CASE REPORT

The Airway That Refused To Open: A Case Report Of Idiopathic Bilateral Vocal Cord Abductor Palsy Complicated By Negative-Pressure Pulmonary Edema And Acute Respiratory Distress Syndrome

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ABSTRACT

Background: Unexpected airway obstruction in the peri-extubation period can result in significant morbidity and may be caused by rare, unrecognized laryngeal pathologies. Negative-pressure pulmonary edema (NPPE) is a serious complication of acute upper airway obstruction and may progress to acute respiratory distress syndrome (ARDS) if severe or prolonged.

Case Presentation: A 35-year-old obese female with a history of childhood poliomyelitis who underwent elective lumbar decompression and fusion surgery under general anesthesia. Preoperative assessment revealed multiple predictors of difficult airway, including Mallampati grade IV, short neck, and restricted neck mobility. Mask ventilation and tracheal intubation were difficult but successfully managed intraoperatively. The patient was extubated after fulfilling standard criteria; however, within one hour postoperatively, he developed acute respiratory distress with inspiratory stridor and rapid desaturation. Despite initial supportive measures, hypoxemia persisted, necessitating re-intubation. Chest radiography revealed bilateral diffuse alveolar infiltrates with a normal cardiac silhouette, consistent with non-cardiogenic pulmonary edema. The clinical course progressed to severe hypoxemic respiratory failure fulfilling criteria for ARDS, requiring invasive mechanical ventilation with high positive end-expiratory pressure and 100% inspired oxygen. Subsequent laryngoscopic evaluation revealed idiopathic bilateral vocal cord abductor palsy. This case highlights a rare and life-threatening sequence of perioperative events wherein unrecognized bilateral vocal cord abductor palsy led to acute airway obstruction, NPPE, and ARDS. Early recognition of post-extubation respiratory distress, prompt airway control, and aggressive supportive management are essential to prevent progression and ensure favorable outcomes.

Keywords: Difficult airway; Bilateral vocal cord abductor palsy; Negative-pressure pulmonary edema; acute respiratory distress syndrome; Post-extubation airway obstruction; Laryngospasm; Mechanical ventilation; High positive end-expiratory pressure; Non-cardiogenic pulmonary edema

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INTRODUCTION

Airway management remains a cornerstone of safe anaesthetic practice, and unanticipated difficult airway continues to be a significant cause of perioperative morbidity and mortality. While most airway challenges can be predicted through careful pre-anaesthetic assessment, certain conditions remain clinically silent until a critical event occurs [1].

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Among these, vocal cord dysfunction—particularly bilateral vocal cord abductor palsy—represents a rare but potentially catastrophic cause of acute airway obstruction that may manifest suddenly during induction, extubation, or in the immediate postoperative period [2].

Bilateral vocal cord abductor palsy results from impaired function of the posterior cricoarytenoid muscles, the sole abductors of the vocal cords, leading to a fixed or near-fixed paramedian position of the cords. This condition may be congenital or acquired and is most commonly associated with thyroid surgery, malignancy, trauma, neurological disorders, or prolonged intubation [3]. Idiopathic bilateral vocal cord abductor palsy is exceedingly rare and often remains asymptomatic until increased ventilatory demand or airway manipulation unmasks the obstruction. Because phonation may remain relatively preserved, the diagnosis is frequently delayed or overlooked in the perioperative setting [4]. Acute upper airway obstruction in the peri-extubation period can precipitate a cascade of life-threatening events. One such consequence is negative-pressure pulmonary edema (NPPE), a form of non-cardiogenic pulmonary edema caused by forceful inspiratory efforts against an obstructed airway [5]. The markedly negative intrathoracic pressures generated increase venous return and pulmonary capillary transudation, resulting in rapid alveolar flooding. NPPE typically presents with sudden hypoxemia, pink frothy sputum, bilateral crackles on auscultation, and diffuse alveolar infiltrates on chest imaging. Although often reversible with prompt recognition and supportive management, severe cases may progress to acute respiratory distress syndrome (ARDS) [6].

ARDS is characterized by acute onset hypoxemia, bilateral pulmonary infiltrates, and reduced lung compliance in the absence of cardiac failure or fluid overload. In the perioperative context, ARDS remains a feared complication, associated with prolonged mechanical ventilation, increased intensive care unit stay, and significant mortality [7]. Airway obstruction-induced lung injury, including NPPE evolving into ARDS, underscores the importance of early recognition and aggressive supportive management, including lung-protective ventilation strategies [8].

