Posterior Glottic Stenosis Type I: Clinical Presentation and Postoperative Course

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Abstract

Posterior glottic stenosis (PGS) is a process that results in partial or total fixation of the vocal folds. Type I PGS (PGS-I) is an uncommon clinical entity that results from an interarytenoid adhesion/scar band that is separate from the posterior interarytenoid mucosa. We present a case series of patients with PGS-I treated at our institution to contribute to the understanding of this complex clinical entity.

Keywords

posterior glottic stenosis, laryngeal stenosis, laryngeal scar

Introduction

The posterior glottis is comprised of the posterior third of the vocal cords, the posterior commissure with its interarytenoid muscle, the cricoid lamina, the cricoarytenoid (CA) joints, the arytenoids, and the overlying mucosa. Posterior glottic stenosis (PGS) is a fibrotic process that results in total or partial fixation of the vocal folds.1

Posterior glottic stenosis is frequently caused by intubation injury to the interarytenoid mucosa, although other mechanisms such as radiation exposure, systemic autoimmune disease, external trauma, and caustic ingestion have been described.2-4 Studies have cited the incidence of PGS in patients intubated between 5 and 10 days to be 5%, whereas those intubated between 11 and 24 days to be as high as 12%.5

Bogdasarian and Olson classified PGS into 4 types. Type I involves an interarytenoid adhesion/scar band that is separate from the posterior interarytenoid mucosa. Type II involves stenosis of the posterior commissure. Type III is posterior commissure stenosis with unilateral CA ankylosis, and type IV is posterior commissure stenosis with bilateral CA ankylosis.6

 Compared to the other types of PGS, PGS type I (PGS-I) is less common, and the current literature is limited. Bogdasarian and Olson described PGS-I in 1 of 10 patients presented. Pinto et al published an incidence of PGS by subtype with 0% for type I, 58.8% for type II, 29.4% for type III, and 11.8% for type IV (n = 17), but this article only examined patients with immobile vocal folds.7 This study was designed to describe our clinical experience with PGS-I in order to improve understanding of the clinical presentation, surgical care, and postoperative course for this disease process.

Methods

A case series of patients diagnosed with PGS-I during a 2-year period (2017-2018) at a single-tertiary medical center is presented and surgical management is described. Posterior glottic stenosis type-I was defined as having an interarytenoid scar band separate from the posterior interarytenoid mucosa. Data were collected from the medical record including operative reports. Comorbidities obtained from the medical record included obstructive sleep apnea (OSA), diabetes mellitus (DM), and gastroesophageal reflux (GERD). Intubation data included duration of intubation, Cormack-Lehane Grade, largest endotracheal tube (ETT) size utilized, and number of extubation attempts. Tracheotomy tube placement was also documented.

Pre and postsurgical intervention, data were collected on subjective shortness of breath and dysphonia. Vocal fold mobility was assessed pre and postoperative with repetitive phonatory task.3 Preoperative stroboscopy was not performed on all patients due to circumstances of presentation; however, postoperative stroboscopy was obtained to fully assess postsurgical vocal fold motion.

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All patients underwent microsuspension laryngoscopy once the diagnosis of PGS-1 was made. Anesthesia technique varied by patient (case 1: general anesthesia via existing tracheotomy tube; case 2: general anesthesia via microlaryngeal ETT; case 3: intermittent apnea technique). All scar bands were excised with a combination of sickle knife and microscissors. Triamcinolone 40 mg/mL was injected at the site of scar band attachment bilaterally with 0.25-0.5 mL used. Postoperatively, patients were instructed to perform sniff exercises. All procedures were performed by the same fellowship trained laryngologist (JM).

**Case Reports/Results**

**Case I**

A 47-year-old female presented to the otolaryngology clinic with a 6-month history of tracheostomy dependence and dysphonia. She had a history of Guillain-Barre syndrome resulting in prolonged intubation, for which she was no longer receiving treatment. Additionally, she had a history of morbid obesity (body mass index: 61.2) and OSA. At the time of presentation, there was no documented history of GERD. On flexible laryngoscopy, she was found to have hypomobile vocal folds, restricted CA-joint movement without jostle sign, and a posterior glottic scar band (Figure 1A). Intraoperatively, this patient had the most extensive scarring. A dense scar with a small amount of granulation tissue was noted extending for the medial portion of posterior vocal fold on the right to the under surface of the vocal fold on the left. After lysis of the scar band, normal CA-joint movement was noted. Following surgical management, she endorsed improvement in voice quality, and on flexible laryngoscopy, she was found to have improved vocal fold mobility with resolution of her scar band. Mucosal wave was impaired posteriorly on the right but intact on the left posteriorly. However, she remained tracheotomy dependent due to underlying diaphragm paralysis, morbid obesity, and severe OSA.

