

Prevalence of Dysphagia in Patients With Non-neoplastic Vocal Fold Pathology

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Summary: Objectives. To examine the prevalence of dysphagia in patients presenting with dysphonia and diagnosed with non-neoplastic vocal fold pathology.

Methods. A total of 45 patients presenting with dysphonia and diagnosed with non-neoplastic vocal fold pathology and a control group matched according to age and gender were included. Patients with recent history of respiratory tract infection, laryngeal surgery or manipulation, neurologic disorders, head and neck tumors, or history of chemotherapy/radiotherapy were excluded. The primary outcome measure for dysphagia was Eating Assessment Tool-10. Patients with a score above three were considered to have dysphagia.

Results. The 45 patients were stratified as 18 males and 27 females, with an overall mean age of 48.23 ± 14.65 years. The most common vocal fold pathology was Reinke edema (28.8%), followed by laryngitis (24.4%), and vocal fold nodules (17.7%) and polyps (13.33%). Out of 45 patients with dysphonia, 37.7% had dysphagia and out of 25 controls, 8% had dysphagia as evidenced by an Eating Assessment Tool-10 score of above three. This prevalence is higher than normative values reported in the literature (16%–22%).

Conclusions. The high prevalence of dysphagia in patients with non-neoplastic vocal fold pathology alludes to the pathogenic role of laryngeal behavior in the development of obstructive swallowing symptoms. The potential benefit of voice and swallowing therapy in the treatment of these patients should be considered.

Key Words: EAT-10—Dysphagia—Vocal fold—Dysphonia—Non-neoplastic pathology.

INTRODUCTION

Dysphagia is a common symptom defined as difficulty in swallowing with inability to pass the bolus of food from the mouth to the stomach.¹ The pathophysiology may lie in the oral phase, pharyngeal phase, or in the esophageal phase. To that end, several etiologic factors have been reported and categorized as extrinsic, intrinsic, or as motility disorders. Proper history-taking and a thorough diagnostic workup using imaging studies and endoscopic examination are invaluable for investigation.²

Dysphagia has been thoroughly reported in relation to gastroesophageal diseases and ailments of the upper aerodigestive system. However, its association with dysphonia has been primarily confined to patients with systemic diseases such as autoimmune and neurogenic disorders, and to patients with vocal fold paralysis.^{3–9} It has been primarily attributed to deficiency in the airway protective mechanism, impairment in laryngeal and pharyngeal mucosal sensation, dysfunction in pharyngeal constriction, and disturbances in upper esophageal sphincter pressure.^{5–8} Heitmiller et al in 2000 reported aspiration in 38% and laryngeal penetration in 12% of patients with unilateral vocal fold paralysis. The authors attributed this high rate to the loss of airway protection rather than to esophageal dysfunction.⁶ These figures were corroborated 2 years later by

Nayak et al who reported penetration in 44.8% and aspiration in 23.9% of patients with unilateral vocal fold immobility. The study was conducted on 67 patients using videofluoroscopic examination, highlighting other causes for aspiration aside from glottal incompetence, namely reduced laryngeal movement, delay in swallowing, and bolus residues.⁹

No previous study has examined the link between dysphagia and dysphonia in patients with non-neoplastic vocal fold pathology, namely exudative lesions of the lamina propria and mucosal lesions and/or inflammation (polyps, nodules, Reinke edema, hemorrhage, scarring, granuloma, and laryngitis). Dysphagia may be a common symptom that is often overlooked in patients presenting with the main complaint of dysphonia. Improved awareness regarding the prevalence of dysphagia in this group of patients may have an impact on the management strategy. Similar to cases of unilateral vocal fold paralysis where swallowing dysfunction has been proven to be common and as important as dysphonia, patients with dysphonia secondary to non-neoplastic vocal fold pathology may suffer from impaired swallowing that is often masked by the phonatory complaint and yet is of major concern to these patients.³

The purpose of this investigation is to examine the prevalence of dysphagia in patients with the presenting symptom of dysphonia and diagnosed with non-neoplastic vocal fold pathology in a tertiary referral center. Given the intricacy in the neuromuscular supply of the pharynx and larynx, we hypothesize a high prevalence of dysphagia in this group of patients in comparison to a control group.

