Comorbid Dysphagia and Dyspnea in **Muscle Tension Dysphonia: A Global** Laryngeal Musculoskeletal Problem

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Abstract

Objective. To characterize the associated symptoms of dysphagia and dyspnea among patients presenting with muscle tension dysphonia (MTD).

Study Design. Retrospective chart review performed over a 14-month period from October 2014 to December 2015.

Setting. Voice and swallowing center of a tertiary academic medical center.

Subjects and Methods. Thirty-eight patients with MTD were included for analysis. Clinical data were collected and analyzed, including perceptual voice evaluation and patientreported outcomes measures.

Results. Among patients with a diagnosis of MTD, the incidence of reported dysphagia during clinical history and examination was 44.7%. Among patients with MTD, 60.5% had an EAT-10 (10-item Eating Assessment Tool) score \geq 3 (ie, abnormal). Patients who reported dysphagia and/ or had abnormal EAT-10 score (\geq 3) had significantly greater voice impairment than that of patients without dysphagia (P = .02). Patients who reported dysphagia also had significantly higher Clinical COPD Questionnaire scores than those of patients who reported only dysphonia (P = .002).

Conclusions. Patients presenting for dysphonia who are diagnosed with MTD have a high rate of comorbid dysphagia. Patients who reported dysphagia had significantly higher self-reported voice impairment and greater severity of breathing dysfunction as measured by the Clinical COPD Questionnaire. The coincidence of these symptoms in this patient cohort may suggest an underlying pathophysiology that has yet to be elucidated. Further prospective studies are needed to clarify the underlying cause of dysphagia and breathing dysfunction in the setting of MTD and to investigate diagnostic and therapeutic paradigms.

Keywords

muscle tension dysphonia, muscle tension dysphagia, muscle tension dyspnea

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he 3 main functions of the larynx—respiration, phonation, and airway protection-are inseparable in function. As a result, laryngeal disorders and surgery often affect all 3 functions with varying severity. Muscle tension dysphonia (MTD) is a voice disorder characterized by excessive tension of the intrinsic and extrinsic laryngeal musculature.¹ This tension can result in altered position and inclination of the laryngeal cartilages and hyperfunction of intrinsic laryngeal muscles, resulting in dysphonia.¹ Primary MTD occurs without organic vocal fold pathology or neurologic cause, and secondary MTD develops in response to concurrent organic vocal fold pathology. Three main etiologic categories have been cited: psychological/personality factors (introversion, anxiety, depression),² vocal abuse/ misuse,³ and compensation for an underlying organic disease (vocal fold lesions, laryngopharyngeal reflux [LPR], presbylaryngis, upper respiratory tract infection).⁴⁻⁶ In addition to dysphonia, patients with MTD most commonly describe vocal fatigue, vocal strain, and pain associated with phonation.³

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Swallowing is a complex process involving precise neurologic coordination of multiple muscles, soft tissue, and cartilaginous structures. The pharyngeal phase of swallowing is initiated when the suprahyoid muscles and tongue muscles are activated to elicit hyolaryngeal excursion (movement of the larynx superiorly and anteriorly during the swallow), which also elicits airway protection by sealing the laryngeal inlet. The bolus is propelled toward the esophagus through the coordinated contraction of the middle and inferior pharyngeal constrictors with the contraction of the styloglossus, which retracts the tongue base, while the longitudinal pharyngeal muscles (stylopharyngeus and salpingopharyngeus) activate to contract the pharynx. As the bolus reaches the esophagus, the pharyngoesophageal segment (composed of the thyropharyngeal and cricopharyngeal portions of the inferior pharyngeal constrictor) relaxes and is actively pulled open via hyolaryngeal excursion, which pulls the cricoid cartilage away from the posterior pharyngeal wall. Most muscles involved in the pharyngeal phase of swallowing insert into the hyoid bone, thyroid, and cricoid cartilages.⁷ The vocal fatigue reported among patients with MTD may be the result of the maladaptive overuse of the extrinsic laryngeal musculature, as the intrinsic laryngeal muscles are considered to be resistant to fatigue.⁸⁻¹⁰ The general physical tension of these muscles may also contribute to the strain and pain that patients with MTD report during vocal use.¹¹ Therefore, the strain and fatigue of the extrinsic larvngeal muscles may contribute to comorbid swallowing dysfunction.

