Received: 7 July 2024 | Accepted: 20 July 2024

DOI: 10.1002/lio2.1316

ORIGINAL RESEARCH

Laryngoscope Investigative Otolaryngology

Revisiting the etiology and clinical characteristics of hemorrhagic polyps of the vocal fold

Chloe Santa Maria MD[1] | Adam D. Rubin MD[2] | Sahiti Vemula BS[3] | Elizabeth A. Shuman MD[3] | M. Eugenia Castro CCC-SLP[3,4] | Karla O'Dell MD[3] | Michael M. Johns III MD[3]

[1]University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

[2]Lakeshore Professional Voice Center, St. Clair Shores, Michigan, USA

[3]USC Voice Center, USC Caruso Department of Otolaryngology - Head and Neck Surgery, Keck School of Medicine, California, Los Angeles, USA

[4]Department of Communication Disorders, California State University, California, Los Angeles, USA

Correspondence
Michael M. Johns III, MD, Caruso Department of Otolaryngology Head and Neck Surgery, 1537 Norfolk St, Suite 5800, Los Angeles, CA 90033, USA.
Email: michael.johns@med.usc.edu

Abstract

Objective: Benign phonotraumatic lesions of the vocal folds (BVFLs) are typically seen in younger female patients with high vocal loads. Hemorrhagic polyps (HPs) do not fit the classic paradigm of most BVFLs, as they tend to occur in an older population, have a male predominance, and report to result from a vocal accident. We present one of the largest cohorts of HPs, to reexamine their etiology and clinical features.

Methods: Retrospective cohort study, inclusive of all patients with HP managed by the senior authors between the years 2016 through 2023. Demographic data, management, phonotraumatic risk factors, pre- and post-treatment VHI-10 were reviewed. We examined patient videostroboscopy, categorized the size of the lesion, and identified any concurrent mucosal abnormality.

Results: One hundred and eleven patients had confirmed HP, 84 males (75.7%). Thirty-five patients were size category 1; pinpoint (28.9%), 57 were category 2; less than 1/3rd the vocal fold (45.5%), and 26 were category 3; greater than 1/3rd the vocal fold (21.5%). Ten patients (9%) had bilateral HPs. Thirty-five patients had an additional 40 mucosal lesions in addition to the HP(s). The onset of symptoms was gradual in 60% of patients. The mean pretreatment VHI-10 was 18.0 (SD 10.7), compared to 6.0 (SD 10.5) post-treatment, ($p < .001$). 57/111 patients reported high voice demand professions or recreational activities. The average self-reported talkative scale score was 7.6/10. Patients were managed with operative microdirect laryngoscopy and microflap excision (53.1%), in-office clinic potassium titanyl phosphate (KTP) laser (24.3%), voice therapy alone (7.2%), and KTP in the operating room (6.3%).

Conclusions: In our cohort, most patients were male, had high vocal demands, reported gradual symptom onset, and almost a third of patients had additional BVFLs.
Level of evidence: Level 3: Retrospective cohort study.

KEYWORDS
benign vocal fold lesions, dysphonia, hemorrhagic polyp, phonotrauma, vocal fold polyp

1 | INTRODUCTION

Phonotraumatic lesions of the vocal folds arise as a result of microtrauma due to collision forces during vocal fold oscillation. The vocal folds vibrate hundreds of times per second, and during these phonatory cycles, there is mechanical trauma that can occur within the superficial layers of the vocal fold. There is inflammation and subsequent remodeling, which can produce a variety of benign vocal fold lesions. The shear forces are highest in the midmembranous portions of the vocal folds.[1] Hemorrhagic polyps are thought to arise due to rupture of immature vessels associated with neovascularization during remodeling post-microtrauma.[2]

Hemorrhagic polyps can be characterized as a more acute onset rather than chronic onset pathology with a predilection for male vocal folds.[3,4] Unlike broad-based translucent polyps, which are chronic trauma-driven changes within the superficial lamina propria, hemorrhagic polyps are classically described acute in onset due to a presumed microvascular hemorrhage. Models developed by Lee et al. (2021) support this, by indicating that they arise from sudden tissue change due to trauma versus more gradual accumulation of tissue trauma in translucent broad-based polyps and midfold fibrous masses.[3] Postulations regarding male susceptibility are thought to be due to the lower fundamental frequency of the biologically male voice, and this lends itself to more mechanical stress affecting the deeper vocal fold layers and vasculature. This can allow for the rupture of vessels in the deeper layer of vocal tissue, leading to formation of a hemorrhagic polyp.[4]

