

Acute Cricoarytenoid Joint Fixation Following Routine Intubation: A Case Report

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Prolonged intubation is a common cause of injury to the posterior larynx often resulting in cricoarytenoid joint (CAJ) fixation and posterior glottic stenosis (PGS). We present a case of respiratory failure due to acute bilateral CAJ fixation and PGS following only 2 days of intubation for routine cardiac surgery. A tracheostomy was placed due to critical airway obstruction. Clinicians should remain vigilant for laryngeal injury presenting as CAJ fixation and PGS. Prompt surgical consultation is advised as early intervention is associated with reduced morbidity. (A&A Practice. 2023;17:e01706.)

GLOSSARY

BMI = body mass index; **CAJ** = cricoarytenoid joint; **ETT** = endotracheal tube; **PGS** = posterior glottic stenosis; **POD** = postoperative day

The posterior glottic region includes the posterior third of the vocal folds, posterior commissure, interarytenoid muscles, cricoid lamina, cricoarytenoid joints (CAJs), arytenoid cartilage complex, and overlying mucosa.¹ Posterior glottic stenosis (PGS) is a laryngeal disorder, which occurs as a sequela of injury to this region. Causes of acquired PGS include prolonged intubation, autoimmune illness, trauma, and tumors.¹ Prolonged intubation remains the most cited etiology.²

Acquired PGS generally presents 4 to 8 weeks after extubation.³⁻⁶ Symptoms include progressive dysphonia, dyspnea, and stridor as ongoing joint fibrosis reduces normal vocal fold motion.^{2,3} Without treatment, PGS can lead to total laryngeal obstruction and respiratory failure.³

Here, we present a case of rapid acute bilateral CAJ fixation following 2 days of intubation for cardiac surgery. This case is of particular interest for anesthesiologists and intensivists who may encounter this as an etiology of respiratory distress and critical airway obstruction. Institutional review board approval was not required for generation of this case report. Written Health Insurance Portability and Accountability Act authorization was obtained from the patient.

CASE DESCRIPTION

A 54-year-old man with a known history of difficult laryngoscopy underwent an uncomplicated aortic valve repair

for heart failure in the setting of aortic regurgitation. Medical history otherwise included generalized anxiety disorder, gastroesophageal reflux disease, hypothyroidism, and hypertension. He was 177.8 cm tall, was 101.6 kg, had a body mass index (BMI) of 32.1, a Mallampati score of 4, and had decreased mouth opening, thick neck, and limited mandibular protrusion. Following airway topicalization with 4% lidocaine and under light remifentanyl sedation, he underwent an atraumatic awake fiberoptic orotracheal intubation with a 7.5-mm endotracheal tube (ETT) and was extubated postoperative day (POD) 2. He developed increasing dyspnea and stridor after extubation. Awake flexible laryngoscopy and a neck computed tomography scan demonstrated laryngeal edema but no specific laryngeal injury. His symptoms gradually worsened and were unresponsive to intravenous corticosteroids and inhaled racemic epinephrine. He required emergent reintubation on POD 10 due to hypoxemic respiratory failure with successful placement of a 7.0-mm standard ETT under videolaryngoscopy guidance. Due to the emergent nature, videolaryngoscopy was attempted, while the fiberoptic endoscope was being prepared, and was successful.

He underwent microlaryngoscopy after another failed extubation attempt on POD 12. Findings included glottic erythema, ulceration of bilateral medial arytenoid bodies, and restriction in passive CAJ motion (Figure 1). He was extubated in the operating room and immediately developed stridor, tachypnea, and respiratory distress. Flexible endoscopic laryngoscopy demonstrated bilateral vocal fold motion impairment. He was reintubated with videolaryngoscopy and returned to the intensive care unit.

Repeat microlaryngoscopy on POD 21 demonstrated significant passive and active CAJ mobility restriction. A surgical tracheostomy was placed due to severe PGS. He underwent debridement of laryngeal granulation tissue, posterior glottis balloon dilation, and triamcinolone injections at this time. He was discharged from the hospital with regular otolaryngology follow-up. Over the next 6 months, his symptoms improved, and he tolerated increasing

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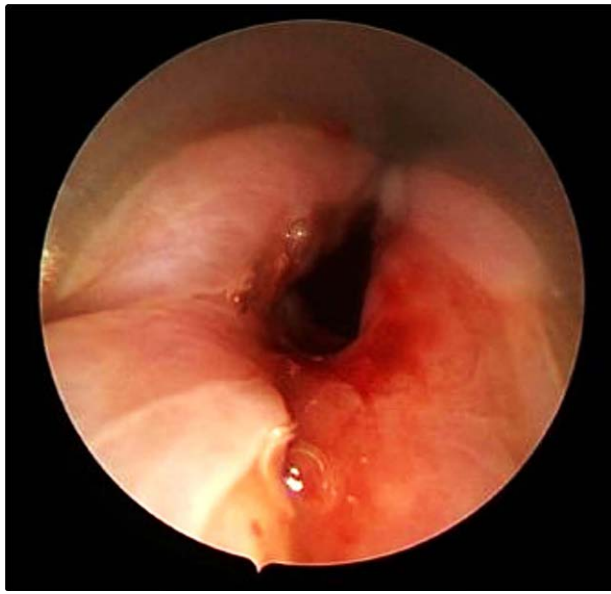