In addition to clinical history, physical examination, and instrumental swallow study, patient-reported assessments are helpful in the characterization and quantification of laryngeal dysfunction and associated quality of life.¹² The 10item Eating Assessment Tool (EAT-10) is a validated patient-reported symptom-specific outcome tool for dysphagia.¹³ A mean \pm SD score of 0.40 \pm 1.01 is considered normal, while a score \geq 3 is abnormal and may be indicative of a swallowing disorder. Use of patient-reported outcome measures, including the EAT-10, Voice Handicap Index–10 (VHI-10),¹⁴ and Clinical COPD Questionnaire (CCQ),¹⁵ assists clinicians in characterizing patient symptoms and tracking changes in these symptoms over time.

Historically, MTD was categorized as functional dysphonia given the lack of organic physiologic parameters available to characterize the disease. Newer diagnostic tools, including stroboscopy, laryngeal electromyography, and electroglottography, have identified key abnormalities that distinguish MTD from normal laryngeal physiology. Videostroboscopic findings such as posterior open chink, supraglottic contraction, and anterior-posterior contraction with reduced epiglottic-arytenoid distance,¹⁶ as well as abnormally elevated hyoid and laryngeal position during phonation, were observed among patients with MTD as compared with controls.¹⁷

Muscle tension dysphagia (MTDg) was identified as a distinct clinical entity, defined as dysphagia with normal videofluoroscopic swallowing study (VFSS) results and excessive laryngeal tension.¹⁸ The degree and nature of

overlap between MTD and MTDg are unclear. The purpose of this study was to characterize the prevalence and severity of coincident swallowing and breathing dysfunction among patients with a primary diagnosis of MTD.

Methods

Following University of Virginia Institutional Review Board approval, a retrospective chart review was completed of all patients with a diagnosis of dysphonia. Patients with an additional diagnosis of MTD were included, and 81 patients were identified with dysphonia and a diagnosis of MTD at the University of Virginia Voice Center from October 2014 to December 2015. Diagnosis of MTD was determined with clinical impression based on history, physical examination, and laryngoscopy findings. Exclusion criteria included absent patient-reported outcome measures, anatomic etiology of dysphagia, known esophageal pathology or prior esophageal surgery that cannot be excluded as cause of dysphagia, prior radiation, and history of head and neck cancer. Thirty-eight patients were included in the analysis. Perceptual voice assessment was completed for all patients and characterized with the GRBAS scale (grade, roughness, breathiness, asthenia, strain).¹⁹ Demographic information was also obtained.

Patient-reported dysphagia was defined as self-reporting difficulty swallowing during clinical history and/or examination. In addition, all patients completed validated patient-reported outcome measures (VHI-10, EAT-10, CCQ). Patients were included in the dysphagia/dysphonia cohort if they reported dysphagia during clinical history and examination or had an EAT-10 score \geq 3. Treatment effect was assessed by physical therapy and voice therapy documentation to assess whether patients exhibited worsened symptoms, no change in symptoms, improvement without resolution, or symptom resolution. Subsets of the patient cohort were compared with unpaired *t* tests and chi-square tests.

Results

Caucasian females represented 68% of the patient cohort (**Table 1**). The mean age was 52 years. The coincidence of patient-reported dysphagia (n = 17) among patients with MTD was 44.7% (**Table 2**). The mean EAT-10 score was 7.6; 60.5% (23 of 38) of patients scored \geq 3, with 31.6% (12 of 38) scoring \geq 10. **Table 3** compares patients with MTD and dysphagia (in addition to their chief complaint of dysphonia) by report or abnormal EAT-10 score (\geq 3) and those with only dysphonia. Patients who reported dysphagia had significantly greater mean self-reported voice impairment (17.1) than that of patients without dysphagia (10.5, *P* = .02). The dysphagia/dysphonia group did not have higher perceptual assessment scores than those of the dysphonia-only cohort as rated with the GRBAS scale (*P* = .20).

Patients with MTD and dysphagia also had significantly higher CCQ scores (mean = 2.2) as compared with the dysphonia-only cohort (0.8, P = .002). After removal of all patients (n = 11) with chronic obstructive pulmonary disease,

Table 1. Patient Characteristics.^a

	n (%)
Sex	
Female	30 (78.9)
Male	8 (21.1)
Race	
White	33 (86.8)
Black	3 (7.9)
Hispanic	2 (5.3)

^aMean age, 52.0 years.

Table	2.	Dysphagia	in	MTD	among	Patients	(N =	= 38).
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Diagnosis or Reported Symptom	n (%)
MTD	
Primary	20 (52.6)
Secondary	18 (47.4)
Patient-reported dysphagia	17 (44.7)
EAT-10 score	
3-9	II (28.9)
\geq IO	12 (31.6)
Patient-reported dysphagia or EAT-10 \geq 3	25 (65.8)
Thyrohyoid tenderness ^a	16 (45.7)
EGD	6 (15.8)
VFSS	3 (7.9)

Abbreviations: EAT-10, 10-item Eating Assessment Tool; EGD, esophagogastroduodenoscopy; MTD, muscle tension dysphonia; VFSS, videofluoroscopic swallowing study.