An integrative literature review by Assad et al. (2017) documented risk factors related to increased incidence in phonotraumatic lesions associated with vocal behaviors with high vocal load and dose. The term vocal dose is used to define the exposure of vocal fold tissue to vibration, and it is dependent on vocal intensity (i.e., loudness), fundamental frequency (i.e., pitch), phonation time, and individual variations in vocal efficiency (i.e., vocal technique or training).[5,6] With increasing vocal dose, and associated risk factors, there is increased likelihood of voice disorders.[7,8]

In addition to increased vocal load and gender, there are potentially other conditions that may predispose the vocal fold to the development of hemorrhagic polyps. The presence of other benign mucosal lesions may make the vocal fold more vulnerable to hemorrhagic polyp formation. Any process that can increase the shear force within the deeper layers of the lamina propria, such as stiffness or scar, could promote hemorrhagic polyps. In addition, conditions that encourage forceful glottic closure such as compensation for glottic insufficiency in paresis or atrophy may play a role. There are some associations regarding concurrent benign vocal fold lesions; however, there is paucity of literature to support this.[9,10]

The standard treatment algorithm for phonotraumatic lesions in general tends to be conservative, with voice therapy being at the forefront of management.[11] The treatment paradigm for hemorrhagic polyps, however, typically favors surgical management, either with microflap excision or with potassium titanyl phosphate (KTP) laser, as voice therapy alone is typically ineffective for resolution of the polyp itself.[12,13] That said, there have been a significant number of hemorrhagic polyps that have spontaneously resolved while awaiting their intervention.[2] Ideal management includes procedures to treat the lesion and perioperative voice therapy to target the inciting behaviors and improve vocal efficiency.[13]

In this study, we present one of the largest cohorts in literature examining hemorrhagic polyps. We aim to revisit the etiology and natural history of hemorrhagic polyps. In addition, we aim to explore the clinical characteristics of this condition and assess outcomes of treatment.

2 | METHODS

We performed a retrospective cohort study, inclusive of all patients with hemorrhagic polyps managed by the senior authors between the years January 2016 through August 2023. This was performed via a search of all patients seen by the senior authors (ES, KO, and MJ), who were assigned the International Classification of Diseases, Tenth Revision (ICD-10) diagnosis code J38.1; vocal fold polyp. This was approved by the University of Southern California (USC) Institutional Review Board (IRB), approval number HS-16-00003.

We performed a chart review of all of these patients and collected information on age, gender, management undertaken, and risk factors for vocal accidents (including profession, hobbies, and self-reported talkative scale). Baseline Voice Handicap Index (VHI-10) was collected and repeated after treatment. The Voice Handicap Index (VHI) and Voice Handicap Index-10 (VHI-10), its corresponding short form, were developed to measure patients' perception of impairment due to a voice disorder.[14,15] The VHI and the VHI-10 are commonly used in clinics and are also the most cited patient-reported outcome (PRO) measures in research internationally.

Data collection regarding vocal load and increased vocal demands leading to potentially at-risk behaviors was examined by collecting information from an intake questionnaire, a self-reported talkative scale, and a review of patients reported histories of vocal use. The

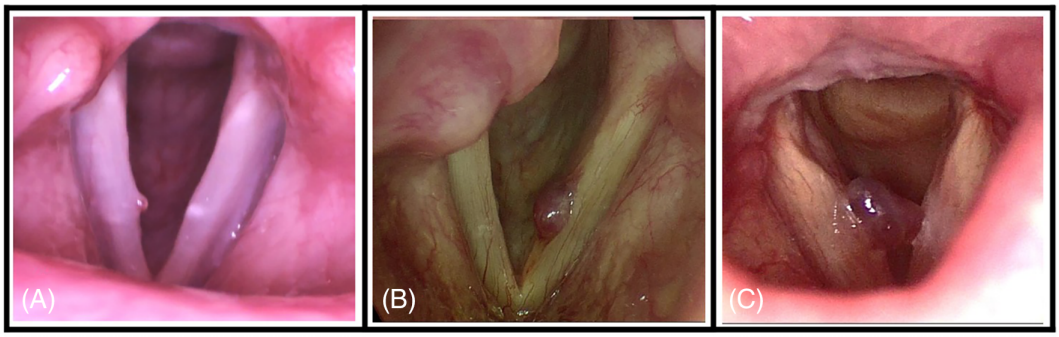

FIGURE 1 Hemorrhagic polyp size classification.

talkative scale consisted of a visual analog scale from 1 to 10 with word anchors at 1 (very quiet), 5 (average), and 10 (very talkative) (see Figure S1). The intake questionnaire and talkative scale were filled by the patient in pen and paper format prior to their appointment. The intake questionnaire included asking the patient to describe the problem in 1–2 sentences, describe the onset of voice symptoms as either "gradual" or "acute," a tick box for voice symptoms (e.g., "hoarseness", "breathiness," "straining to speak,", "shaky voice"), voice demands and occupation, and vocal hygiene habits.

