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# Vocal Process Granuloma and Glottal Insufficiency: An Overlooked Etiology?

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**Objectives/Hypothesis:** Vocal process granuloma has been attributed to intubation, laryngopharyngeal reflux, and phonotraumatic/hyperfunctional vocal behaviors. Vocal process granuloma has recurrence rates following surgical excision approaching 92%. We hypothesize that a portion of persistent or idiopathic cases of vocal process granuloma result from underlying glottal insufficiency (GI) caused by paresis, scar, or atrophy. Our goal was to examine our vocal process granuloma population and determine the incidence of GI, treatment interventions, and outcomes.

**Study Design:** Retrospective chart review.

**Methods:** Thirty-four patients with vocal process granuloma were divided into surgically or conservatively managed groups. Patients were identified if they carried a diagnosis of GI. The time to resolution and number of recurrences within the overall treatment period was recorded and compared between subgroups. Pre- and post-treatment Voice Handicap Index-10 (VHI-10) and Reflux Symptom Index (RSI) scores were compared.

**Results:** Eighteen of 34 patients (53%) carried an underlying diagnosis of GI, 13/34 (38%) were treated surgically, and 8/13 (62%) surgical patients had underlying GI. VHI-10 and RSI scores significantly improved after disease resolution ($P < .05$).

**Conclusions:** The incidence of GI among patients with vocal process granuloma was 53%. Conservative therapies including treatment of laryngopharyngeal reflux and voice therapy may lead to resolution despite underlying glottal incompetence. If conservative measures fail, recognizing and treating glottal incompetence with true vocal fold augmentation may lead to a shorter surgical treatment course.

**Key Words:** Vocal process granuloma, vocal fold granuloma, vocal cord granuloma, intubation granuloma, contact granuloma, laryngeal contact ulcer, glottal insufficiency.

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## INTRODUCTION

Vocal process granuloma (VPG), first described by Chevalier Jackson in 1928 as "contact ulcer of the larynx," is known in the literature by many names, including laryngeal contact ulcer, contact granuloma, vocal fold granuloma, postintubation granuloma, and arytenoid granuloma.[1,2] VPG has become an accepted term for this benign lesion of the posterior glottis seen over the vocal processes of the arytenoid cartilages.[2,3] VPG continues to stir debate in the literature with regard to its optimal treatment modality. Most papers written in the past 25 years have identified three primary causes of VPG formation: intubation, phonotrauma/hyperfunctional, or laryngopharyngeal reflux (LPR).[2–22] As observed by Hoffman et al., this designation system fails to account for patients whose symptoms are multifactorial and may overlap multiple categories, in addition to those who have idiopathic, recalcitrant granulomas.[2] The treatments offered to patients with VPG attempt to be etiology driven, but the cause is not always known.[2] Often, a conservative combination of empiric medical and behavioral therapy (i.e., voice therapy) is initially tried in the symptomatic patient to overcome LPR and hyperfunctional vocal behaviors.[4] Surgery (cold steel or laser ablation/photothermolysis) remains an option when conservative measures fail, if airway obstruction is present, or when the diagnosis is uncertain (i.e., ruling out malignancy). These procedures can be performed in the operating room or an in-office setting.[2,5–7,9–10] Previous case series have demonstrated high recurrence rates (as high as 92%) despite surgery and possibly prolonged recovery time.[5] Other forms of treatment have been proposed including antibiotics, corticosteroids, postexcision irradiation, and botulinum toxin A injection.[2,5,11,12–14] Of these, botulinum toxin A injections have shown the most promise at the expense of a high incidence of symptomatic dysphagia and dysphonia during the treatment period due to the side effects from botulinum toxin A.[12–14]