Patients with prior neurological disorders, such as poliomyelitis, may have subclinical involvement of bulbar or laryngeal muscles, potentially predisposing them to airway compromise. In addition, obesity, restricted neck mobility, and unfavorable airway anatomy further compound the difficulty in airway management [9]. When such risk factors coexist with an undiagnosed dynamic or fixed laryngeal pathology, routine airway management may rapidly escalate into a “cannot ventilate, cannot oxygenate” scenario [10].

This case report highlights a rare and challenging presentation of idiopathic bilateral vocal cord abductor palsy in a patient with multiple predictors of difficult airway, leading to peri-extubation airway obstruction, negative-pressure pulmonary edema, and subsequent ARDS. The case underscores the limitations of conventional airway assessment, the need for heightened vigilance during extubation, and the critical role of preparedness, multidisciplinary coordination, and prompt intervention in managing unexpected airway catastrophes.

CASE PRESENTATION

Patient Information: A 35-year-old female with a known history of childhood poliomyelitis presented with complaints of chronic low back pain and progressive difficulty in walking for one year. He had residual right upper limb weakness attributable to poliomyelitis. The patient was obese and a known hypertensive on regular medication. There was no prior history of dyspnea, stridor, voice change, dysphagia, sleep-disordered breathing, or previous airway interventions. He was planned for elective lumbar decompression and fusion surgery following radiological confirmation of L4–L5 and L5–S1 prolapsed intervertebral discs.

Pre-anaesthetic Evaluation: Preoperative airway assessment revealed multiple predictors of difficult airway. The patient had obesity with a short neck, Mallampati grade IV airway, limited mouth opening, restricted neck extension, and presence of acanthosis nigricans with excessive buccal and cervical adiposity. Thyromental distance was reduced. Cardiovascular and respiratory system examinations were unremarkable. Baseline oxygen saturation on room air was 98%. Routine hematological and biochemical investigations, electrocardiography, and preoperative chest radiograph were within normal limits. The patient was classified as American Society of Anesthesiologists (ASA) physical status II.

Anaesthetic Induction and Intraoperative Course: Standard monitoring including electrocardiography, non-invasive blood pressure, pulse oximetry, and capnography was instituted. Anaesthesia was induced with intravenous

glycopyrrolate 0.2 mg, midazolam 2 mg, fentanyl 100 µg, and propofol 100 mg. Following induction, mask ventilation was difficult with poor chest rise and rapidly falling oxygen saturation, which dropped to 40%. Oxygenation improved to approximately 80% with airway maneuvers, jaw thrust, and two-handed mask ventilation with an oral airway.

Direct laryngoscopy revealed poor glottic visualization. An initial attempt at bougie-guided intubation using a 7.5 mm flexometallic endotracheal tube was unsuccessful. A subsequent attempt using a 7.0 mm cuffed endotracheal tube over a bougie was successful, following which oxygen saturation improved to 95%. Endotracheal tube placement was confirmed with capnography and bilateral chest auscultation. Anaesthesia was maintained with inhalational agents and neuromuscular blockade. The surgical procedure was completed uneventfully, and intraoperative oxygenation remained stable.

Extubation and Postoperative Airway Complication: At the conclusion of surgery, the patient was extubated after fulfilling standard extubation criteria, including adequate spontaneous ventilation, protective airway reflexes, and acceptable oxygen saturation on room air. However, within one hour of extubation in the postoperative recovery area, the patient developed acute respiratory distress characterized by inspiratory stridor, tachypnea, use of accessory muscles, and rapid oxygen desaturation to 80%.

Acute upper airway obstruction due to laryngospasm was suspected. Continuous positive airway pressure with 100% oxygen was initiated along with airway maneuvers. Intravenous bronchodilators and corticosteroids were administered; however, the patient continued to exhibit severe respiratory distress with inadequate oxygenation. Given worsening hypoxemia, bougie-assisted re-intubation was performed with difficulty using a 6.5 mm cuffed endotracheal tube. Post-intubation auscultation revealed bilateral coarse breath sounds with rhonchi.