We also examined every patient videostroboscopy at time of diagnosis and after treatment. We classified the size of the hemorrhagic polyp as either pinpoint (size 1), less than 1/3rd the length of the vocal fold (size 2), or greater than 1/3rd the length of the vocal fold (size 3), as previously described by Klein et al. (2009) (Figure 1). In addition, we identified the presence or absence of any concurrent mucosal abnormality of the affected or contralateral vocal fold. In the event an additional mucosal abnormality was identified, a second fellowship-trained laryngologist evaluated the exam to confirm the diagnosis. Hemorrhagic polyps can deform the medial vocal fold edge and make normal lamina propria appear abnormal. For patients with an identified sulcus vocalis at the time of hemorrhagic polyp diagnosis, we followed multiple postmanagement videostroboscopies to confirm the persistent presence of lamina propria deficiency before diagnosing a sulcus.

A two-sample student *t*-test was used to evaluate the mean difference in age and talkative scale, by gender. A paired samples student *t*-test was used to evaluate mean differences in change in pre- and postintervention VHI-10. Pearson's x^2 and two-sample tests of proportion were used to evaluated for associations between hemorrhagic polyp size and management undertaken. A multinomial logistic regression was used to evaluate differences in age for the different management groups. Statistical analyses were performed using STATA version 18 (StataCorp. 2023. Stata Statistical Software: Release 18. College Station, Texas: StataCorp LLC).

3 | RESULTS

The ICD-10 search yielded 260 patients with the diagnosis of vocal fold polyp. A chart review and review of videostroboscopy on all of the patients resulted in 111 patients with confirmed diagnosis of hemorrhagic polyp. Videostroboscopy exams were available in 108 of 111 patients.

3.1 | Demographics

Among the cohort of 111 patients, there were 84 males (75.7%) and 27 females (24.3%). The mean age of the males was 50.9 years (range 20–90 years), which was significantly older than the female cohort (44.9 years, range 25–82 years); $t(110) = 2.17$, $p = .03$, (Table 1).

3.2 | Symptoms

The most common symptom reported was dysphonia (as described by "hoarseness," "voice loss," and "change in voice"). No patients reported pain or odynophagia in presenting symptoms.

One hundred and two patients reported on the onset of their symptoms; of note, only 42 patients (41.1%) reported that their symptoms were acute in onset, versus 60 (58.9%) who felt their symptoms were gradual.

Pre- and post-VHI-10 questionnaires were collected in 66 patients, with a significant reduction postmanagement irrespective of type, mean pre-VHI-10 was 18.0 (SD 10.7) compared to 6.0 (SD 10.5) postintervention; $t(65) = 8.1$, $p < 0.001$.

3.3 | Hemorrhagic polyp features and associated mucosal pathology

There were 42 left-sided, 59 right-sided, and 10 bilateral, for a total of 121 hemorrhagic polyps. All 10 patients with bilateral hemorrhagic polyps were male. Out of the 121 polyps, 35 were size category 1 (28.9%), 57 were size category 2 (45.5%), and 26 were size category 3 (21.5%). Three were not sized due to inability to view videostroboscopy (Table 2).

Of the 108 patient videostroboscopy exams assessed, there were 40 additional mucosal lesions across 35 patients, with 5 patients

TABLE 1 Demographic information for hemorrhagic polyp cohort, by gender.

	Male $n = 84$ (75.7%)	Female $n = 27$ (24.3%)	Cohort $n = 111$ (100%)
Age	50.9 years	44.9 years	49.4 years
High vocal demand professions			
Professional voice users	23	11	34
Other high voice demand professions	15	2	17
High recreational users	3	3	6
	$n = 41/84$	$n = 16/27$	$n = 57/111$
Mean self-reported talkative scale (out of 10)	7.4 $n = 38$	8.4 $n = 13$	7.6 $n = 51$
VHI-10			
Pre intervention; mean (SD)	17.7 (11.1)	18.9 (9.3)	18.0 (10.7)
Post intervention; mean (SD	6.0 (10.4)	5.4 (9.2)	6.0 (10.5)
	$t(49) = 6.84$, $p < .001$	$t(15)=4.7$, $p < .001$	$t(65) = 8.1$, $p < .001$

TABLE 2 Hemorrhagic polyp features and management.

