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CASE REPORT

Tracheal Stenosis in Neuro Myelitis Optica Spectrum Disorders: Airway Management in the ICU

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ABSTRACT

Background: Neuro Myelitis Optica Spectrum Disorder (NMOSD) attacks can be life-threatening, leading to respiratory failure requiring orotracheal intubation (OTI). Patient with a history of prolonged and repeated intubation in the ICU may develop post-intubation tracheal stenosis (PITS)

Case: A woman, 32 years old, with NMOSD came to the emergency department complaining dyspnea, productive cough, wheezing and low oxygen levels. She was intubated using an uncuff endotracheal tube (ETT) size. 5. Subsequently, an inadvertent dislodgement of the ETT in the intensive care unit (ICU). A Thoracic CT imaging showed tracheal narrowing at the thoracic 1-2 level, approximetely 58% of the lumen width. A Multidisciplinary case conference was convened to plan balloon dilatation bronchoscopy with a bedside tracheostomy as a backup crash airway protocol.

Discussion: The patient experienced ETT dislodgement, prompting reintubation with ETT cuffs number 4 and 6 using a video laryngoscope and an adult bougie. Reintubation with a larger ETT size was is necessitated by reduced mucosal edema following adequate steroid, inhalation, and antibiotic therapy. Balloon dilatation bronchoscopy of mucosal stenosis via LMA was successfully performed, followed by intubation using ETT cuff no. 8 with guided bronchoscopy. The patient was successfully weaned from mechanical ventilation with a leak test before extubation.

Conclusion: Airway management by considering the location and degree of stenosis as well as the patient's general condition. Balloon dilatation with bronchoscopy offers good results in patients with tracheal stenosis who are not eligible for surgery.

Keywords: Post-Intubation Tracheal Stenosis, Neuro Myelitis Optica Spectrum Disorder (NMOSD), intubation, balloon dilatation bronchoscopy, mechanical ventilation

INTRODUCTION

Tracheal stenosis is a rare yet potentially life-threatening condition that may result from congenital abnormalities, post-intubation injury, trauma, primary tracheal tumors, or extrinsic tumor compression of the trachea. Although the exact prevalence remains uncertain, the estimated incidence of post-intubation tracheal stenosis is approximately 4.9 cases per million population per year.[1] Post-intubation tracheal stenosis most commonly occurs in critically ill patients requiring prolonged mechanical ventilation.[2] The etiology of tracheal stenosis can be broadly classified into congenital, idiopathic, and acquired causes. Acquired tracheal stenosis represents the most common category in adults and is typically associated with airway trauma, with the majority of cases related to endotracheal intubation. Contributing factors include the duration of intubation, excessive endotracheal cuff pressure, cuff movement, hypotension, infection, and other comorbid conditions.[2]

Autoimmune inflammatory diseases constitute another potential cause of tracheal stenosis.[3] One such disorder that may lead to respiratory failure is Neuromyelitis Optica Spectrum Disorder (NMOSD), a rare autoimmune condition affecting the central nervous system (CNS). Initially recognized as a disease limited to the optic nerves and spinal cord, its clinical spectrum has expanded to include involvement of the area postrema, brainstem, diencephalon, and cerebral hemispheres.[4] NMOSD attacks can be life-threatening and may precipitate respiratory failure, necessitating orotracheal intubation (OTI) in the intensive care unit (ICU). Without immunosuppressive therapy, approximately one-third of NMOSD cases may result in severe disability, relapse, or death, frequently due to respiratory failure.[4]

Anesthetic management particularly airway management in patients with tracheal stenosis presents significant challenges for anesthesiologists. The approach depends on the severity and location of the stenosis, as well as the type of surgical procedure planned. Various airway management strategies have been described, including face mask ventilation, laryngeal mask airway (LMA), tracheal intubation, cardiopulmonary bypass, and extracorporeal membrane oxygenation (ECMO).[1] In this case report, we present a patient with severe tracheal stenosis requiring a strategic airway management approach in the ICU.

CASE

A 32-year-old woman presented to the emergency department (ED) with progressive dyspnea. Her symptoms had worsened over the preceding two days prior to admission, accompanied by productive cough with difficulty expectorating sputum and wheezing. According to her family, two days before admission her oxygen saturation remained between 97–99% on room air. Prior to ED presentation, her oxygen saturation decreased to 89% despite supplemental oxygen therapy. The dyspnea was more pronounced in the morning.