Radiological Findings (Image Description): A portable anteroposterior chest radiograph obtained immediately after re-intubation demonstrated bilateral, diffuse, fluffy alveolar opacities involving both lung fields, with relative central and peripheral distribution. The cardiac silhouette was normal in size, and there were no features suggestive of cardiogenic pulmonary edema or pleural effusion. The radiographic appearance was consistent with acute non-cardiogenic pulmonary edema.

A subsequent chest X-ray showed progression of bilateral air-space opacities with worsening lung involvement, correlating with persistent hypoxemia despite high inspired oxygen concentration. These radiological findings, in conjunction with the clinical scenario of acute upper airway obstruction followed by severe hypoxemic respiratory failure, were consistent with negative-pressure pulmonary edema progressing to acute respiratory distress syndrome (ARDS).

First ICU Admission (Postoperative Period – First Event): The patient was transferred to the intensive care unit for one day for further monitoring and ventilatory support. After stabilization, she showed clinical improvement and was planned for extubation.

Planned Extubation Failure and Tracheostomy (Second Event): During planned extubation from the OT, the patient again developed a similar episode of acute respiratory distress with rapid desaturation, consistent with recurrent upper airway obstruction. Endotracheal reintubation was performed with 6.5mm ETT , and otolaryngology consultation was obtained. In view of repeated extubation failure and suspected laryngeal pathology, an emergency tracheostomy was performed. Following tracheostomy, the endotracheal tube was removed. Subsequently, fibreoptic evaluation using an ambuscope was carried out through the upper airway, which revealed bilateral vocal cord abductor palsy, with both vocal cords fixed in a median/paramedian position and minimal abduction during inspiration, as demonstrated in the recorded images. There was no evidence of trauma, edema, mass lesion, or surgical injury. Neurological evaluation and imaging did not reveal any central cause, and in the absence of an identifiable etiology, the diagnosis of idiopathic bilateral vocal cord abductor palsy was established.

Second ICU Admission: Post-Tracheostomy Negative-Pressure Pulmonary Edema and Ventilatory Management: Following tracheostomy, the patient developed worsening hypoxemia. A post-tracheostomy chest radiograph revealed bilateral air-space opacities consistent with negative-pressure pulmonary edema secondary to acute upper airway obstruction. The patient was readmitted to the intensive care unit and managed with deep sedation and invasive mechanical ventilation.

Due to refractory hypoxemia, ventilatory support was escalated to a lung-protective strategy with high positive end-expiratory pressure (PEEP up to 14 cmH_2O) and an inspired oxygen fraction of 100%. Despite optimization of ventilatory parameters, oxygenation initially remained impaired, fulfilling the Berlin criteria for severe acute respiratory distress syndrome. Hemodynamic parameters remained stable throughout, with no evidence of fluid overload or cardiac dysfunction.

Outcome and Follow-up: With continued supportive care, sedation, and lung-protective ventilation, the patient demonstrated gradual clinical improvement. Oxygenation parameters improved progressively, and follow-up chest radiographs showed resolution of bilateral alveolar infiltrates. The patient was successfully weaned from mechanical ventilation and decannulated after stabilization. She was discharged from the intensive care unit after 5 days in stable condition and subsequently discharged from hospital with advice for regular otolaryngology follow-up and long-term airway surveillance.

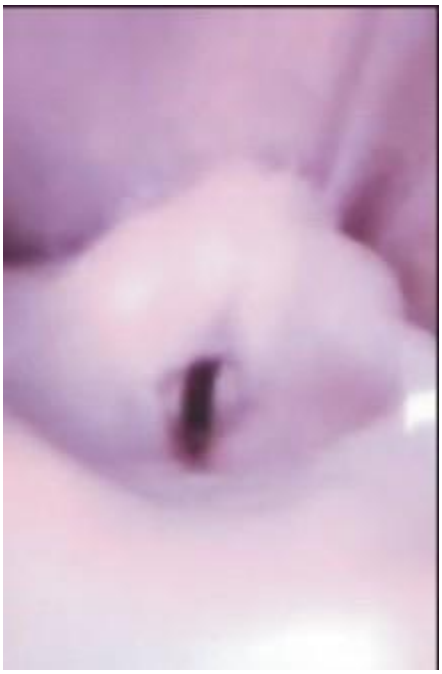

Figure 1. Direct videolaryngoscopic view demonstrating bilateral vocal cord abductor palsy.