Hemorrhagic polyp features				
Laterality	Left $n = 42$			
	Right $n = 50$			
	Bilateral $n = 10$			
	Total of 121 polyps			
Size category	Size 1 – $n = 35$ (28.9%)			
	Size 2 – $n = 57$ (45.5%)			
	Size 3 – $n = 26$ (21.5%)			
		Size category of hemorrhagic polyp		
Management	**Average age (SE), *n***	**Size 1**	**Size 2**	**Size 3**
OR microflap with perioperative voice therapy	54.7 yrs (8.6), $n = 59$	$n = 11$	$n = 29$	$n = 19$
Clinic KTP	61.7 yrs (10.8), $n = 27$	$n = 12$	$n = 14$	$n = 1$
OR KTP	67.3 (15.8), $n = 7$	$n = 3$	$n = 3$	$n = 1$
Primary voice therapy (VT)	32.3 yrs (2.9), $n = 8$	$n = 3$	$n = 2$	$n = 1$
None	$n = 10$	$n = 1$	$n = 1$	
Spontaneous resolution	44 yrs (6.5), $n = 3$			$n = 1$
Recommended procedure but patient did not pursue	49 yrs (5.8), $n = 4$			
Recommended VT but patient did not pursue	48 yrs (6.4), $n = 3$			

Note: SE refers to standard error. For purposes of comparison the patients who were recommended a form of management but did not pursue were removed from the Pearson's chi squared testing for hemorrhagic polyp size.

having more than 1 mucosal abnormality (Table 3). The most common additional mucosal abnormality was contralateral reactive lesion and midmembranous edema. Sulcus and atrophy were also frequently identified (Figure 2).

3.4 | Vocal demands

Among patients, there were 34 self-reported professional voice users, including performing singers, actors, comedians, television news anchors, and teachers. There were also 17 patients, who self-reported heavy vocal demands as part of their profession, which included fitness instructors, attorneys, sales assistants, and real estate professionals. In addition, there were six participants who self-reported heavy recreational voice use, in the form of regular recreational karaoke, singing in a rock band and recreational podcasting. These were presented grouped as "high vocal demand" in Table 1.

Self-reported talkative scale was collected on 51 out of the 111 patients in the cohort. The mean score was 7.6/10 (SD 2.01, range 4–10). When stratified by gender, there was no significant difference in self-reported talkative scale; $t(49) = -1.57$, $p = .12$.

3.5 | Management

Patients were managed with operative microdirect laryngoscopy and microflap excision, with concomitant peri-operative voice therapy, most commonly; $n = 59$ (53.1%), in-office clinic potassium titanyl phosphate (KTP) laser; $n = 27$ (24.3%), voice therapy alone; $n = 8$ (7.2%), and KTP in the operating room; $n = 7$ (6.3%). Seven patients (6.3%) deferred recommended intervention and an additional 3 (2.7%) had planned intervention (clinic or operating room procedure) but had spontaneous resolution prior to procedure (Table 2).

One patient in the in-office KTP group had incomplete resolution of HP. In addition, one patient included in the in-office KTP group had tried voice therapy first as primary management and then ultimately underwent the procedure. Voice therapy in isolation had three patients having HP resolution, two with persistent HP, and three patients who did not have follow-up for videostroboscopy.

There was no significant association between hemorrhagic polyp size and management undertaken; $\chi^2(8) = 13.4$, $p = .10$. Nor was there any relationship between gender and management; $\chi^2(4) = 6.3$ $p = .18$. In addition, there were no differences between post-treatment VHI-10 score between the different treatment groups; $t(57) = -1.13$, $p = .87$.

TABLE 3 Mucosal abnormalities on videostroboscopy.