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The designation of "idiopathic" VPG is given as a diagnosis of exclusion. We posit that another etiology may exist. The concept of incomplete closure of the membranous vocal folds causing increased forces to be applied to the posterior glottis is supported by Hillman's objective evaluation of aerodynamics during voicing.[15] These increased forces are likely present with glottal insufficiency (GI) from any etiology including vocal fold paralysis, paresis, scar, or atrophy. The patient with GI provides increased effort to close their vocal folds and may subsequently cause undue pressure on the vocal processes of the arytenoids. A sole publication associating GI and VPG comes from Koufman et al.: "patients with glottal closure problems such as paresis may have vocal nodules, vocal process granulomas, or other lesions that result from hyperkinetic compensation."[16] The concept of vocal fold augmentation to treat VPG is not novel. However, most reports are anectodal.[2] The recalcitrant VPGs in our practice have implored us to re-examine the underlying etiology of these cases. We hypothesize that a portion of our difficult cases, especially those with an idiopathic or nonintubation etiology, may carry a concomitant diagnosis of GI, and thus treatment success will not occur until the underlying GI is addressed. Our primary aim in this study was to examine our VPG population, determine the incidence of GI in this cohort of patients, and to evaluate our treatment interventions and subsequent outcomes among those with and without GI. We are reporting our experience in an effort to determine if GI should be included in the various etiologic factors considered for patients with recurrent and/or recalcitrant VPG.

## MATERIALS AND METHODS

Institutional review board approval was obtained prior to patient medical record review from the University of Pittsburgh Voice Center. Patients were identified who carried a diagnosis of "granuloma." Exclusion criteria included the granuloma being present in locations other than the vocal process or patients with concomitant diagnosis or history of epithelial dysplasia or laryngeal carcinoma. All patients had been followed for at least 1 month with at least one return visit. Prior surgeries by referring physicians were noted, but not utilized to determine the date of initial diagnosis. Seventy-eight patients were identified as having a granuloma by chart review, and 34 patients met all inclusion criteria.

Patients were stratified based on conservative or surgical management. Conservative management included antireflux therapy with or without voice therapy. Typical voice therapy for this patient population includes vocal hygiene education, elimination of phonotrauma, and voice therapy techniques that use both flow phonation and resonant voice therapy. For most VPG patients in our center, four to six weekly sessions of voice therapy (lasting 45 minutes each) are followed by 4 weeks of the patient working to transfer the voice therapy techniques to their daily lives. Patients receiving any other intervention besides antireflux or voice therapy before resolution of their VPG were categorized in the surgical group for analysis. All VPGs removed surgically were done so using microsuspension laryngoscopy with cold knife excision (MSL excision). Adjunctive measure may have included the following: fibrin glue placement after removal of the VPG, preoperative or single modality electromyogram-guided botulinum toxin A injection to the ipsilateral thyroarytenoid/lateral cricoarytenoid complex, vocal fold injection augmentation (VFIA) at the time of MSL excision or as a separate procedure, and steroid injection to the site of the excised VPG. Patients in the surgical group usually received reflux management and/or voice therapy as a part of their treatment course (Table I).

Patients were identified if their VPG was associated with a recent endotracheal intubation (intubation), a concomitant diagnosis of GI, or a truly idiopathic diagnosis of exclusion (idiopathic). We defined GI as a comorbid presence of true vocal fold (TVF) atrophy, decreased TVF mobility (paralysis, paresis, immobility, or hypomobility), or VF scar (including sulcus vocalis). This diagnosis was sometimes made with the VPG present (subject 4), and at other times as a realization after excision (subjects 1 and 11).

Treatment outcome was stratified as follows: 1) resolved, if there was complete resolution of the VPG; 2) improved/asymptomatic, if the patient's lesion was persistent but asymptomatic; or 3) persistent, if the VPG remained present and symptomatic despite treatment. Any recurrences were also noted. Respective pretreatment and most recent symptom specific quality-of-life indices, which consisted of the Voice Handicap Index 10 (VHI-10) and Reflux Symptom Index (RSI), were collected (Table I).[23,24]