The patient was bedridden in her daily activities. She denied fever, headache, new-onset focal neurological deficits, or worsening of other neurological symptoms. The patient had a known history of Neuromyelitis Optica Spectrum Disorder Aquaporin-4 immunoglobulin G (NMOSD AQP4-IgG), diagnosed in January 2024. Initial symptoms included blurred vision, lower extremity weakness, numbness, a burning sensation extending from the feet to the neck, paresthesia, and intermittent episodes of cough and dyspnea since August 2023.

In October 2023, she was admitted to another hospital and underwent lumbar puncture and spinal magnetic resonance imaging (MRI) with an initial suspicion of Guillain–Barré syndrome. She required intensive care unit (ICU) admission with mechanical ventilation for three days due to respiratory distress. She received intravenous methylprednisolone 125 mg four times daily. Subsequently, she developed worsening bilateral lower limb weakness progressing to complete paralysis and urinary retention requiring catheterization. After clinical improvement, she was discharged home. Despite discharge, the patient continued to experience recurrent cough and dyspnea, requiring home oxygen supplementation. During home care, her dyspnea progressively

worsened despite oxygen therapy. She reported difficulty expectorating sputum and chest pain radiating to the toes. Consequently, she presented to Cipto Mangunkusumo Hospital in January 2024.

During hospitalization from January to March 2024 at RSCM, her respiratory symptoms persisted. Upon re-evaluation, she was diagnosed with NMOSD AQP4-IgG. She underwent therapeutic plasma exchange (TPE) once using 3.5 bottles of 5% albumin and received high-dose methylprednisolone. Hypotension occurred during TPE and was managed with 1,000 mL of 0.9% sodium chloride, stabilizing her blood pressure at 100/60 mmHg after an episode of hypovolemic shock. Following TPE, numbness improved from the breast to the umbilical level, and the burning sensation became milder. Episodes of chest tightness associated with dyspnea decreased in frequency and severity, allowing improved sleep. However, blurred vision, weakness, paresthesia, and numbness below the umbilicus persisted. One week later, a second TPE session was performed. Although hypotension did not recur during the procedure, she developed fever, dyspnea, and wheezing afterward. Another hypotensive episode occurred and was managed with a 500 mL fluid bolus of 0.9% sodium chloride, resulting in hemodynamic improvement. She also experienced hematochezia, prompting temporary discontinuation of methylprednisolone.

During her hospitalization at RSCM, the patient required intubation for one week. Although her condition initially improved, she subsequently deteriorated due to an infectious focus at the femoral central venous catheter insertion site and phlebitis. After receiving appropriate antimicrobial therapy, her clinical status improved, vital signs stabilized, fever resolved, dyspnea decreased, and sleep quality improved. However, neurological deficits showed no significant recovery, and rituximab therapy was planned. After nearly two months of hospitalization, her respiratory symptoms partially improved, and she was discharged home on 2 L/min oxygen supplementation with outpatient plans for rituximab therapy. Shortly thereafter, she was readmitted due to recurrent worsening dyspnea and wheezing. She received one cycle of rituximab (March 30, 2024) and underwent laryngoscopic evaluation. Her respiratory symptoms improved and were controlled during admission. An interdisciplinary intervention meeting was conducted on April 4, 2024, after imaging revealed tracheal narrowing at the T1–T2 vertebral level with thickened tracheal walls. Outpatient tracheal stenting or balloon dilatation was planned by the pulmonology team. She was discharged once clinically stable.

On April 8, 2024, the patient was readmitted with worsening dyspnea for two days prior to admission. In the ED, she was alert and oriented but appeared dyspneic, with difficulty expectorating sputum, particularly while speaking. Visual disturbance and numbness persisted. She remained catheterized and had fecal incontinence. Two hours after ED arrival, dyspnea recurred despite oxygen saturation above 95%, with a respiratory rate of 30 breaths per minute. Copious secretions were suctioned without improvement. Nebulized bronchodilator therapy resulted in transient relief; however, she later developed oxygen desaturation to 86–88% despite 15 L/min oxygen via mask. Endotracheal intubation was performed. Multiple endotracheal tube (ETT) sizes were attempted, but larger sizes could not be advanced into the trachea. Ultimately, a size 5 uncuffed ETT was successfully inserted and connected to mechanical ventilation. Two hours later, desaturation occurred due to ETT dislodgement, requiring re-intubation with the same size tube. Deep sedation was initiated.