Abnormality detected	Count
Sulcus	6
Atrophy	6
Mid-membranous edema or polypoidal edema	8
Contralateral reactive lesion	10
Varices and ectasia	2
Vocal fold hemorrhage	4
Vocal process granuloma	1
Anterior glottic web	1
Contralateral intracordal cyst	1
Contralateral broad based translucent polyp	1
Total	40

4 | DISCUSSION

Hemorrhagic polyps are benign vocal fold lesions that are phonotraumatic in origin. Unlike most phonotraumatic lesions, hemorrhagic polyps are classically acute in nature. The presumed pathologic basis is acute microtrauma and inflammation, neovascularization, and hemorrhage from immature blood vessels.[2]

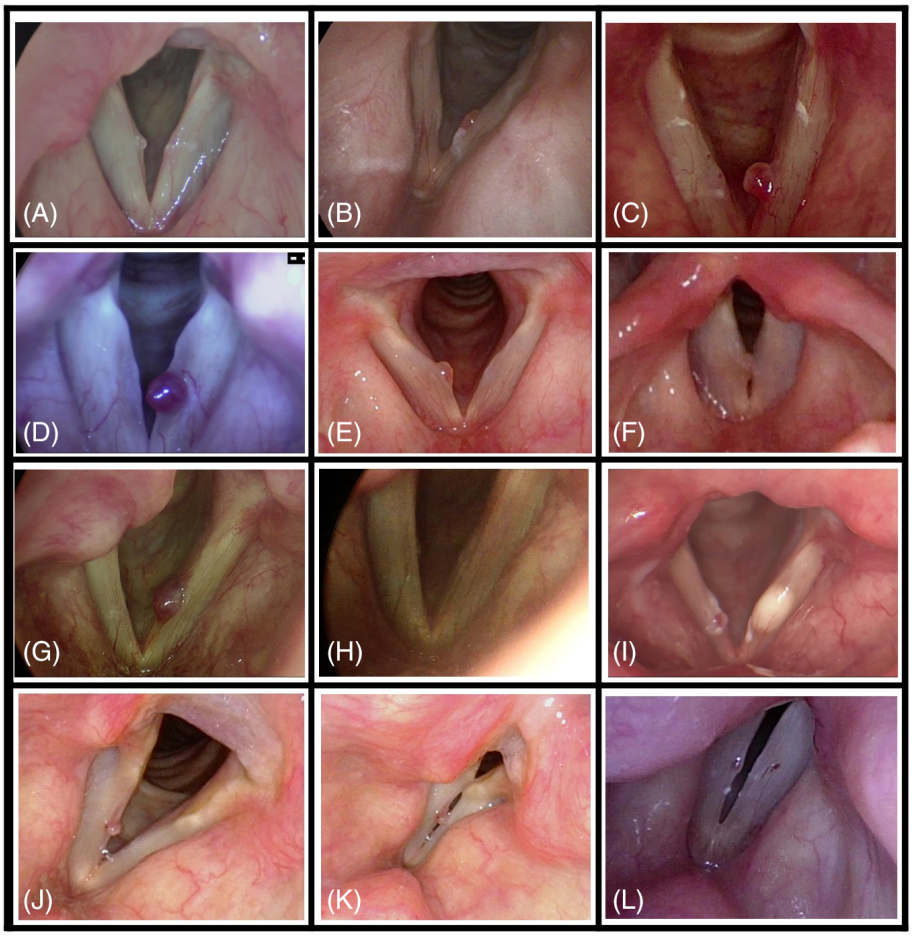

FIGURE 2 Concurrent vocal fold mucosal pathologies.

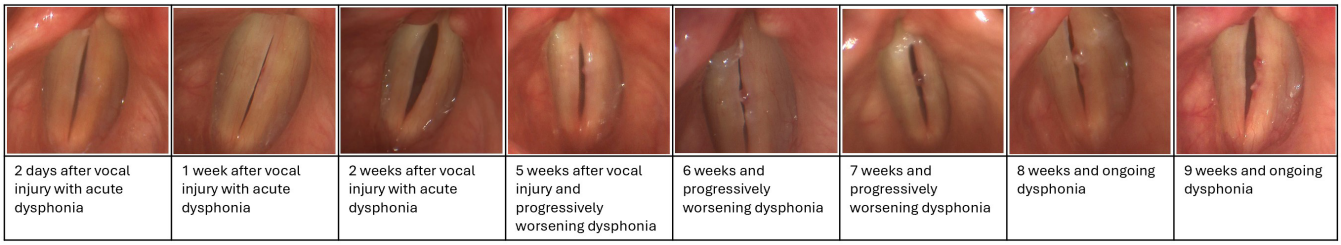

FIGURE 3 A single subjects experience of an acute vocal injury and subsequent hemorrhagic polyp development over time.