As the presence of a VPG can induce GI, this precluded accurate, blinded judgment of vibratory parameters of laryngovideostroboscopy (LVS), and thus video perceptual analysis was not attempted. LVS was reviewed for the entire cohort looking for segments that would allow frame-by-frame analysis of one glottal cycle without a VPG present and prior to any VFIA. Eleven segments were available from our cohort of GI patients with subjects phonating at most comfortable pitch and loudness. One healthy, age-matched control's LVS was also used in the analysis. Frame-by-frame analysis was performed in the following fashion. The authors measured the number of frames at the first evidence of vocal fold closure, ending with the frame before the first evidence of vocal fold opening. This designation of frames represented a stroboscopic illusion of the closed phase of one illusionary vibratory cycle. The frame that began with the first evidence of vocal fold opening was also identified, and then the number of frames were counted, ending with the frame before the first evidence of vocal fold closure. These frames represented a stroboscopic illusion of the opening and closing phase of one vibratory cycle. The final mathematical formula for calculating the percentage of closure duration was a simple ratio of the number of closed phase frames divided by the total number of all frames (opening, closing, closed phase) in one illusionary cycle (Fig. 1). The term "illusion" is used to remind the reader that the stroboscopic image is not a representation of cycle-by-cycle behavior, but rather is a composite of a number of cycles; therefore, the use of closure duration in discussing stroboscopic observation is not entirely accurate, but rather an extrapolation from the inherent limitations provided by LVS. However, with that understanding, it is a convenient and practical way that we determine closure duration using LVS in our clinical practice. To our knowledge, this method of counting frames to determine GI, or perhaps short versus long closure duration, is novel in the peer-reviewed literature.

A Student $t$ test was used to compare the VHI-10 and RSI scores from pre- and post- treatment. The Mann-Whitney $U$ test was utilized to compare the number of days to resolution for those subjects with GI treated conservatively versus those with GI treated surgically. The Mann-Whitney $U$ test was also utilized to determine statistical difference in days to resolution between those surgically treated patients with GI who received VFIA versus those who did not.

TABLE I.
Subject Treatment Outcome.

| Subject No., Sex | Surgical or Conservative | Reflux Therapy | Voice Therapy Y/N | VHI Pre | VHI Post | RSI Pre | RSI Post |
|---|---|---|---|---|---|---|---|
| 1M | S | BID | Y | 11 | 2 | 14 | 1 |
| 2F | S | BID + QHS | N | 31 | 14 | 34 | 24 |
| 3M | S | BID + QHS | Y | 2 | 9 | NA | NA |
| 4M | S | QD + QHS | Y | 15 | 15 | 25 | 9 |
| 5M | S | QD + QHS | N | 34 | 10 | 21 | 1 |
| 6M | S | QD + QHS | Y | 8 | 11 | NA | NA |
| 7M | S | BID + QHS | Y | 21 | 15 | 23 | 13 |
| 8M | S | BID | Y | 25 | 12 | 13 | 15 |
| 9M | S | None | N | NA | NA | NA | NA |
| 10M | S | BID + QHS | Y | 18 | 10 | 17 | 9 |
| 11M | S | BID + QHS | Y | 17 | 8 | 42 | 7 |
| 12M | S | BID + QHS | Y | 3 | 1 | 13 | 12 |
| 13F | S | BID | N | 2 | 2 | 26 | 6 |
| 14M | C | QD + QHS | N | 0 | 3 | 9 | 11 |
| 15M | C | BID + QHS | Y | 12 | 6 | NA | NA |
| 16F | C | BID + QHS | Y | 15 | 2 | 35 | 8 |
| 17M | C | BID + QHS | Y | 20 | 20 | NA | NA |
| 18M | C | QD + QHS | Y | 12 | 3 | NA | NA |
| 19M | C | QD + QHS | Y | 2 | 0 | 28 | 5 |
| 20F | C | QD | Y | 0 | 0 | 2 | 5 |
| 21F | C | BID + QHS | Y | 14 | 1 | 22 | 1 |
| 22F | C | QD + QHS | N | 14 | 14 | 30 | 25 |
| 23M | C | BID + QHS | Y | 16 | 4 | 23 | 6 |
| 24M | C | BID | Y | 9 | 13 | 26 | 20 |
| 25M | C | QD + QHS | Y | 27 | 9 | 19 | 7 |
| 26F | C | BID + QHS | Y | 21 | 28 | 38 | 38 |
| 27M | C | QD + QHS | Y | 1 | 1 | 9 | 11 |
| 28M | C | BID + QHS | Y | 0 | 0 | 5 | 4 |
| 29M | C | QD + QHS | Y | 3 | 16 | 20 | 16 |
| 30M | C | QD + QHS | Y | 0 | 2 | 7 | 0 |
| 31M | C | BID + QHS | Y | 9 | 11 | 19 | 4 |
| 32F | C | BID + QHS | Y | 14 | 3 | 19 | 3 |
| 33M | C | QD + QHS | Y | 12 | 0 | 7 | 4 |
| 34M | C | BID | Y | 10 | 18 | NA | NA |