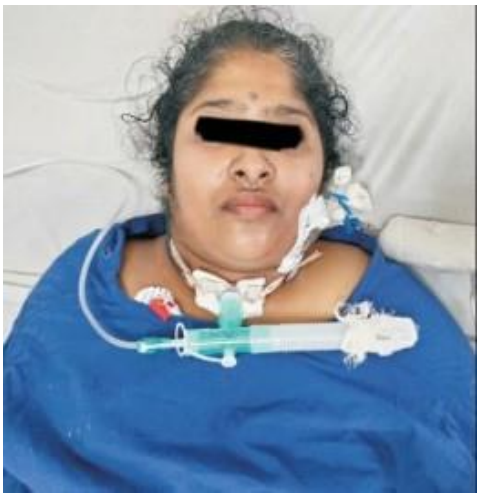

Figure 2. Patient following emergency tracheostomy for recurrent post-extubation airway obstruction

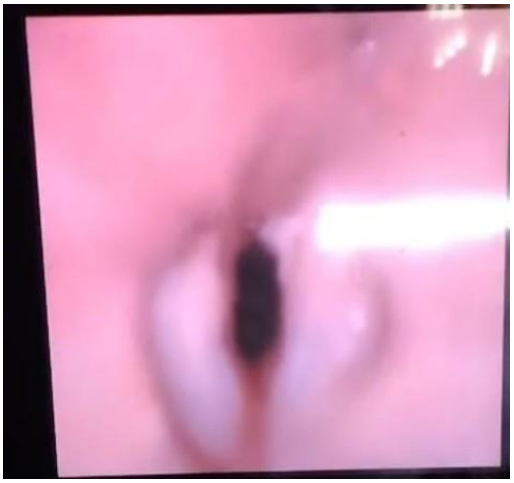

Figure 3. Fibreoptic ambuscope view of the larynx after tracheostomy showing bilateral vocal cord abductor palsy

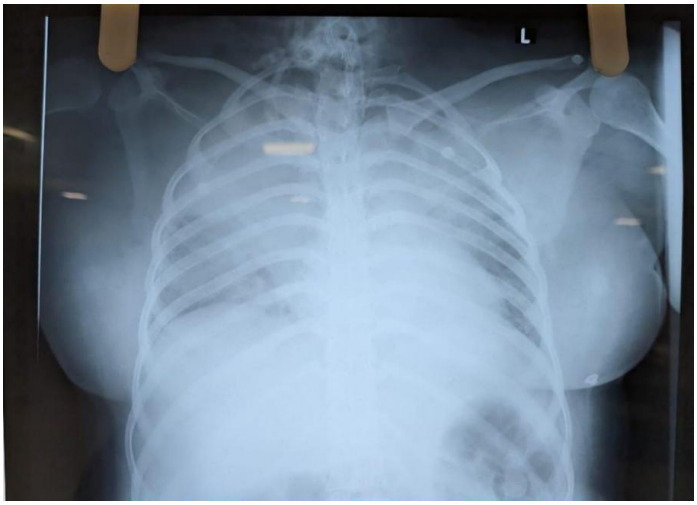

Figure 4. Post-tracheostomy chest radiograph

demonstrating acute respiratory distress syndrome

DISCUSSION

This case highlights a rare but potentially catastrophic perioperative airway complication in the form of idiopathic bilateral vocal cord abductor palsy, leading to acute upper airway obstruction, negative-pressure pulmonary edema (NPPE), and subsequent progression to acute respiratory distress syndrome (ARDS). The case underscores the limitations of conventional airway assessment, the challenges of extubation in high-risk patients, and the importance of early recognition and aggressive management of post-obstructive pulmonary complications.

Bilateral vocal cord abductor palsy is an uncommon condition characterized by impaired function of the posterior cricoarytenoid muscles, resulting in fixed or near-fixed vocal cords in a paramedian position. While phonation may be relatively preserved, inspiratory airflow is significantly compromised [3]. Most cases are secondary to thyroid surgery, malignancy, trauma, prolonged intubation, or central neurological pathology. Idiopathic cases are exceedingly rare and are often diagnosed only after an episode of acute airway obstruction [11]. In the present case, the absence of preoperative symptoms such as stridor or voice change likely contributed to the delayed diagnosis. Additionally, the patient's history of poliomyelitis raises the possibility of subclinical involvement of bulbar or laryngeal musculature, potentially predisposing him to airway compromise under stress.