Upon transfer to the ICU, the ETT was again dislodged and replaced. A size 4 cuffed ETT was successfully inserted. Fiberoptic bronchoscopy via the ETT failed to provide adequate visualization. The patient was ventilated using pediatric mechanical ventilation settings. Peak airway pressure (Ppeak) was elevated (25–29 cmH_2O) with low tidal volumes (4–6 mL/kg). To prevent further tube displacement, deep sedation was maintained using midazolam, morphine, and dexmedetomidine. Urgent consultations with pulmonology, otolaryngology, and thoracic

Figure 1. Ventilator screen display while the patient was intubated with a size 4 cuffed endotracheal tube (pediatric ventilation mode, elevated peak airway pressure, and low tidal volume

surgery were requested to consider emergency tracheostomy. However, tracheostomy was deferred as the airway was deemed secured with the ETT.

Dexamethasone 5 mg intravenously three times daily was administered due to suspected tracheal mucosal edema, along with inhaled bronchodilators and mucolytics. A thoracic computed tomography (CT) scan with three-dimensional airway reconstruction demonstrated tracheal stenosis at the T1–T2 vertebral level, with approximately 58% luminal narrowing over a length of 2.3 cm, suspicious for tracheal stricture. During ICU care, the patient remained intermittently agitated and hypercapnic, with end-tidal carbon dioxide ($ETCO_2$) levels reaching 50–60 mmHg. Sedation was escalated with morphine (2 mg/hour), midazolam (3–5 mg/hour), dexmedetomidine (0.3 µg/kg/hour), propofol (40–60 mg/hour), and haloperidol (1 mg orally twice daily, with additional 2.5 mg intramuscularly as needed). Ventilator-associated pneumonia (VAP) was suspected due to copious purulent secretions and elevated inflammatory markers. Sputum culture isolated Acinetobacter species. Antibiotic therapy with meropenem (1 g intravenously three times daily) and tigecycline (loading dose 100 mg intravenously, followed by 50 mg intravenously twice daily) was initiated. Hemodynamic instability required vasopressor support with norepinephrine up to 0.5 µg/kg/min and dobutamine 5 µg/kg/min.

On ICU day 6, a multidisciplinary case conference involving anesthesiology, radiology, pulmonology, otolaryngology, thoracic surgery, infectious disease, and neuroinfection specialists was conducted. The plan was bedside bronchoscopy under operating room–level preparation using a laryngeal mask airway (LMA), followed by balloon dilatation and intralesional triamcinolone injection by pulmonology, with emergency tracheostomy backup by otolaryngology. The patient's family was counseled regarding risks and benefits. On

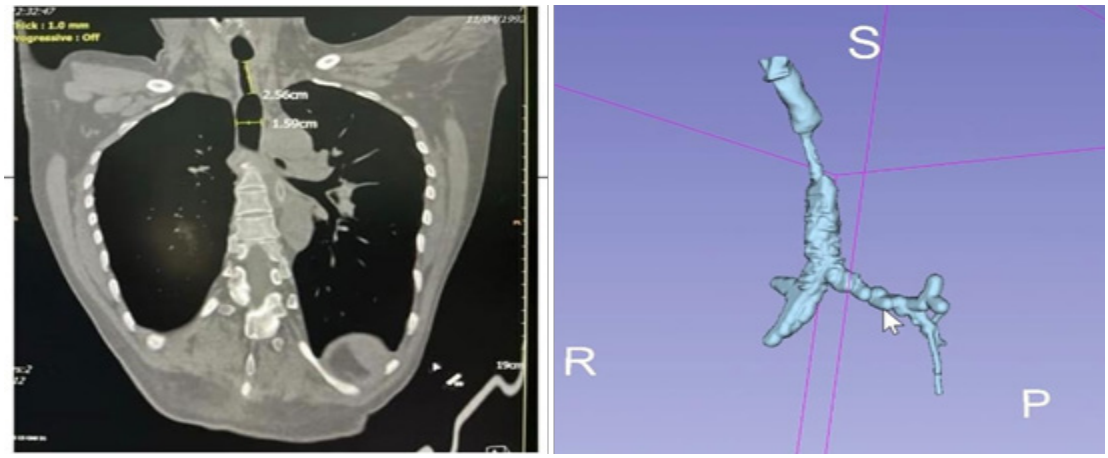

Figure 2. Thoracic computed tomography (CT) scan (left) with three-dimensional airway reconstruction (right) demonstrating tracheal stenosis at the T1–T2 vertebral level, with approximately 58% luminal narrowing over a length of 2.3 cm

ICU day 9, suspected ETT dislodgement led to inadequate tidal volume delivery. Re-intubation using a video laryngoscope and adult bougie was successfully performed with a size 6 cuffed ETT. After replacement, tidal volume improved, Ppeak decreased to 19–21 cmH_2O, and $ETCO_2$ normalized.