Y = yes; N = no; VHI = Voice Handicap Index; Pre = pretreatment; Post = post-treatment; RSI = Reflux Symptom Index; M = male; S = surgical; BID = twice daily proton pump inhibitor; F= female; QHS = 300 mg ranitidine at bedtime; NA = not available; C = conservative; QD = daily proton pump inhibitor.

## RESULTS

Thirty-one of the 34 subjects (25 males) had unilateral VPGs. The average age was 51.4 years (range, 20 to 78 years). Thirteen patients (38%) were treated with surgery, and 21 (62%) were managed conservatively. Fifty-three percent (18/34) carried a concomitant diagnosis of GI (eight surgical and 10 conservative management, respectively), and 26% (9/34) of patients presented with a recent history of endotracheal intubation (two subjects [5 and 14] within each of the GI and intubation groups overlapped). Subject 5 had surgical removal of his VPG, without augmentation for his underlying TVF atrophy; he had small recurrence and required 10 months to resolve his lesion with further conservative therapy. Subject 14 had a temporary unilateral TVF paresis that was discovered after intubation, and as the paresis resolved on its own, the VPG resolved after 4 months of conservative therapy. Twenty-six percent (9/34) of patients were also classified as having VPG of idiopathic origin. After treatment, 76% (26/34) of all VPGs had resolved, 15% (5/34) were improved/asymptomatic, and 9% (3/34) were persistent (one from surgical and two from conservative groups) (Tables II and Table III).

Table I details the subjects' distributions within the categories, VHI-10 and RSI scores, voice therapy use, and the type of antireflux treatment received, if any. Overall, 69% (9/13) of the surgical and 90% (19/21) of the conservative patients underwent voice therapy, and 92% (12/13) of the surgical and 95% (20/21) of the conservative received reflux treatment. The average VHI-10

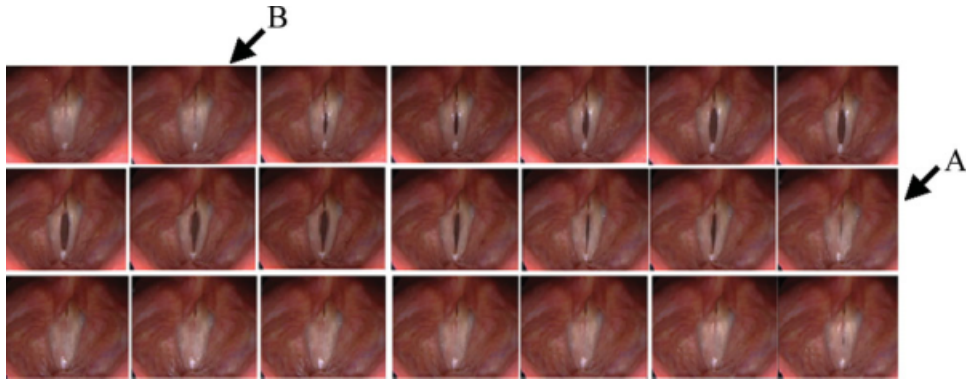


Fig. 1. Illusionary frame-by-frame analysis for glottal insufficiency. Box A represents the first frame of the illusionary closed phase of vibration, and Box B represents the last frame. Please note that the last frame on the bottom line is followed in sequence by the first frame of the top line in this representation of the glottal cycle. Percentage of closure duration was calculated as the number of frames showing the closed phase (10), divided by the total number of frames (21), resulting in a closed phase duration of 48% of the illusionary cycle. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

score improved significantly from 12.90 pretreatment to 7.96 post-treatment ($P < .05$), and the RSI improved significantly from 20.22 pretreatment to10.18 post-treatment ($P < .05$) for all subjects.