METHODS

After having obtained the approval from the institutional review board of the American University of Beirut Medical

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Center, all consecutive patients presenting to the Voice Unit between October 2016 and August 2017 with the presenting symptom of dysphonia and diagnosed with non-neoplastic vocal fold pathology were invited to participate in this study. A control group, matched according to age and gender and with normal laryngeal examination or no history of dysphonia, was included. These were patients presenting to the ENT clinic with no symptoms related to the laryngopharyngeal complex. A control group matched according to age and gender was included. Patients with recent history of respiratory tract infection, laryngeal surgery, or manipulation were excluded. Patients with history of neurologic disorders, head and neck tumors, and chemotherapy/radiotherapy were excluded as well. Demographic data included age, gender, and smoking status. Etiology of dysphonia in the case study group was recorded. The assessment of dysphagia in this study was made using Eating Assessment Tool (EAT)-10, which is a self-administered questionnaire developed for subjective assessment of dysphagia.¹⁰ Patients with a score above three were considered to have dysphagia based on the normative data derived from a large cohort study conducted on healthy individuals who did not have any history of airway, swallowing, voice, neurologic, or neoplastic disorders.¹⁰ The Voice Handicap Index (VHI)-10 described by Rosen et al and which consists of 10 self-administered questions was also reported.¹¹

Statistical method

Descriptive statistics was used to compute the means and the standard deviation of the continuous variables and the frequencies of the categorical variable. Mann-Whitney *U* test was used to compare the means of the continuous variables between patients and controls. Data were analyzed using SPSS version 23 (IBM Corp, Armonk, NY).

RESULTS

Demographic data and etiology

A total of 70 patients were enrolled in this study stratified as 45 with dysphonia and 25 as controls matched according to age and gender (Table 1).

TABLE 1.
Demographics

	Patients	Controls
Age (years)		
Mean $\pm$ SD	48.23 $\pm$ 14.65	43.68 $\pm$ 14.66
Range	24-84	16-17
Gender		
Male	18	11
Female	27	14
Smoking status		
Smokers	21 (46.67%)	10 (40%)

Abbreviation: SD, standard deviation.

The most common vocal fold pathology in patients with dysphonia was Reinke edema (28.8%), followed by laryngitis/mucosal inflammation (24.4%), and vocal fold nodules (17.7%) and polyps (13.33%) (Table 2).

Prevalence of dysphagia and VHI-10 score in patients versus controls

Of 45 patients with dysphonia, 37.7% had dysphagia as evidenced by an EAT-10 score of above three. In the control group, only 2 of 25 had dysphagia. The difference between the two groups was statistically significant ($P = 0.008$). Of the 45 patients with dysphonia, 27 had a VHI-10 score above or equal to 11. The total mean of VHI-10 in the total group was 13.66 ± 7.99 in patients with dysphonia and 1.25 ± 2.10 in controls (Table 3).

DISCUSSION

The prevalence of dysphagia in patients with dysphonia secondary to non-neoplastic vocal fold pathology has not been previously reported in the literature. The results of this investigation clearly indicates a significantly higher prevalence of dysphagia in patients compared to controls ($P < 0.05$), with two fifth having reported difficulty in swallowing as evidenced by the EAT-10 score of above three. This figure is also higher than the prevalence of dysphagia reported in the literature.¹²⁻¹⁵ Although population-based studies are rare, the prevalence of dysphagia is estimated to be between 16% and 22%.¹ Eslick and Talley reported that close to 16% (110 of 672) of a random sample of people in Sydney complained of dysphagia,¹⁶ whereas another study by Lindgren et al showed that obstructive symptoms occur in about 3% of men and women between the ages of 50 and 79 years.¹⁷

The pathophysiology of dysphagia in patients with dysphonia lies heavily on the intersection in the neuromuscular innervation of the upper airway and digestive system. In the pharyngeal phase of swallowing, there is anterior movement and elevation of the larynx, epiglottic closure, pharyngeal muscle contraction, and upper esophageal sphincter release. A dysfunction in any of these stages may result in swallowing disorder. In patients with unilateral vocal fold paralysis,

TABLE 2.
Etiology of Dysphonia

Etiology	
Vocal fold pathology	45
Bamboo	1
Cyst	1
Nodules	8
Polyp	6
Hemorrhage	1
Reinke edema	13
Granuloma	2
Laryngitis	11
Scarring	2

TABLE 3.
Comparison of the Prevalence of EAT-10 and VHI-10 Among Patients and Controls

		Organic (N = 45)	Controls (n = 25)	PValue
EAT-10	Mean $\pm$ SD	2.67 $\pm$ 3.99	0.65 $\pm$ 1.71	0.008 (<0.05)
	Prevalence, N (%)	17 (37.7%)	2 (8%)	
VHI-10	Mean $\pm$ SD	13.66 $\pm$ 7.99	1.25 $\pm$ 2.10	0.000 (<0.05)
	Prevalence, N (%)	27 (60%)	0	

Abbreviation: SD, standard deviation.