^aPatients, n = 35.

asthma, and smoking history from both groups, the dysphagia/dysphonia group still had significantly higher CCQ scores (mean = 1.9) versus the dysphonia-only group (0.7, P = .03). Patients with MTD and dysphagia reported more frequent cough (mean cough question score: 3.2 vs 2.0, P = .010). In addition, patients with MTD and dysphagia had more severe symptomatic breathing dysfunction based on the symptomatic domain of the CCQ (9.8 vs 4.4, P = .002). After removal of the 2 symptomatic domain questions regarding cough and phlegm (Nos. 5 and 6), patients with dysphagia still reported more severe dyspnea (mean score: 4.6 vs 2.0, P = .008).

There were no statistically significant differences in comorbidities between the dysphagia/dysphonia and the dysphonia-only groups (**Table 4**).

In the dysphagia/dysphonia group, 24 of 25 patients underwent voice and/or physical therapy for dysphonia related to MTD with the anticipation that their dysphagia symptoms would improve as well (1 patient deferred due to medical illness). After treatment, most patients saw either symptom improvement or resolution (**Table 5**).

Patients were evenly distributed in the MTD diagnostic categories: 52.6% for primary MTD versus 47.4% for

secondary MTD (**Table 6**). Patients with primary MTD reported dysphagia more often than patients with secondary MTD (P = .01) and had greater incidence of thyrohyoid tenderness (P < .001). Differences in scores between groups on all questionnaires were not significantly different.

Discussion

MTDg was recently described as a distinct clinical entity defined by dysphagia, normal VFSS results, and excessive laryngeal tension.¹⁸ In this study, we sought to characterize the coincidence of patient-reported dysphagia and breathing symptoms among patients with MTD. We describe a subset of the MTD population with excessive laryngeal tension and dysphagia. As compared with patients with dysphonia only, patients with dysphagia/dysphonia had higher self-reported voice impairment and higher self-reported breathing dysfunction. This suggests that MTDg and dyspnea could be part of a more global laryngeal musculoskeletal disorder when present with MTD. The constellation of symptoms appears to represent those of a unique group of patients with severe intrinsic and extrinsic laryngeal muscle tension, resulting in muscle tension dysphonia, dysphagia, and dyspnea (MTD³).

The CCQ was used to assess patient function as it relates to respiratory function and cough. Ex-smokers with normal spirometry results have a total CCQ of roughly 0.8.¹⁵ The CCQ was validated for adult patients with laryngotracheal stenosis; patients with Cotton-Myer grades I/II had a mean CCQ of 1.96.²⁰ The scores of the dysphagia/dysphonia and dysphonia-only groups are consistent with previous research on patients with chronic respiratory disease and healthy patients, respectively.²¹⁻²³ At their initial assessment, patients with dysphagia/dysphonia scored a mean 2.2, while patients with dysphonia scored only 0.8. After removal of 11 patients with chronic obstructive pulmonary disease, asthma, and smoking history, the mean CCQ among patients with dysphagia/dysphonia was still significantly higher (1.8) as compared with the dysphonia-only group (0.7). In addition, after removal of potential confounding questions focusing on cough and phlegm, the symptomatic breathing dysfunction of patients with dysphagia was still worse than those without dysphagia. The severity of the CCQ scores in this cohort was alarming, as it approached those of mild/moderate respiratory disease and laryngotracheal stenosis. Whether these findings represent a true decrease in respiratory capacity, comorbid paradoxical vocal fold motion or dysfunctional breathing from extrinsic laryngeal muscle and respiratory muscle tension is unclear and warrants investigation.

Analysis of the primary versus secondary MTD groups revealed that patients with primary MTD have higher rates of dysphagia and more severe extrinsic laryngeal muscle tension, as evidenced by thyrohyoid tenderness on physical examination. The increased extrinsic laryngeal muscle tension of patients with primary MTD may be related to increased reports of dysphagia, but further investigation is needed to determine the significance of the relationship.