**Case II**

A 61-year-old female was referred from interventional pulmonology due to bronchoscopy findings and dyspnea on exertion. She had a history of prolonged intubation and tracheotomy placement following appendectomy and acute respiratory distress a few months prior. She had been decannulated for several weeks but developed dyspnea following closure of her stoma. Flexible bronchoscopy for evaluation of her dyspnea by pulmonology found tracheomalacia and PGS-1 (Figure 1B). Additionally, she had a history of OSA, GERD, and DM. Her DM and GERD was medically managed and controlled at time of presentation. In addition to dyspnea on exertion, she described dysphonia, and on flexible laryngoscopy, she was found to have hypomobile vocal folds, restricted CA-joint movement without jostle sign with a posterior glottic scar band. Intraoperatively, there was no granulation tissue but a thin, dense band connecting the posterior-medial portions of both vocal folds. With palpation, CA-joint movement was found to be normal. Postoperatively, she had an improvement in her dysphonia without abnormality of mucosal wave and complete glottic closure. Vocal fold motion was also noted to be normal, but she had a delayed resolution (3 months) of her dyspnea. Over a year later, she remains symptom free.

**Case III**

Following treatment for a ruptured cerebral arteriovenous malformation (AVM), a 64-year-old female presented for airway evaluation given a history of difficult intubation and need for further surgery. She had a history of obesity (Body Mass Index: 34.9) and GERD, which was medically managed at time of presentation. Due to her ruptured AVM, she required prolonged intubation and had a failed extubation attempt. During reintubation, she was noted to be at difficult intubation, and the anesthesia provider reported difficulty passing an ETT but had no difficulty visualizing the larynx. For these reasons, tracheotomy was performed, and she was eventually decannulated.
without difficulty. Subsequent AVM surgery was required prompting airway evaluation. She was mainly asymptomatic, but she did endorse very mild dysphonia. She had no shortness of breath. During flexible laryngoscopy, she was found to have a poster glottic scar band, restricted CA-joint movement without jostle sign, and hypomobile vocal folds (Figure 1C). Intraoperatively, she had normal CA-joint mobility after lysis of scar. Her thin dense scar band connected the posterior-medial surface of both vocal folds. Postoperatively, she reports an improvement in her dysphonia with normal mucosal wave. Normal vocal fold mobility was noted and subsequent intubation was performed without difficulty. The patient data are summarized in Tables 1 and 2.

Discussion

When evaluating risk factors for PGS, studies commonly examine all types of PGS together. It is unclear as to what processes cause more severe grade of disease in some patients. Several factors have been shown to increase the overall risk of PGS including duration of intubation, ischemic conditions, and DM. Additional risk factors that have been proposed including reflux, congestive heart failure, stroke, and bacterial infections. Based on White prolonged intubation, greater than 11 days has shown to increase risk, as well. Patient II in our study had DM and was intubated for 11 days prior to tracheotomy placement. All patients were intubated with a 7.5 ETT. Per prior research, ETT larger than 7.5 increases the risk for glottic stenosis. No patients within our cohort had this risk factor. Obesity has been suggested to play a role, which was present in the remaining 2 patients, but studies have yet to show this to be a risk factor. It is known that obesity can inaccurately prompt an increase in ETT size which does increase risk. Reﬂux has been considered to be a risk factor in the etiology of PGS, but it was not found to be significant in a prior study. Patient II and III had GERD and required medical therapy for management. In the future, examining types of PGS separately may yield more information, though this is somewhat limited by the incidence of disease.

In regard to PGS-1, the literature shows a variety of postoperative airway findings and symptoms. In the Meyer’s article, several patients intraoperatively had CA-joint ﬁxation that remained after lysis of the scar. Ankylosis of the CA-joint implies more severe disease, potentially an indistinct aspect of the grading system when considering type 1. When comparing our patients to patients in the literature without CA-joint ﬁxation, our patients had inconsistent results. Two of the patients had additional causes of shortness of breath (diaphragm paralysis and tracheomalacia) that impaired recovery following lysis of the scar band likely impacting our results. The third patient in our cohort was asymptomatic except for a history of difﬁcult intubation. Taking these ﬁndings into consideration, the scar band and restricted mobility played a less substantial role in any respiratory symptoms for our patients.

In our study, all patients reported an improvement in their voice, though their preoperative voice complaints were not severe. The voice of patient II changed the most postoperatively, in that, there was less strain in voice quality, and the patient felt her voice had returned to normal following the procedure. Patient I had the most extensive scar and subsequently had unilateral impaired mucosal wave posteriorly, but this did not subjectively impact the patient’s voice quality postoperatively. Objective voice measure for all patients may have added to this analysis. When comparing these patients to patients with normal CA-joint movement with PGS-1, our voice results are consistent with the literature. Specially, surgical treatment resulting in improved vocal fold motion, typically resulted in an improved voice quality.

Our surgical approach did not vary from the traditional literature; however, a recent report has demonstrated in-office procedures approach. Our case series adds to the clinical understanding of this rare entity by evaluating the postoperative results in 3 patients with normal CA-joint movement. For
PGS-1, we feel that patients with CA-joint ankylosis, with persistent postoperative hypo or immobile vocal folds, may represent a subset of this population. Those patients with normal joint movement as seen in our study, all had improved dysphonia postoperatively, but the impact of the scar band in these patients on their pre and postoperative respiratory symptoms are less pronounced than those with CA-joint ankylosis.

**Conclusion**

Posterior glottic stenosis is a complex and challenging clinical entity. Of the 4 types, PGS-1 is the least common, and reviewing these cases helps to collectively expand our understanding of this entity. For our patients, surgical intervention lead to an improvement in voice quality, but respiratory symptoms had a variable response to surgery.

**Authors’ Note**

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