for instance, there is alteration in the pharyngeal constriction ratio and upper esophageal sphincter opening as reported by Domer et al.⁵ The study was conducted on 25 patients with unilateral vocal fold immobility using videofluoroscopic examination. In conclusion, the authors alluded to the presence of additional biomechanical alterations in swallowing aside from glottic incompetence.⁵ Similar results were reported by Jang et al in their videofluoroscopic study in a group of 28 patients with vocal fold paralysis. The authors reported abnormalities in both the oral and pharyngeal phases of swallowing, namely a delay in the trigger of the pharyngeal swallow and upper esophageal sphincter release.⁷

The etiology behind the high prevalence of dysphagia in patients with non-neoplastic vocal folds pathology has not been previously described and surely warrants further investigation. It is common knowledge that these patients invariably exhibit hyperfunctional laryngeal behavior, which in turn accelerates the stress at the midmembranous portion of the vocal folds and results in the development and perpetuation of vocal fold lesions.^{18,19} In keeping with the aforementioned, the pathogenic role of laryngeal tension in patients with benign vocal fold lesions has been described in a study by Hsiung and Hsiao.²⁰ The authors reported that 90% of patients with exudative lesions of the vocal folds and undergoing suspension microlaryngeal surgery exhibited at least one muscle tension dysphonia (MTD) characteristic feature. In that study, the MTD patterns described by Morrison and Rammage²¹ were assessed using frame by frame analysis of videolaryngeal stroboscopic recordings.²⁰ With that premise at hand, one can extrapolate that the hyperfunctional laryngeal behavior and MTD characteristic patterns seen in patients with non-neoplastic vocal fold lesions, although not documented in this study, may disturb one or more of the kinematics of swallowing. Possible other mechanisms include restricted movement of the laryngeal framework, restricted pharyngeal constriction during swallowing, or alteration in the upper esophageal sphincter pressure during phonation as reported by Perera et al in their study on 17 subjects who were asked to perform tasks at different frequencies and intensities.²² These suggested mechanisms remain hypothetical in the absence of videofluoroscopic or manometric studies of swallowing.

Another potential confounding variable for the high prevalence of dysphagia in patients with dysphonia and non-neoplastic vocal folds lesions is smoking. There are numerous studies in the literature that concur the strong association

between smoking and reflux disease. In a population-based study performed in 1745 students using structured questionnaires, Chen et al showed that cigarette smoking, in addition to other factors such as allergy and asthma, independently correlated with the prevalence of symptoms of reflux with an odds ratio of 1.53 (95% confidence interval).²³ Similar results were reported by Fujiwara et al in a cross-sectional study on 2680 subjects. The authors reported a strong association between smoking and overlaps between gastroesophageal reflux disease, functional dyspepsia, and irritable bowel syndrome.²⁴ Suggested mechanism include the inadvertent effect of cigarette smoking on the pharyngo-upper esophageal sphincter contractile reflex and pharyngoglottal closure reflex, which are considered as protective to the airway.²⁵ Based on a study by Smit et al using 24-hour double-probe pH monitoring, smokers had a pH lower than four at the level of the upper esophageal sphincter and above the lower esophageal sphincter for a longer period than nonsmokers, thus alluding again to the adverse effect of smoking on the protective barriers to the laryngeal inlet.²⁶ Although in our investigation there was no statistically significant difference in the prevalence of smoking between the case study group and controls, the high percentage of smokers in both groups and its association with gastroesophageal reflux should be considered in the interpretation and understanding of dysphagia in affected patients. Further investigation on the pathophysiology of dysphagia in patients with dysphonia secondary to non-neoplastic vocal fold pathology is needed.

The results of this study carry clinical significance in regard to treatment. Traditionally, with the purpose of improving voice quality, voice therapy has been geared to reduce the excessive laryngeal tension using breathing exercises, semiocclusive exercises, and the resonant voice. With the increased prevalence of dysphagia in patients with dysphonia, a physician may want to consider swallowing therapy in addition to voice therapy as a treatment modality. Circumlaryngeal manual therapy commonly performed in patients with laryngeal hyperfunctional behavior, be it primary or secondary, may also be considered in the treatment of dysphagia in these patients.

This study has two main limitations; one is the lack of information on the prevalence of laryngopharyngeal reflux disease, which is a confounding disease reported to be present in up to 50% of patients with dysphonia,²⁷ and another is the lack of objective measure of dysphagia, given the

limitations in the resources available to us. A third limitation is the relatively small number of subjects enrolled in this study.

CONCLUSIONS

Two of five patients with dysphonia secondary to non-neoplastic vocal folds pathology reported dysphagia. This high percentage alludes to the pathogenic role of the laryngeal behavior in the development of swallowing symptoms in this group of patients. The potential benefit of voice and swallowing therapy in the treatment of these patients should be considered.

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