Figure 1. Stenotic, erythematous, and edematous posterior glottis after keyhole balloon dilation during the tracheostomy placement surgery. Ulceration and granulation tissues over the medial arytenoids bilaterally also seen.

intervals of tracheostomy capping trials (Figure 2). He was decannulated 6 months postoperatively without additional surgical interventions.

DISCUSSION

Prolonged intubation is the most cited etiology of adult-acquired PGS, developing in 5% of patients intubated between 5 and 10 days.² However, as this case demonstrates, severe PGS can occur after just 2 days of routine intubation. Even in the setting of atraumatic intubation, inflammatory processes due to the presence of ETT alone can result in severe PGS. This is the shortest period of intubation leading to class IV PGS and critical airway obstruction reported in literature up to this time. Risk factors for laryngotracheal stenotic lesions secondary to intubation include hypovolemic or shock states, gastroesophageal reflux disease, diabetes mellitus, elevated BMI, greater number of intubations, larger ETT size, and longer duration of intubation.^{7,8}

CAJ motion is essential for laryngeal functions of ventilation (vocal fold abduction), airway protection, and phonation (vocal fold adduction).^{1,9} ETTs traverse the posterior glottis, applying pressure to sensitive posterior glottic

mucosa as they become coaxial with the trachea.¹⁰ The CAJs are particularly vulnerable, as only a few millimeters of fragile mucosa separate the joint space from the laryngeal lumen.⁹ This pressure leads to mucosal ischemia, ulceration, and ultimately fibrosis compromising the normal CAJ motion.¹ The resulting vocal fold motion impairment is associated with poorer voice and breathing outcomes and an increased risk of tracheostomy dependence.¹

Stridor and respiratory distress in recently extubated adults can have many causes, including laryngeal edema, vocal cord paresis, arytenoid subluxation or dislocation, laryngotracheal stenosis, and laryngotracheomalacia (Table).^{1,12} Diagnosis of PGS is based on history and examination findings.^{1,13} Flexible nasal laryngoscopy in an extubated patient is ideal for examining vocal fold motion and the posterior glottis.^{1,13} Operative microlaryngoscopy and bronchoscopy to palpate and evaluate the vocal fold and arytenoid cartilage is the definitive diagnostic test and is best performed in an extubated patient.¹ Fixed CAJ will not be mobile even on direct palpation of the joint.¹ Pulmonary function tests will demonstrate a fixed-lesion flow-volume loop and decreased peak inspiratory flows.^{1,13} Computed tomography or magnetic resonance imaging will often reveal glottic airway narrowing with redundant interarytenoid soft tissue and adducted vocal folds.¹³

In their 1980 article, Bogdasarian and Olson classified PGS into 4 categories (Figure 3).^{1,3} An interarytenoid scar band with posterior sinus tract defines class I PGS. Laryngoscopy often reveals diminished vocal fold abduction.^{1,3} Reduced arytenoid mobility and a posterior commissure scar characterize class II PGS.^{1,3} Class III patients have unilateral CAJ fixation. They present with a unilateral, adducted, and immobile vocal fold due to the fixated side and a contralateral hypomobile vocal fold.^{1,3} In the most severe form or class IV, the CAJs are fixed bilaterally.^{1,3}

Tracheostomy is often temporarily necessary for severe PGS to maintain airway patency.¹⁴ Initial treatment includes intralesional steroids, laser scar lysis, and balloon dilation.¹⁴ Additional surgical interventions include arytenoidectomy, cordectomy, cricoid split, and mucosal advancement flaps.¹ In a cohort of 29 patients, Lowery et al² found that surgical intervention within 45 days of intubation was associated with a decreased duration of tracheostomy dependence, a higher rate of decannulation, and fewer numbers of surgical procedures.¹⁴ In this case, early surgical consultation and intervention facilitated the diagnosis and management of class IV PGS.

