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Prevalence of dysphagia in patients with muscle tension dysphonia

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Abstract

Background. It is hypothesised that patients with muscle tension dysphonia have a high prevalence of dysphagia in comparison to normative values reported in the literature.

Methods. This prospective study included 44 subjects diagnosed with muscle tension dysphonia, based on symptoms and laryngoscopic findings, and 25 control subjects with no history of dysphonia and normal laryngeal examination findings. Demographic data included age, gender and smoking history. The aetiology of muscle tension dysphonia was classified as primary or secondary. Evaluation involved the Eating Assessment Tool ('EAT-10') questionnaire.

Results. Patients' mean age was 45.93 ± 14.95 years, with a female to male ratio of 1.2:1. Fourteen patients had primary muscle tension dysphonia, while 30 had secondary muscle tension dysphonia. Among patients with secondary muscle tension dysphonia, Reinke's oedema was the most common aetiology. There was a significant difference in the prevalence of dysphagia between the study group and the control group (40.9 per cent *vs* 8 per cent respectively, $p < 0.05$).

Conclusion. This study demonstrates a higher prevalence of dysphagia in patients with the presenting symptom of dysphonia and diagnosed with muscle tension dysphonia in comparison to subjects with no dysphonia.

Introduction

Muscle tension dysphonia is a functional voice disorder characterised by a spectrum of laryngopharyngeal symptoms in the presence of extrinsic and intrinsic laryngeal muscle constriction. It is categorised as either primary or secondary, depending on the absence or presence of laryngeal structural changes and/or neurogenic disorders.[1] A common pathophysiology to both is excessive muscle tension with an imbalance in laryngeal muscle activity. This hyperkinetic laryngeal behaviour may lead to excessive stress at the membranous portion of the vocal folds, resulting in the development, and/or exacerbation or perpetuation, of pre-existing lamina propria lesions.[2]

Affected individuals are invariably under a significant level of stress and vocal demand, with rates up to 19 per cent and 86 per cent respectively.[1] Allergy and reflux have been reported as aetiological factors contributing to this disease entity. Based on a study by Altman *et al.*, almost 50 per cent of patients with muscle tension dysphonia have symptoms of gastroesophageal reflux disease and 37 per cent have a history of allergy.[1]

Given the contiguity of the laryngeal framework to the pharyngeal structures, and considering that dysphagia may also be caused by an imbalance in pharyngeal muscle activity, we decided to investigate the prevalence of dysphagia in patients with muscle tension dysphonia.

A PubMed literature review was conducted using the words 'dysphagia', 'dysphonia' and 'muscle tension dysphonia'. The search revealed only one study on the benefit of laryngeal manipulation in patients with muscle tension induced dysphagia, by Depietro *et al.*,[3] and two reports on the correlation between upper oesophageal sphincter (UOS) pressure and phonation.[4,5]

The study by Depietro *et al.* described improvement in dysphagia in 71.4 per cent of patients with muscle tension induced dysphagia with no anatomical cause.[3] Perera *et al.* investigated changes in UOS pressure in relation to intensity and pitch in a group of healthy volunteers who were asked to perform various phonatory tasks.[4] That study showed significant changes in UOS pressure at low and high pitches.

In keeping with the aforementioned, Van Houtte *et al.* hypothesised that patients with muscle tension dysphonia may have an increase in UOS pressure.[5] Surprisingly, the results of their cross-sectional study using manometry failed to demonstrate their hypothesis. The authors attributed the lack of significant difference in UOS pressure during phonation in muscle tension dysphonia patients versus controls to several factors. These included: the type of probe used, in terms of number of holes and the interspace between the sensors; the subtlety in the inclination of the thyroid and cricoid cartilage in muscle tension dysphonia patients and their effect on the UOS muscles; and the reduced phonatory capacity of muscle tension dysphonia patients, namely in terms of pitch and intensity range, in comparison to controls. This last factor was substantiated

Main Article

Dr A-L Hamdan takes responsibility for the integrity of the content of the paper

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by an increase in UOS pressure at high pitch in muscle tension dysphonia patients, compared to a relative decrease in matched control subjects.[5]

In line with these studies, and given the cross-cutting in the neuromuscular supply of the pharyngeal and laryngeal structures, the authors of this manuscript elected to investigate the prevalence of dysphagia in a group of patients with muscle tension dysphonia. The hypothesis was that patients with muscle tension dysphonia have a high prevalence of dysphagia, in comparison to a control group and to normative values reported in the literature.

Materials and methods

After obtaining institution review board approval, all patients who presented to the voice unit at a tertiary referral medical centre with dysphonia, and who were diagnosed with muscle tension dysphonia, between October 2016 and August 2017, were invited to participate in this study. Muscle tension dysphonia was diagnosed by the presence of dysphonia, sore throat and/or neck pain, in addition to the presence of hyperkinetic laryngeal behaviour. The latter can occur in the form of mediolateral compression of the supraglottic structures, antero-posterior compression or shortening of the distance between the petiole and interarytenoid area, or sphincter-like closure of the supraglottis during phonation.[1] A group of subjects matched according to age and gender, with no history of dysphonia and with normal laryngeal examination findings, was considered as the control group.

Subjects were excluded if they had: a history of upper respiratory tract infection; undergone recent laryngeal manipulation or surgery; a history of neurological disorders, or head and neck tumours; or a history of chemo/radiotherapy. Demographic data collected included age, gender and smoking history.