The peri-extubation period is particularly vulnerable to airway-related adverse events, especially in patients with difficult airway predictors such as obesity, limited neck mobility, and high Mallampati grade [12]. Although the patient fulfilled standard extubation criteria, the development of inspiratory stridor and rapid desaturation within one hour strongly suggests dynamic upper airway obstruction rather than residual anesthetic effect. In such scenarios, bilateral vocal cord dysfunction should be considered as a differential diagnosis, particularly when conventional measures for laryngospasm fail to improve oxygenation [13].

Negative-pressure pulmonary edema is a well-recognized but underdiagnosed consequence of acute upper airway obstruction. It results from forceful inspiratory efforts against an occluded airway, generating markedly negative intrathoracic pressures. This leads to increased venous return, elevated pulmonary capillary hydrostatic pressure, and disruption of the alveolar-capillary membrane, culminating in rapid transudation of fluid into the alveolar spaces [14]. Radiologically, NPPE manifests as bilateral diffuse alveolar opacities with a normal cardiac silhouette, as seen in the chest radiographs of the present case. The rapid onset following airway obstruction and absence of cardiac dysfunction strongly support the diagnosis of NPPE.

While NPPE is often self-limiting with prompt airway relief and supportive care, severe cases may progress to ARDS, as observed in this patient. The evolution from NPPE to ARDS likely reflects sustained hypoxemia, ongoing alveolar injury, and inflammatory cascade activation [14]. The diagnosis of ARDS was supported by persistent hypoxemia requiring high inspired oxygen concentrations, bilateral pulmonary infiltrates on imaging, reduced lung compliance, and the absence of cardiac failure or fluid overload. This progression emphasizes the need for close monitoring and early escalation of respiratory support in patients with post-obstructive pulmonary edema [7].

Management of such patients requires a multidisciplinary approach involving anesthesiologists, intensivists, and otolaryngologists. Early re-establishment of a patent airway is paramount [15]. Mechanical ventilation using lung-protective strategies, including low tidal volumes and appropriate positive end-expiratory pressure (PEEP), remains the cornerstone of ARDS management [16]. In this case, the requirement of high PEEP (up to 14 cmH_2O) and FiO_2 100% reflects the severity of lung injury. Timely ICU admission and controlled ventilation were crucial in achieving clinical recovery.

This case also highlights the importance of thorough postoperative airway evaluation in patients with unexplained respiratory distress. Direct laryngoscopy played a key role in diagnosing bilateral vocal cord abductor palsy, allowing appropriate planning for airway management and follow-up. From a preventive standpoint, heightened vigilance during extubation, consideration of delayed or staged extubation, and availability of advanced airway equipment are essential in patients with difficult airway predictors.

In conclusion, idiopathic bilateral vocal cord abductor palsy is a rare but life-threatening cause of perioperative airway obstruction. When unrecognized, it may precipitate negative-pressure pulmonary edema and progress to ARDS. Early

recognition, prompt airway control, and aggressive supportive management are critical to improving outcomes [4]. This case reinforces the need for a high index of suspicion for occult laryngeal pathology in patients with unexpected post-extubation respiratory compromise.

CONCLUSION

This case underscores the potential for rare and unanticipated airway pathology to result in severe perioperative respiratory complications. Idiopathic bilateral vocal cord abductor palsy may remain clinically silent and escape routine preoperative evaluation, yet can precipitate acute upper airway obstruction in the peri-extubation period. The resultant forceful inspiratory efforts against an obstructed airway can rapidly lead to negative-pressure pulmonary edema, which, if severe or prolonged, may progress to acute respiratory distress syndrome. The presence of multiple difficult airway predictors further compounds the risk and challenges airway management. Early recognition of post-extubation respiratory distress, prompt re-establishment of airway patency, and timely initiation of lung-protective ventilation strategies are crucial for favorable outcomes. This case highlights the importance of heightened vigilance during extubation in high-risk patients, consideration of underlying laryngeal dysfunction when conventional measures fail, and the value of multidisciplinary management. Awareness of this rare but life-threatening sequence can aid anesthesiologists and intensivists in preventing delays in diagnosis and improving perioperative patient safety.

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