Table 3. Dysphonia vs Dysphagia/Dysphonia: Patient-Reported Assessments.^a

Assessment Dy:	sphonia (n = 13)	Dysphonia/Dysphagia (n = 25)	P Value
	10.5	17.1	.03
GRBAS grade	1.6	2.0	.20
EAT-10	0.27	11.30	<.001
CCQ	0.75	2.22	.002
Symptoms	1.10	2.46	.002
"Did you cough?"	2	3.2	.01
Symptomatic domain less cough/phlegm questions (Nos. 5 and 6)	2	4.6	.008
Functional	0.42	2.05	.01
Mental	0.73	2.08	.02

Abbreviations: CCQ, Clinical COPD Questionnaire; EAT-10, 10-item Eating Assessment Tool; GRBAS, grade, roughness, breathiness, asthenia, strain; VHI-10, Voice Handicap Index–10.

^aValues are presented as means. P values are based on unpaired t test. Bold indicates significance, P < .05.

Table 4. Patient Comorbidities.^a

Comorbidity	Patients, n (%)			
	All (N = 38)	Dysphonia (n = 13)	Dysphagia/Dysphonia (n = 25)	P Value
Depression/anxiety	17 (44.7)	4 (30.8)	3 (52.0)	.25
Cervical spine disease	5 (13.2)	I (7.7)	4 (16.0)	.50
Fibromyalgia	2 (5.3)	0 (0.0)	2 (8.0)	.31
IBS	3 (7.9)	0 (0.0)	3 (12.0)	.21
Migraine	2 (5.3)	0 (0.0)	2 (8.0)	.31
GERD	20 (52.6)	8 (61.5)	12 (48.0)	.43
Allergic rhinitis	8 (21.1)	3 (23.1)	5 (20.0)	.74
CRS	3 (7.9)	I (7.7)	2 (8.0)	.97
Smoking	3 (7.9)	0 (0.0)	3 (12.0)	.21
Asthma	9 (23.7)	4 (30.8)	5 (20.0)	.38
Chronic pain	2 (5.3)	0 (0.0)	2 (8.0)	.31

Abbreviations: CRS, chronic rhinosinusitis; GERD, gastroesophageal reflux disease; IBS, irritable bowel syndrome. ^aP values are based on chi-square test. Significance at P < .05.

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Treatment	Patients, n (%)
Voice therapy ^a	24
Resolved	17 (70.8)
Improved	6 (25.0)
Lost to follow-up	I (4.2)
Physical therapy ^b	7
Resolved	2 (28.6)
Improved	2 (28.6)
No improvement	I (14.3)
Lost to follow-up	2 (28.6)

Table 5.
Treatment
Outcomes
in
the
Dysphagia/Dysphonia

Cohort.

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^aTwenty-five patients referred for voice therapy, and 24 were treated. ^bThirteen patients referred for physical therapy, and 7 were treated.

The question remains regarding the underlying physiology of dysphagia in this cohort. Gastroesophageal reflux disease (GERD) can cause dysphagia through a variety of mechanisms, including esophagitis,²⁴ motility disturbance,²⁴ cricopharyngeal hypertrophy,²⁵ Zenker's diverticulum,²⁵ cricopharyngeal spasm,²⁶ and esophageal stricture.²⁷ One study reported that 65% of patients with moderate to severe GERD reported dysphagia,²⁸ and 78% of patients with MTD were shown to have LPR.⁶ The incidence of self-reported GERD in the MTD population in the current study—dysphagia/dysphonia (48%) and dysphonia only (52.6%)—is not as high as previously recorded. There was also no significant difference between cohorts. Without better characterizing the presence and severity of GERD or LPR through clinical testing (eg, 24-hour pH probe impedance testing), the role of GERD or LPR in either group in this population remains unclear.

Increased muscle tension of the extrinsic laryngeal muscles may contribute to dysphagia among patients with MTD, owing to the close anatomic and functional relationship of the voice and swallowing systems. Extrinsic

Table 6. Primary vs Secondary MTD.^a

	Primary MTD (n = 22)	Secondary MTD (n = 18)	P Value
Patient-reported symptomology, n (%)			
Symptomatic dysphagia	12 (54.5)	5 (27.8)	.01
Thyrohyoid tenderness	14 (63.6)	2 (13.3)	<.00 l
EAT-10			
\geq 3	14 (63.6)	9 (50.0)	.102
>10	9 (40.9)	3 (16.7)	.07
Mean assessment score			
VHI-10	15.2	14.4	.80
EAT-10	9.3	5.6	.17
CCQ	1.8	1.6	.72
Etiologies of secondary MTD, n ^b			
Irritable larynx syndrome	6		
Glottic insufficiency	5		
Inflammatory	4		
Vocal fold pathology	3		
Dehydration	2		
Neck pain	I		

Abbreviations: CCQ, Clinical COPD Questionnaire; EAT-10, 10-item Eating Assessment Tool; MTD, muscle tension dysphonia; VHI-10, Voice Handicap Index–10.