Fifty-four percent (6/13) of patients treated surgically had a recurrence of the VPG at some point during their treatment period prior to their final outcome. There were no recurrences in the conservatively treated group; however, two of these patients had persistent VPG despite treatment. Sixty-two percent (8/13) in the surgical group had a diagnosis of GI. Of these eight, three received VFIA either at the time of surgical excision (patient 4) or after realization of underlying GI after excision of the VPG (patients 1 and 11). Two of the three patients (67%) treated with VFIA resolved without recurrence, whereas only one of the remaining five (20%) surgical patients with GI proceeded without a recurrence. The subject (subject 4) who had a persistent VPG after autologous fat injection augmentation was realized to be underaugmented, but remained improved and asymptomatic. He did not want further augmentation. The other two subjects (1 and 11) received injectables that will be resorbed over time, and thus they will likely require a permanent TVF augmentation.

The number of days to resolution between those surgically treated via injection augmentation (330 days) versus those who did not (410 days) was not statistically significant ($P = .571$), likely due to the small sample sizes. The time to resolution (VPG resolved or improved) was significantly less in the conservative group (177

TABLE II.
Surgically Treated Vocal Process Granuloma Subjects.

| Subject No., Sex | History/Diagnosis | Surgical Intervention | Outcome | Mo. to Outcome* | Recurrence Prior to Final Outcome |
|---|---|---|---|---|---|
| 1M | GI/B atrophy, U paresis | VFIA | R | 17 | N |
| 2F | Intubation | MSE | R | 4 | Y |
| 3M | GI/U paresis (by EMG) | BTX/MSE/G | R | 7 | N |
| 4M | GI/U atrophy/paresis | MSE/G/VFIA | I | 5 | N |
| 5M | GI/Intubation, atrophy | MSE | R | 10 | Y |
| 6M | GI/U paresis | BTX | R | 9 | Y† |
| 7M | Idiopathic | BTX, MSE | R | 4 | N |
| 8M | Idiopathic | MSE‡ | P | 9 | Y |
| 9M | SD | BTX | R | 10 | N |
| 10M | GI/B atrophy | MSE twice | R | 19 | Y |
| 11M | GI/B atrophy | MSE, VFIA, SI§ | R | 7 | Y |
| 12M | GI/B atrophy | MSE | R | 23 | N |
| 13F | Intubation | MSE | I | 5 | N |

*Months are listed. Days were used for statistical analyses.
†Original treatment episode used for study. Recurred 5 years from initial resolution.
‡Remains in voice and reflux therapy currently.
§SI as a second procedure after recurrence.
M = male; GI = glottal insufficiency; B = bilateral; U = unilateral; VFIA = vocal fold injection augmentation; R = resolved; N = no; F = female; MSE = microsuspension laryngoscopy with excision; Y = yes; EMG = electromyogram; BTX = Botox preoperatively or as isolated treatment; G = tissue glue on site of excision; I = improved/asymptomatic P = persistent SD = spasmodic dysphonia; SI = steroid injection into site of excision.

TABLE III.
Conservatively Treated Vocal Process Granuloma Subjects.

| Subject No., Sex | History/Diagnosis | Outcome | Mo. to Outcome* | Recurrence Prior to Final Outcome |
|---|---|---|---|---|
| 14M | Idiopathic | I | 2 | N |
| 15M | Idiopathic | R | 9 | N |
| 16F | Intubation, GI/U paresis | R | 4 | N |
| 17M | Idiopathic | R | 3 | N |
| 18M | GI/U scar, U paresis | I | 14 | N |
| 19M | Idiopathic | R | 9 | N |
| 20F | Intubation | R | 12 | N |
| 21F | Intubation | R | 6 | N |
| 22F | Intubation | R | 6 | N |
| 23M | GI/U paresis | R | 9 | N |
| 24M | GI/B atrophy | I | 8 | N |
| 25M | Idiopathic | P | 13 | NA |
| 26F | Intubation | P | 19 | NA |
| 27M | GI/U paresis | R | 3 | N |
| 28M | GI/B atrophy | R | 3 | N |
| 29M | GI/U paralysis | R | 2 | N |
| 30M | GI/U paresis, B atrophy | R | 2 | N |
| 31M | GI/B sulcus, B atrophy | R | 10 | N |
| 32F | Intubation | R | 2 | N |
| 33M | Idiopathic | R | 4 | N |
| 34M | GI/B atrophy | I | 2 | N |