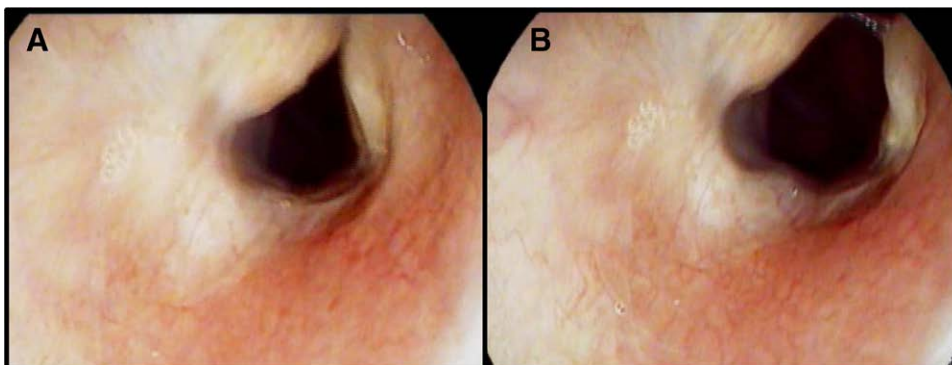


Figure 2. Flexible laryngoscopy 5 mo after initial injury performed before decannulation. Images obtained during quiet respiration (A) and inspiration in a sniffing position (B) showing normal vocal fold motion and recovery of posterior glottic stenosis.

Table. Differential of Postextubation Respiratory Distress in Adults

Presentation	Diagnosis	Treatment
Complete laryngospasm Acute onset of total laryngeal obstruction inability to ventilate and oxygenate patients.	Laryngeal visualization demonstrating complete glottic closure.	Positive pressure typically first line followed by intravenous anesthetic like propofol or neuromuscular blockade.
Laryngeal edema ¹¹ Respiratory distress and stridor immediately postextubation.	Cuff leak test, ultrasonography, and videolaryngoscopy are emerging as diagnostic tools for identifying at-risk patients before extubation.	Reintubation is often required in severe cases. This condition responds well to intravenous corticosteroids and nebulized racemic epinephrine.
Posterior glottic stenosis ¹² Inspiratory stridor, dyspnea, hoarseness, and respiratory distress.	Endoscopic evaluation of the posterior glottis, joint mobility assessment demonstrating decreased arytenoid mobility, and inability of vocal cord abduction.	Ranges from balloon dilation and scar excision to more extensive open or endoscopic procedures.
Subglottic stenosis ¹² Stridor, respiratory distress, and dyspnea.	Endoscopic visualization. Difficulty passing endotracheal tube past rima glottidis.	Temporizing maneuvers include helium-oxygen, racemic epinephrine, and intravenous corticosteroids; however, surgical interventions, such as dilation, laser excision, or more extensive reconstructive procedures, may be necessary.
Tracheal stenosis ¹² Dyspnea, stridor, or wheezing, recurrent respiratory infections.	Imaging studies and bronchoscopy. Pulmonary function testing can demonstrate intrathoracic obstruction.	Surgical management that can include endoscopic techniques or reconstructive procedures.
Vocal cord paresis ¹² Acute dysphonia with a weak breathy voice, increased vocal effort, dysphagia, and increased aspiration risk. This can be unilateral or bilateral.	Based on flexible or mirror laryngoscopy for vocal cord mobility assessment. Paralyzed vocal cord usually found in the paramedian, less often in lateral or midline position.	Recovery may be spontaneous. A variety of endoscopic and surgical procedures are available.
Arytenoid subluxation or dislocation ¹² Hoarseness, breathiness, vocal fatigue, throat discomfort, andodynophagia.	Rare with true incidence unknown. Flexible laryngoscopy often gives the appearance of vocal cord paresis. Direct laryngoscopy and arytenoid palpation can demonstrate complete arytenoid immobility and malrotation suggesting arytenoid subluxation or dislocation.	Closed reduction of the arytenoid joint typically endoscopically.
Laryngotracheomalacia ¹² Dyspnea, chronic cough, wheezing, stridor, and respiratory failure.	Diagnosis is made through endoscopy showing airway collapse during expiration.	Surgical management can include reconstructive procedures, stenting, or splinting.