4.1 | Demographics

Similar to other published literature on hemorrhagic polyps, our cohort was predominantly male.[2,4] Different patterns in phonotraumatic lesions have been identified based on gender. Explanations for gender differences in patterns of injury revolve around differences in baseline fundamental frequency and differences in mechanical stress. Females tend to be more prone to phonotraumatic lesions in general due to a higher fundamental frequency and higher collision forces.[4] One theory is that at higher fundamental frequencies, there is more superficial (epithelial) trauma that can give rise to vocal fold nodules. In addition, females are more likely to have a physiologic posterior glottic gap, which may result in compensation and forceful closure. In males where there is a lower fundamental frequency, the deeper lamina propria vasculature becomes vulnerable to mechanical stress and more prone to hemorrhage.[4]

4.2 | Etiology and development

Despite the acute nature of hemorrhage and presumed hemorrhagic polyp formation, the majority of our cohort felt that their symptoms were actually more gradual in onset. The development of hemorrhagic polyps is presumed to be the result of phonotrauma and subsequent inflammation, remodeling, and neovascularization.[2] It is the vulnerable, immature vasculature that is then thought to hemorrhage with repeated trauma and result in a polyp. In some cases, small vessel rupture combined with repeated trauma leads to the gradual increase in size of the lesion and worsening gradual onset dysphonia. We have included stills of a subject not managed at this institution and not included in this study that highlight the gradual development and hemorrhagic polyp formation after an acute vocal injury as a result of intense vocalization (Figure 3). While the acute hemorrhage may result in a significant polyp and symptom onset in some patients, others may have a smaller lesion that develops and progresses over time. And while this is a single case study, with its own limitations, the figure below does highlight how in some patients the repeated trauma after an acute event can lead to a gradual increase in lesion size and symptoms. Hemorrhagic polyps have always been considered phonotraumatic in origin, but this sequence of microtraumatic events does change how we consider their onset and development.

It is well established that increasing vocal load and dosing, whether that be due to actual phonation time, increasing fundamental frequency, vocal intensity, or variations in vocal efficiency, increases the risk of developing benign vocal fold lesions.[3,5–7,11] The higher the vocal load, the higher the exposure of the vocal folds to vibratory trauma. It is then unsurprising that we identified a high proportion of our cohort as either professional voice users (professional singers, actors, comedians, teachers) (30.6%) or otherwise self-rated high voice demand professions (including sales assistants, attorneys, and hairdressers) (15.3%). In addition, we identified patients who had self-reported high recreational voice use, including singing in a recreational rock band or frequent karaoke singer (5.4%). The average self-report talkative scale was also high (7.6/10). A clear limitation of the current study is the lack of objective vocal dose measurements to make further statements on the role of vocal dose in the incidence of vocal fold hemorrhagic polyps. This was not feasible for the current study secondary to retrospective design. Self-reported vocal dose is often overestimated (including likely in this current study), and ambulatory voice monitors such as voice dosimeters would have been able to provide a more objective estimate.[5,16]

4.3 | Co-occurrence with other VF pathology

Multiple concurrent vocal fold mucosal abnormalities are not well described in the literature. In our study, we noted that many patients had evidence of phonotrauma and contralateral lesions. Many patients had concurrent phonotrauma at the time of diagnosis, and when the videostroboscopy was followed over time, they had persistent mid-membranous fullness/early nodules, edema, and irregular vocal fold edges many months after the hemorrhagic polyp was managed and resolved. Contact lesions tended to resolve after the removal of the inciting lesion. We also noted a group of patients with sulcus vocalis concurrent with hemorrhagic polyps. Vocal fold sulci and scar result in reduced pliability of the vocal folds, asymmetric vibration, and a restricted mucosal wave. The presence of vocal fold sulci may predispose to higher sheering forces and are also hypothesized to be implicated in the development of benign vocal fold lesions.[9] It can also result in glottic insufficiency, which may result in compensation and potentially phonotraumatic behaviors. The association between sulcus vocalis and benign vocal fold lesions is mixed, with a number of studies reporting a relationship[9,17,18] and others

finding no association.[19] One of the challenges with sulcus vocalis lies with regard to the difficulty in diagnosis, the heterogeneity of the disorder, and the high diagnostic inter-rater variability.