^aP values are based on chi-square test (patient-reported symptomology) and unpaired *t* test (mean assessment scores). Bold indicates significance, P < .05. ^bTwenty-one etiologies for 18 patients.

laryngeal muscle tension may create dysphagia due to the limitation of hyolaryngeal elevation during deglutition. While limited research showed that patients with MTD have abnormal hyolaryngeal elevation,¹⁷ further assessment of extrinsic laryngeal muscle tension through electromyography or other means is warranted.

Upper esophageal sphincter (UES) dysfunction is another potential cause of dysphagia in this population. Van Houtte et al²⁹ investigated this among 14 patients with MTD versus 14 controls, using manometry. No statistically significant difference was found between absolute UES pressure among patients with MTD and controls at rest. UES pressure among patients with MTD increased during all types of phonation tested (varying by a factor of 1.29-1.7 vs rest), while UES pressure among controls stayed relatively stable versus rest (0.91-1.23). These differences were not statistically significant except for high pitch phonation (1.7 vs 0.91, P =.027).²⁹ These data suggest that although patients with MTD may lack baseline cricopharyngeal tension as compared with controls, they may experience a hyperfunction of cricopharyngeus upon nearby muscle activation. In addition, baseline UES pressure obtained in the study was significantly lower than reported UES baseline values, typically between 100 and 150 mm Hg.30 Prospective studies of manometry, pH/ impedance, and extrinsic laryngeal muscle electromyography in this population will help determine the relative importance of these components of deglutition on dysphagia and dysphagia-related symptoms in this population.

This patient population was treated with a combination of medical management, voice therapy, and physical therapy. In general, our treatment algorithm is to treat comorbid conditions affecting laryngeal inflammation and to refer all appropriate patients to voice therapy. For those with severe symptoms or excessive laryngeal tension on examination, we additionally refer to a physical therapist who performs manual therapy designed to release neck and laryngeal muscle tension. Based on the proportion of patients with dysphagia/dysphonia who improved or resolved in each treatment group, voice therapy alone appears effective for patients with less severe symptoms. In the group with more severe symptoms requiring bimodal therapy, roughly 50% of those who sought treatment showed symptom improvement or resolution.

Our study is limited by its small sample size and retrospective nature. Given that our patient cohort consisted of those presenting with a chief complaint of dysphonia who were diagnosed with MTD, few of our patients presented with VFSS or other evaluations of swallowing. However, most patients improved with voice therapy alone, suggesting a nonanatomic cause of reported dysphagia symptoms. As such, we cannot definitively rule out cricopharyngeal hypertrophy and other anatomic causes of dysphagia in this population or make direct comparisons between our patients and those from Kang et al,¹⁸ all of whom had normal VFSS results. Our study is limited in other forms of objective evaluation, including pH probe and impedance testing. In addition, many of our patients did not return to see our laryngologist upon completion of treatment. As such, we were not able to assess degree of improvement in reported outcomes of voice, swallowing, and dyspnea after treatment.

Conclusion

MTDg was recently described as a new clinical entity. We sought to characterize the incidence and nature of dysphagia and breathing-related symptoms in the MTD population. Approximately 50% of patients with MTD reported dysphagia and had abnormal EAT-10 scores. Patients with MTD who also reported dysphagia had significantly higher selfreported voice and breathing impairment as compared with patients with MTD who reported dysphonia only. These patients with MTD₃ (muscle tension dysphonia, dysphagia, and dyspnea) may be a distinct classification from either MTD or MTDg. Alternatively, patients with MTD₃ may represent a more severe variant of MTD that could be related to a global laryngeal tension syndrome. In alignment with prior reports, voice therapy and physical therapy are effective for treating the symptoms of excessive laryngeal muscle tension and may have reduced reported dysphagia symptoms. Further prospective research would elucidate the underlying pathophysiology of dysphagia and dyspnea in this population, as well as clarify ideal treatment regimens.

Author Contributions

Patrick O. McGarey Jr, designed study with Daniero, collected clinical chart review, interpreted data and drafted manuscript, edited manuscript; Nicholas A. Barone, drafted sections of manuscript, extensive editing of tables and manuscript references; Michael Freeman, assisted in chart review and manuscript editing; James J. Daniero, designed study with McGarey, helped obtain Institutional Review Board approval, helped obtain clinical data, assisted with interpretation of data, helped edit manuscript.

Disclosures

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