*Months are listed. Days were used for statistical analyses.
M = male; I = improved/asymptomatic; N = no; R = resolved; F = female; GI = glottal insufficiency; U = unilateral; B = bilateral, P = persistent; NA = not available.

days) when compared with the surgical group (379 days) ($P < .05$).

Frame-by-frame analysis data is listed in Table IV. Eight of 11 (73%) subjects had closure pattern percentages (closed frames divided by all frames) that were substantively less than our healthy control. An example of how we counted the frames is included in Figure 1. We did not intend to draw any conclusions from this data, and it is offered as an anecdotal point of interest. Future studies will enable us to compare the LVS frame-by-frame analysis technique to high-speed video and determine if this method of determining glottal competence is valid.

## DISCUSSION

Vocal process granulomas can be a formidable problem for the patient, speech language pathologist, and the otolaryngologist.[1,2] There is no other paper that we can identify that cites the incidence of GI among patients with VPG. Although our population is small, likely due to the natural incidence of VPG being low, we found that 53% (18/34) of our VPG patient population had GI. We contend that difficult to treat, idiopathic VPGs may result from under-recognized cases of glottic insufficiency.

Intubation-related VPG is often diagnosed shortly after extubation and resolves rapidly with conservative therapy. This may be anecdotally evident, although not proven, in our study by numerous patients who were seen once for postextubation VPG and never returned for follow-up as presumably their symptoms and VPG resolve. With a minimum follow-up of 1 month, nearly one half of our eligible subjects identified through medical record review were eliminated, as they did not return for their follow-up visit.

Laryngopharyngeal reflux and voice therapy are often identified and/or empirically started in patients with VPG who have no other identifiable etiology for their lesion. Peacher and Hollinger (1947) were the first to

TABLE IV.
Frame-by-Frame Analysis of Percentage of Closure Duration for the GI Cases and the Normal Control Using Laryngovideostroboscopy.

| Cases | Closed Frames/Total No. of Frames | % of Closure Duration |
|---|---|---|
| A | 5/21 | 24 |
| B | 6/19 | 32 |
| C | 7/20 | 35 |
| D | 3/22 | 14 |
| E | 3/21 | 17 |
| F | 9/19 | 47 |
| G | 9/19 | 47 |
| H | 8/21 | 38 |
| I | 1/20 | 5 |
| J | 9/19 | 47 |
| K | 2/21 | 10 |
| L | 11/21 (healthy control) | 52 |

GI = glottal insufficiency.

describe the positive effects of voice therapy on contact granulomas.[18] However, vocal imbalance and phonotrauma cannot be the sole etiology for developing these lesions, as they are of higher prevalence than VPGs.[19] Perhaps GI should be considered as another predisposing factor in this patient population, especially in those who are not resolving despite adequate treatment and compliance with medical and behavioral therapy.

Most recently, botulinum toxin A has received attention as an effective treatment modality for recalcitrant VPG.[12–14] The dysphagia and breathiness reported following botulinum toxin A therapy is suboptimal for a patient who needs his or her voice for their livelihood, or in whom aspiration could lead to pneumonia.[13,14] In our series, one patient (subject 5) had a recurrence of his VPG 60 months after his first botulinum toxin A treatment, presumably due to a previously unrecognized diagnosis of GI. In our opinion, botulinum toxin A therapy may limit the physician, as they are trying to identify the root cause of the VPG. If the underlying GI has not been realized and treated by either VFIA or medialization laryngoplasty, a recurrence may still occur in the long term.