Phonotraumatic lesions related to voice use are typically a younger person phenomenon, while lesions related to cumulative irritant exposure, such as leukoplakia or Reinke's edema, are found in older populations.[4] A new finding from this study is that while HPs are voice-use related, the average age of this cohort is considerably older, at almost 50 years of age. It is unclear why vocal accidents may be found in an older population; one hypothesis may be related to increased incidence of glottic insufficiency with age and phonotraumatic compensatory behaviors. We noted a number of patients with concurrent atrophy in our cohort. Again, there is paucity in the literature, but other causes of insufficiency have been associated with the development of benign vocal fold lesions. Koufman et al. noted a relationship between vocal fold paresis, glottal insufficiency, and translucent broad-based polyps (also known as pseudocysts), and coined the term "paresis podule."[10] It is unclear if insufficiency is playing a role, and while we did not have premorbid videostroboscopy on these patients, certainly the majority of our cohort did not have glottic insufficiency evident after treatment. Potentially, the development of HPs is reflecting more global changes that occur with older age. With increasing age comes delays in wound healing,[20] and thus it is possible that there is an increasing vulnerability of immature vasculature to acute hemorrhage due to impaired or delayed wound healing after phonotrauma. Further conclusions are out of the scope of this current study.

4.4 | Treatment considerations

Hemorrhagic polyps are known to be poorly responsive to conservative treatment with voice therapy alone; therefore, most cases necessitate surgical intervention.[13] We know that the addition of perioperative voice therapy has been shown to improve surgical outcomes and voice quality, and reduce lesion recurrence and vocal effort.[21–24] What is notable from our cohort is that everyone responded well to treatment irrespective of type. For the VHI-10, there was no significant difference between the management, with a significant reduction for all treatment groups. There were very few treatment failures overall. When making treatment decisions, it was a shared decision-making process between the clinician and the patient. The size, morphology, and vocal use/occupational characteristics of the patient may make a certain treatments more appropriate than another. Despite these relationships not being clearly identified within this cohort, larger lesions and more complex morphologic appearance may lend themselves to better outcomes within an operative setting. Professional vocal performers and dynamic voice users were preferentially treated in the operating room due to the increased precision it allows. Within the limits of this study, we can say that overall all treatment options are valid choices (with the exception of voice therapy in isolation), and there was little difference in outcomes for this cohort. It is reasonable for the decision-making for management to reflect patient preferences, procedure tolerability, risk tolerance, fitness for anesthesia, patient comorbidities, and available resources.

5 | CONCLUSIONS

We present one of the largest cohorts in the literature of patients with hemorrhagic polyps. Our cohort was consistent with existing literature regarding the male predominance of these lesions. In addition, our cohort showed significant self-reported vocal demands and a high proportion were professional voice users, again consistent with the fact that these are phonotraumatic lesions in origin. Interestingly, more than half of our cohort felt that their symptom onset was gradual rather than acute, which makes us reconsider the developmental paradigm for hemorrhagic polyps. Our cohort also showed that almost a third of patients had a concurrent mucosal lesion, often also phonotraumatic in origin (e.g., contralateral reactive lesions, midmembranous edema, subepithelial cyst, ectasia). The presence of a concurrent sulcus may be considered a precipitant or again another marker of phonotrauma. Atrophy and glottic insufficiency as a precipitant for further phonotraumatic compensatory behaviors is controversial but potential.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ORCID

Chloe Santa Maria https://orcid.org/0009-0000-2734-3677
Adam D. Rubin https://orcid.org/0000-0002-6571-7307
Elizabeth A. Shuman https://orcid.org/0009-0009-0872-869X
M. Eugenia Castro https://orcid.org/0000-0002-8904-8806

REFERENCES

1. Titze IR. Mechanical stress in phonation. *J Voice*. 1994;8(2):99-105. doi:10.1016/s0892-1997(05)80302-9
2. Klein AM, Lehmann M, Hapner ER, Johns MM 3rd. Spontaneous resolution of hemorrhagic polyps of the true vocal fold. *J Voice*. 2009;23(1):132-135. doi:10.1016/j.jvoice.2007.07.001
3. Lee M, Mau T, Sulica L. Patterns of recurrence of phonotraumatic vocal fold lesions suggest distinct mechanisms of injury. *Laryngoscope*. 2021;131(11):2523-2529. doi:10.1002/lary.29531
4. Zhukhovitskaya A, Battaglia D, Khosla SM, Murry T, Sulica L. Gender and age in benign vocal fold lesions. *Laryngoscope*. 2015;125(1):191-196. doi:10.1002/lary.24911
5. Assad J, Magalhães M d C, Santos JN, Gama ACC. Vocal dose: an integrative literature review. *Revista CEFAC*. 2017;19(3):429-438.
6. Svec JG, Popolo PS, Titze IR. Measurement of vocal doses in speech: experimental procedure and signal processing. *Logoped Phoniatr Vocol*. 2003;28(4):181-192. doi:10.1080/14015430310018892
7. Hunter EJ, Cantor-Cutiva LC, van Leer E, et al. Toward a consensus description of vocal effort, vocal load, vocal loading, and vocal fatigue. *J Speech Lang Hear Res*. 2020;63(2):509-532. doi:10.1044/2019_jslhr-19-00057
8. Sapienza C, Hoffman B. *Voice Disorders*. 4th ed. Plural Publishing; 2020.
9. Miaśkiewicz B, Panasiewicz A, Gos E, Szkiełkowska A, Skarżyński PH, Włodarczyk E. Voice aspects in sulcus coexisting with benign lesions