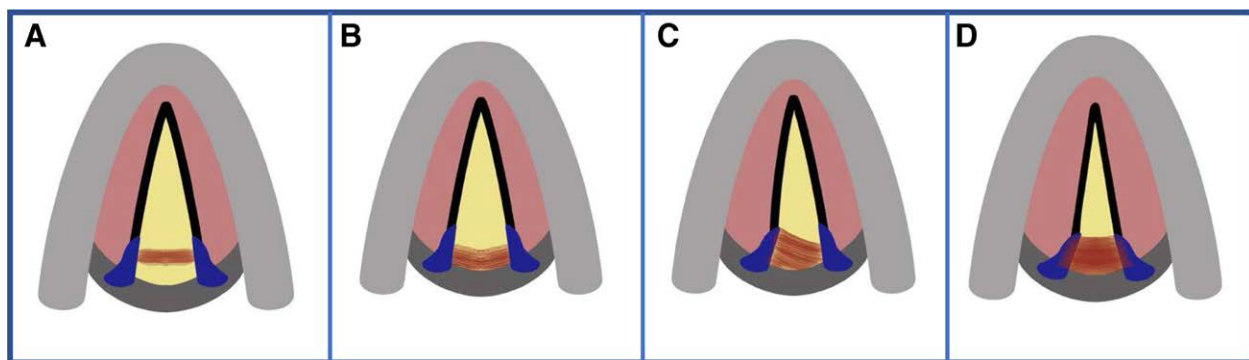


Figure 3. Stages of PGS. A, Class I PGS, characterized by the presence of an interarytenoid scar band and a posterior sinus tract. B, Class II PGS, characterized by a more posterior interarytenoid scar band without a posterior sinus tract. C, Class III PGS, which is characterized by fibrosis and unilateral CAJ fixation. D, Class IV PGS, the most severe form, characterized by fibrosis and bilateral CAJ fixation. CAJ indicates cricoarytenoid joint; PGS, posterior glottic stenosis.

The recent Severe Acute Respiratory Syndrome Coronavirus 2 pandemic, commonly known as COVID-19, has seen an unprecedented number of patients requiring orotracheal intubation and mechanical ventilation.^{1,15} Intensivists, anesthesiologists, and otolaryngologists are

likely to see an increasing number of patients with airway pathologies presenting for care.¹⁵ Posterior glottic injury and its sequelae should be considered for any patient with respiratory distress following even short periods of intubation, and early surgical consultation should be obtained. ■

DISCLOSURES

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REFERENCES

1. Lackey TG, Chabuz CA, Fink DS. Posterior glottic stenosis: a review of surgical management outcomes. *Int J Head Neck Surg.* 2022;13:47–54. Available at: <https://www.ijhns.com/doi/IJHNS/pdf/10.5005/jp-journals-10001-1524>.
2. Lowery AS, Malenke JA, Bolduan AJ, Shinn J, Wootten CT, Gelbard A. Early intervention for the treatment of acute laryngeal injury after intubation. *JAMA Otolaryngol Head Neck Surg.* 2021;147:232–237.
3. Gaffey MM, Sun RW, Richter GT. A novel surgical treatment for posterior glottic stenosis using thyroid ala cartilage—a case report and literature review. *Int J Pediatr Otorhinolaryngol.* 2018;114:129–133.
4. Shinn JR, Kimura KS, Campbell BR, et al. Incidence and outcomes of acute laryngeal injury after prolonged mechanical ventilation. *Crit Care Med.* 2019;47:1699–1706.
5. Carpenter DJ, Hamdi OA, Finberg AM, Daniero JJ. Laryngotracheal stenosis: mechanistic review. *Head Neck.* 2022;44:1948–1960.
6. Cooper JD. Tracheal injuries complicating prolonged intubation and tracheostomy. *Thorac Surg Clin.* 2018;28:139–144.
7. Brodsky MB, Akst LM, Jedlanek E, et al. Laryngeal injury and upper airway symptoms after endotracheal intubation during surgery: a systematic review and meta-analysis. *Anesth Analg.* 2021;132:1023–1032.
8. Hillel AT, Karatayli-Ozgursoy S, Samad I, et al; North American Airway Collaborative (NoAAC). Predictors of posterior glottic stenosis: a multi-institutional case-control study. *Ann Otol Rhinol Laryngol.* 2016;125:257–263.
9. Paulsen FP, Jungmann K, Tillmann BN. The cricoarytenoid joint capsule and its relevance to endotracheal intubation. *Anesth Analg.* 2000;90:180–185.
10. Benjamin BHL. Laryngeal complications of endotracheal intubation. *Ann Otol Rhinol Laryngol.* 2008;117:1–20.
11. Pluijms WA, van Mook WN, Wittekamp BH, Bergmans DC. Postextubation laryngeal edema and stridor resulting in respiratory failure in critically ill adult patients: updated review. *Crit Care.* 2015;19:295.
12. Pacheco-Lopez PC, Berkow LC, Hillel AT, Akst LM. Complications of airway management. *Respir Care.* 2014;59:1006–1019.
13. Gardner GM. Posterior glottic stenosis and bilateral vocal fold immobility: diagnosis and treatment. *Otolaryngol Clin North Am.* 2000;33:855–878.
14. Nouraei SAR, Singh A, Patel A, Ferguson C, Howard DJ, Sandhu GS. Early endoscopic treatment of acute inflammatory airway lesions improves the outcome of postintubation airway stenosis. *Laryngoscope.* 2006;116:1417–1421.
15. Piazza C, Filauro M, Dikkers FG, et al. Long-term intubation and high rate of tracheostomy in COVID-19 patients might determine an unprecedented increase of airway stenoses: a call to action from the European Laryngological Society. *Eur Arch Otorhinolaryngol.* 2021;278:1–7.