There were no recurrences in our conservatively treated group, yet this group had 10/21 subjects with GI. This demonstrates a proportion of patients who could resolve with conservative measures despite their GI. Those challenging GI patients who failed to resolve with conservative treatment alone and went on to have a surgical therapy (or had failed previous surgical excision and were seen in consultation) required a longer time for recovery because of the repeated surgical trauma, healing time, and intermittent additional conservative therapies offered. This reveals that if a patient with GI is likely to resolve with a conservative treatment they will do so, and often in a significantly shorter time. Possibly, if GI is recognized and treated earlier in the clinical course as part of the surgical management, patients could avoid the longer treatment times currently demonstrated when conservative methods fail.

Of the 10 subjects in the surgical group who were not treated with augmentation, five had a diagnosis of GI and five did not. Three of the five (60%) with GI who were not augmented had a recurrence of their VPG during the course of treatment; this is compared to one recurrence and one persistent case among those surgical subjects without GI. The five remaining surgical patients who did not recur may be a reflection of misdiagnosis, as we labeled our subjects with GI based on the chart diagnosis, or they may have reflected a competent glottis with severe reflux or primary muscle tension dysphonia that required further work-up, therapy, or alternative treatment. In our series, when GI was realized and VFIA performed, those VPGs resolved or improved such that patients were asymptomatic in 5 months or less from the time of VFIA. Although the difference in time to resolution among those surgical patients with GI treated with VFIA versus those surgical patients with GI not treated with VFIA was not statistically significant, this may be due to the small sample size and would hopefully yield significance in a larger sample.

When reviewing success rates of voice therapy for treatment of VPGs, demographic factors, such as age and gender, may be important cofactors in optimizing treatment. The literature is replete with references that demonstrate males being treated for VPGs with surgical intervention, including Botox, more than females, when VPG was not associated with intubation.[4,7,9,12–14] Studies identifying intubation as a cause of VPG have a majority of female subjects.[5] Males are reportedly more predisposed to age-related changes of the larynx, and this may lead to more secondary hyperfunctional behaviors (muscle tension dysphonia) that exacerbate an already traumatized vocal process.[21] Perhaps, for those patients recalcitrant to medical, behavioral, and surgical treatment, an underlying etiological factor may be glottal insufficiency (e.g., vocal fold atrophy or scar). In our study, there was a high male preponderance, especially among those subjects with GI (one female, 17 males). This suggests that males may have underlying TVF atrophy and/or posterior closure patterns predisposing the vocal processes to injury.

When identifying a patient population retrospectively, inherent flaws in data collection and potential bias abound. In this study we were unable to successfully perform a video-perceptual analysis due to lack of sufficient video segments, and thus could not attempt to corroborate our chart diagnoses with a blinded video review. The frame-by-frame LVS analysis is, to our knowledge, not yet described in the literature. Having a protocol such as this to judge short versus long glottal closure duration by a simple technique using LVS could be a useful method, especially to improve communication among investigators performing research and clinicians involved in patient care. Further studies are needed to compare LVS to high-speed video and/or electroglottography in a frame-by-frame fashion to prospectively evaluate if a short duration of closure correlates with clinical GI and dysphonia.

## CONCLUSION

Vocal process granuloma often presents in one of two varieties. One subset of patients may be managed conservatively, and often includes a higher percentage of patients with the etiologies of a recent intubation or LPR with hyperfunctional vocal behaviors. The second subset appears to be recalcitrant to conservative interventions and often progresses to require an invasive procedure (surgery, botulinum toxin A injection, or others). In this study, we explored the hypothesis that patients with recalcitrant VPG may have a concomitant underlying diagnosis of GI. Of the patients examined, more than one half (53%) carried an underlying diagnosis of GI. We contend that GI should be considered in the etiology of recalcitrant idiopathic VPG, and a trend toward male predominance may exist. It is unclear from this small cohort if augmentation alone is as effective as VPG excision with concomitant augmentation. Permanent TVF augmentation should be considered as an option or adjunct to treat VPG due to hyperfunctional behaviors secondary to an incompetent glottis.

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