of the vocal folds. *Acta Otorhinolaryngol Ital*. 2020;40(4):262-269. doi:10.14639/0392-100x-n0555
10. Koufman JA, Belafsky PC. Unilateral or localized Reinke's edema (pseudocyst) as a manifestation of vocal fold paresis: the paresis podule. *Laryngoscope*. 2001;111(4 Pt 1):576-580. doi:10.1097/00005537-200104000-00005
11. Johns MM. Update on the etiology, diagnosis, and treatment of vocal fold nodules, polyps, and cysts. *Curr Opin Otolaryngol Head Neck Surg*. 2003;11(6):456-461. doi:10.1097/00020840-200312000-00009
12. Courey MS, Shohet JA, Scott MA, Ossoff RH. Immunohistochemical characterization of benign laryngeal lesions. *Ann Otol Rhinol Laryngol*. 1996;105(7):525-531. doi:10.1177/000348949610500706
13. Naunheim MR, Carroll TL. Benign vocal fold lesions: update on nomenclature, cause, diagnosis, and treatment. *Curr Opin Otolaryngol Head Neck Surg*. 2017;25(6):453-458. doi:10.1097/moo.0000000000000408
14. Rosen CA, Lee AS, Osborne J, Zullo T, Murry T. Development and validation of the voice handicap index-10. *Laryngoscope*. 2004;114(9):1549-1556. doi:10.1097/00005537-200409000-00009
15. Jacobson B, Johnson A, Cynthia G, et al. The voice handicap index (VHI). *Am J Speech Lang Pathol*. 1997;6(3):66-70.
16. Mehta D, Cheyne HA, Wehner A, Heaton JT, Hillman RE. Accuracy of self-reported estimates of daily voice use in adults with Normal and disordered voices. *Am J SLP*. 2016;25(4):634-642.
17. Eckley CA, Corvo MA, Yoshimi R, Swensson J, Duprat AC. Unsuspected intraoperative finding of structural abnormalities associated with vocal fold polyps. *J Voice*. 2010;24(5):623-625. doi:10.1016/j.jvoice.2009.02.001
18. Sünter AV, Kırgezen T, Yiğit Ö, Çakır M. The association of sulcus vocalis and benign vocal cord lesions: intraoperative findings. *Eur Arch Otorhinolaryngol*. 2019;276(11):3165-3171. doi:10.1007/s00405-019-05623-1
19. Saraniti C, Patti G, Verro B. Sulcus vocalis and benign vocal cord lesions: is there any relationship? *Int J Environ Res Public Health*. 2023;20(9):5654. doi:10.3390/ijerph20095654
20. Gosain A, DiPietro LA. Aging and wound healing. *World J Surg*. 2004;28(3):321-326. doi:10.1007/s00268-003-7397-6
21. Koufman J, David Blalock P. Is voice rest never indicated? *J Voice*. 1989;3(1):87-91.
22. Ju YH, Jung KY, Kwon SY, et al. Effect of voice therapy after phonomicrosurgery for vocal polyps: a prospective, historically controlled, clinical study. *J Laryngol Otol*. 2013;127(11):1134-1138. doi:10.1017/s0022215113002454
23. Tang SS, Thibeault SL. Timing of voice therapy: a primary investigation of voice outcomes for surgical benign vocal fold lesion patients. *J Voice*. 2017;31(1):129.e1-129.e7. doi:10.1016/j.jvoice.2015.12.005
24. Lin L, Sun N, Yang Q, et al. Effect of voice training in the voice rehabilitation of patients with vocal cord polyps after surgery. *Exp Ther Med*. 2014;7(4):877-880. doi:10.3892/etm.2014.1499

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Santa Maria C, Rubin AD, Vemula S, et al. Revisiting the etiology and clinical characteristics of hemorrhagic polyps of the vocal fold. *Laryngoscope Investigative Otolaryngology*. 2024;9(5):e1316. doi:10.1002/lio2.1316