On ICU day 11, balloon dilatation was performed by the pulmonology team under crash airway protocol with tracheostomy backup. Bronchoscopy via LMA revealed glottic edema and tracheal stenosis approximately 4 cm below the subglottis. The tracheal mucosa appeared hyperemic and edematous with white plaques. Brushing, balloon dilatation, and intralesional injection of 2 mL triamcinolone were performed. At the end of the procedure, a size 8 cuffed ETT was inserted under bronchoscopic guidance to a depth of 20 cm.

Subsequently, the patient's condition improved significantly. Dyspnea resolved, hemodynamics stabilized with minimal vasopressor support, and agitation subsided. Gradual weaning of sedation, vasopressors, and mechanical ventilation was undertaken. On ICU day 14, extubation was performed after a positive cuff leak test. Two days post-extubation, the patient remained hemodynamically stable and was transferred to the general ward for further management.

DISCUSSION

Post-intubation tracheal stenosis was first described in 1969, although it had been recognized earlier as a clinical entity by Abraham Colles in 1886. Colles reported four cases of tracheal stenosis among 57 patients who underwent tracheostomy for the treatment of diphtheria.[2] Post-intubation tracheal stenosis has been recognized as a rare but serious complication, most frequently occurring in critically ill patients requiring prolonged endotracheal intubation.[2] Despite advances in airway device technology and improvements in critical care management, the incidence of post-intubation tracheal stenosis in the ICU remains between 6% and 21%, although only 1–2% of patients develop clinically significant severe stenosis. The introduction of high-volume, low-pressure cuffed endotracheal tubes has significantly reduced the incidence to approximately 1%.[2,5] The affected tracheal segment is typically about 2 cm in length and commonly involves the anterior and lateral walls of the trachea, whereas the posterior membranous wall is relatively protected due to its ability to expand toward the esophagus.[2] Clinical manifestations usually develop gradually, typically 1–6 weeks after extubation.[5] In the present case, symptoms occurred approximately 4–6 weeks after ICU admission with mechanical ventilation. The patient experienced progressive dyspnea, stridor, cough, wheezing, and recurrent pneumonia, which are consistent with reported clinical features.[5]

Neuromyelitis Optica Spectrum Disorder (NMOSD) is a rare autoimmune inflammatory disorder of the central nervous system associated with aquaporin-4 immunoglobulin G (AQP4-IgG). Acute NMOSD-AQP4-IgG attacks may be life-threatening, particularly when brainstem or cervical spinal cord involvement leads to respiratory failure requiring orotracheal intubation (OTI) in the ICU. Without prompt immunosuppressive therapy—including high-dose intravenous corticosteroids and therapeutic plasma exchange (TPE)—patients are at risk of severe disability or death.[4]

In this case, the patient with NMOSD-AQP4-IgG presented with acute respiratory failure necessitating immediate OTI. She had received TPE and high-dose corticosteroid therapy. However, her history of repeated and prolonged ICU admissions with mechanical ventilation predisposed her to post-intubation tracheal stenosis. Patients with NMOSD involving cervical spinal cord and medullary lesions are particularly susceptible to respiratory compromise and may require repeated airway interventions.[4] Prolonged and repeated intubation, combined with external compression from a nasogastric tube, may increase mucosal pressure between the tracheal tube cuff and surrounding structures, thereby contributing to progressive tracheal injury. Sustained cuff pressure can compromise mucosal perfusion, leading to ischemia, fibrotic scar formation, and

subsequent stenosis.[2,6] Although high-volume, low-pressure cuffs reduce the risk, inadequate monitoring of cuff pressure may still result in tracheal wall injury.[6]

In the present case, thoracic computed tomography (CT) with three-dimensional airway reconstruction demonstrated tracheal stenosis at the T1–T2 vertebral level with approximately 58% luminal narrowing over a 2.3 cm segment, consistent with grade II stenosis (50–70% obstruction) according to the Cotton-Myer Grading System.[6] Initially, smaller endotracheal tubes were required due to the narrowed lumen. When a size 4 cuffed ETT was used, elevated peak airway pressures (25–29 cmH_2O) and low tidal volumes (4–6 mL/kg) were observed, resulting in hypercapnia and agitation. Deep sedation was therefore administered using continuous infusions of morphine 2 mg/hours, midazolam 3-5 mg/hours, dexmedetomidine 0,3-0,5 mg/kg/hours, and propofol 40-60 mg/hours, with adjunctive haloperidol 2,5 mg IM to minimize agitation and reduce the risk of tube dislodgement. Intracuff pressure was routinely monitored and maintained below 30 mmHg to prevent further ischemic injury.

