cardiomegaly, while arterial blood gas analysis demonstrates varying degrees of hypoxemia. In all of our cases, radiological findings were consistent with non-cardiogenic pulmonary edema, and cardiac causes were excluded through clinical assessment and echocardiography where indicated. Differential diagnoses such as aspiration pneumonitis, pulmonary embolism, fluid overload, acute respiratory distress syndrome, and cardiogenic pulmonary edema were carefully considered before confirming the diagnosis.[2,6]

Young, healthy adults are reported to be at increased risk because they are capable of generating strong inspiratory efforts during acute airway obstruction.[3] However, obesity, obstructive sleep apnea, difficult airway management, recent upper respiratory tract infection, excessive airway secretions, inadequate reversal of neuromuscular blockade, repeated extubation attempts, and upper airway surgery are recognized predisposing factors.[2,5] One patient in our series was obese, while the remaining patients were young adults without significant comorbidities, illustrating that NPPE may occur even in otherwise healthy individuals.

Management of NPPE primarily focuses on rapid restoration of airway patency and optimization of oxygenation. Immediate administration of 100% oxygen, application of continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP), and re-intubation with mechanical ventilation in severe cases remain the cornerstone of treatment.[2,7] Although intravenous diuretics such as furosemide are frequently administered, their benefit remains controversial because NPPE is primarily caused by hydrostatic forces rather than fluid overload.[1,7] Nevertheless, they may be considered selectively after ensuring adequate intravascular volume. In our series, patients managed with prompt airway stabilization and ventilatory support showed rapid clinical improvement, with complete radiological recovery occurring within 24–48 hours, consistent with previous reports.[2,7]

Preventive strategies are equally important in reducing the incidence of NPPE. Adequate suctioning of airway secretions, complete reversal of neuromuscular blockade, extubation at an appropriate depth of anesthesia, gentle airway manipulation, early recognition and treatment of laryngospasm, and careful postoperative monitoring of high-risk patients are essential preventive measures.[2,5,7] Immediate intervention during the first signs of upper airway obstruction may prevent progression to pulmonary edema and reduce the need for intensive care admission.[7]

The principal limitation of this case series is the small number of patients and its descriptive nature, which limits generalizability. However, the consistent clinical presentation, radiological findings, favorable response to supportive therapy, and complete recovery in all patients reinforce the characteristic features of NPPE.[6,7] Reporting such cases contributes to greater awareness among anesthesiologists, surgeons, and perioperative physicians and highlights the importance of early diagnosis and prompt management.[8]

Overall, this case series demonstrates that post-extubation negative pressure pulmonary edema, although rare following laparoscopic appendicectomy, should always be considered in patients who develop acute respiratory distress immediately after extubation. Early recognition, prompt airway management, and appropriate ventilatory support remain the key determinants of favorable outcomes.[2,7,8]

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.

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