The American Association for Respiratory Care recommends maintaining cuff pressures below 30 mmHg, as higher pressures may impair mucosal perfusion and contribute to fibrosis and progressive stenosis.[7] Adjunctive therapy included intravenous dexamethasone 3 x 5 mg IV to reduce mucosal edema, inhaled bronchodilators, mucolytics, and targeted antibiotic therapy (Meropenem 3 x 1 gr IV and Tygecyclin 2 x 50 mg IV) for ventilator-associated pneumonia caused by Acinetobacter species. Hemodynamic instability required vasopressor support, which influenced the decision to perform bedside bronchoscopy rather than transport the patient to the operating room.

On ICU day 6, a multidisciplinary case conference involving anesthesiology, pulmonology, otolaryngology, thoracic surgery, radiology, and infectious disease specialists determined that bedside bronchoscopic balloon dilatation was the most appropriate intervention. Bronchoscopic balloon dilatation was performed in consideration of the patient's grade II stenosis (50–70% luminal obstruction according to the Cotton-Myer Grading System).[8]

Interventional bronchoscopy is an established therapeutic option for non-resectable or selected cases of tracheal stenosis, with modalities including airway dilatation, fibrotic tissue ablation, intralesional steroid injection, and airway stenting.[9,10] Bronchoscopy was performed at the bedside due to the patient's unstable clinical condition, rendering her unsuitable for transport. She required vasopressor support with norepinephrine at 0.5 µg/kg/min and dobutamine at 5 µg/kg/min. In addition, bedside intervention minimized the risk of endotracheal tube (ETT) dislodgement during transfer to the operating room. The bronchoscopic procedure was planned using a laryngeal mask airway (LMA), as the patient was initially intubated with a size 4 ETT. Balloon dilatation and intralesional triamcinolone injection were performed by the pulmonology team, with an emergency ("crash airway") tracheostomy protocol prepared by the otolaryngology team as backup.

Two days prior to the planned procedure, inadequate tidal volume and suspected ETT displacement necessitated re-intubation using video laryngoscopy and a bougie, successfully placing a size 6 cuffed ETT. The improvement in airway edema likely permitted placement of a larger tube. After upsizing the ETT, tidal volume normalized, peak airway pressure decreased to 19–21 cmH_2O, and $ETCO_2$ returned to normal range, suggesting that size 6 represented the minimal adequate diameter to achieve effective ventilation.[6] The management of patients with post-intubation tracheal stenosis varies according to the location of the lesion, the severity of the stenosis, the initial precipitating airway injury, and the presence of comorbid conditions.

Definitive surgical management, such as tracheal resection with end-to-end anastomosis (REEA), is considered the gold standard for selected patients; however, it may not be feasible in unstable or complex cases.[9,10] Therefore, bronchoscopic balloon dilatation with intralesional triamcinolone injection was selected as a less invasive and immediately

feasible option. The bedside procedure was successfully performed via laryngeal mask airway (LMA) with tracheostomy backup under a crash airway protocol. Bronchoscopy revealed glottic edema and tracheal stenosis approximately 4 cm below the subglottis, with hyperemic and edematous mucosa and white plaques. Balloon dilatation, brushing, and intralesional triamcinolone injection were performed, followed by re-intubation with a size 8 cuffed ETT under bronchoscopic guidance. Following the intervention, oxygenation and ventilation improved significantly. The patient was gradually weaned from mechanical ventilation and successfully extubated. Recurrence of stenosis at the intervention site remains possible due to abnormal wound healing and fibrotic remodeling. Therefore, staged bronchoscopic interventions may be required, and tracheostomy should be considered as a long-term conservative option if repeated balloon dilatation fails to achieve sustained airway patency.[10]

CONCLUSION

Post-intubation injury represents one of the most common causes of tracheal stenosis, particularly in conditions such as Neuromyelitis Optica Spectrum Disorder (NMOSD), which may precipitate respiratory failure requiring orotracheal intubation (OTI). Airway management in patients with tracheal stenosis poses significant challenges for anesthesiologists and necessitates close multidisciplinary collaboration with pulmonology, otolaryngology, radiology, and surgical teams. The management of post-intubation tracheal stenosis varies according to the location and severity of the stenosis, the initial precipitating airway injury, and the presence of comorbid conditions. Bronchoscopic balloon dilatation represents an effective therapeutic option in selected patients with tracheal stenosis who are not suitable candidates for definitive surgical intervention.

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