The 10-item Eating Assessment Tool ('EAT-10'), which is a self-administered questionnaire developed for the subjective assessment of dysphagia, was used as a primary outcome measure of dysphagia.[6] Patients with a score above 3 were considered to have dysphagia, based on the normative data derived from a large cohort study conducted on healthy individuals with no history of airway, swallowing, voice, neurological or neoplastic disorders.[6]

Statistical method

Descriptive statistics were used to compute the means and standard deviations of the continuous variables and the frequencies of the categorical variables. The Mann–Whitney U test was used to compare the means of the continuous variables between patients and controls. Data were analysed using SPSS statistical software, version 23 (SPSS, Chicago, Illinois, USA).

Results

Demographic data and aetiology

A total of 44 patients with muscle tension dysphonia were enrolled in this study. There were 20 males and 24 females, with an overall mean age of 45.93 ± 14.95 years. Fourteen patients had primary muscle tension dysphonia and 30 patients had secondary muscle tension dysphonia. The most common vocal fold pathology in patients with secondary muscle tension dysphonia was Reinke's oedema, followed by nodules and polyps (Table 1).

Table 1. Demographics and clinical characteristics of study population

Characteristic	Patients	Controls
Age (years)		
– Mean ± SD	45.93 ± 14.95	4.68 ± 14.66
– Range	24–73	16–77
Gender (*n*)		
– Males	20	11
– Females	24	14
Smoking (*n*)		
– Smokers	19	10
– Non-smokers	25	15
Secondary muscle tension dysphonia (*n*)	30	
– Bamboo	1	
– Cyst	1	
– Nodules	5	
– Polyp	4	
– Haemorrhage	2	
– Reinke's oedema	8	
– Oedema	4	
– Granuloma	1	
– Scarring	2	
– Lesions	2	
– Bamboo	1	
Primary muscle tension dysphonia (*n*)	14	

SD = standard deviation

Dysphagia prevalence

Of the 44 patients with muscle tension dysphonia, 40.9 per cent ($n = 18$) had dysphagia, as evidenced by an Eating Assessment Tool score above 3. Eight of these patients were in the primary muscle tension dysphonia group and 10 were in the secondary muscle tension dysphonia group.

Of the 25 controls, 8 per cent ($n = 2$) had dysphagia. There was a significant difference in the prevalence of dysphagia between the two groups ($p = 0.002$) (Table 2).

Discussion

Swallowing is a complex sensorimotor physiological process that transports saliva and ingested material from the mouth into the stomach.[7,8] It is commonly divided into four phases: the preparatory, oral, pharyngeal and oesophageal phases. The preparatory phase involves mastication and mixing the bolus with saliva. In the oral phase, the bolus is propelled from the oral cavity to the pharynx.[9] During the pharyngeal phase, bolus propulsion through the pharynx and into the UOS involves a posterior tongue drive combined with sequential contraction of pharyngeal constrictor muscles.[10] At the onset of swallowing, the hyoid bone moves anteriorly and superiorly. This movement induces anterior movement of

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Table 2. EAT-10 scores and dysphagia prevalence in study population

Parameter	Muscle tension dysphonia patients			Controls**	*P*-value
	Primary dysphonia*	Secondary dysphonia†	Total dysphonia‡		
EAT-10 score (mean ± SD)	6.93 ± 10.03	1.9 ± 2.95	3.5 ± 6.47	0.65 ± 1.71	0.002
Dysphagia prevalence (*n* (%))	8 (18.18)	10 (22.72)	18 (40.9)	2 (8)	<0.005

*$n = 14$; †$n = 30$; ‡$n = 44$; **$n = 44$. EAT-10 = 10-item Eating Assessment Tool; SD = standard deviation

the larynx that imparts anterior traction on the cricoid, which in turn exerts forward traction of the UOS, leading to its relaxation and passage of the bolus.[10] The final oesophageal phase is when the bolus moves from the oesophagus into the stomach.[7]

In patients with dysphagia there is difficulty in moving food from the mouth to the stomach. Although population-based studies are rare, the prevalence of dysphagia is estimated to be between 16 per cent and 22 per cent.[11–14] Dysphagia can be caused by a variety of conditions, including structural disorders, myopathies (polymyositis and dermatomyositis) and central nervous system disorders (Parkinson's disease and stroke).[15]

Several studies have analysed the kinematic motion of laryngopharyngeal structures during the pharyngeal phase of swallowing in patients with dysphagia. Pharyngeal muscle weakness, disturbed movement patterns of different laryngeal structures such as the hyoid bone and epiglottis, and reduced activity of UOS muscles have been implicated to various degrees depending on the aetiology of the dysphagia.[16] The cricopharyngeus muscle in particular, which is the main constituent of the UOS that keeps its constant basal tone at rest and enables its relaxation during swallowing, seems to play a crucial role in oropharyngeal dysphagia. Impairment in cricopharyngeal muscle activity, such as higher than normal residual pressure, can lead to the delayed onset of UOS relaxation, with consequent outflow obstruction.[17,18]

In patients with muscle tension dysphonia, there is also abnormal laryngeal muscle activity, with subsequent excessive tension. The disturbed laryngeal behaviour is described as a mediolateral or anteroposterior contraction of the supraglottic structures, with complete sphincter-like closure of the endolaryngeal structures in severe cases.[1] This hyperkinetic endolaryngeal behaviour, coupled with the high-positioned larynx, shortened thyrohyoid distance and disturbed angles of the laryngeal framework, result in a spectrum of vocal and neck symptoms.[5] Patients more often than not complain of a change in voice quality, vocal fatigue and an inability to project the voice during phonation. Together with these complaints, patients may experience ill-defined symptoms such as throat pain and discomfort.

The results of this investigation revealed the high prevalence of an additional obstructive symptom, namely dysphagia. Indeed, 40.9 per cent of patients with muscle tension dysphonia reported having dysphagia, as evidenced by an elevated Eating Assessment Tool score. It is important to note that dysphagia was a secondary complaint to dysphonia in all cases, unlike the study by Depietro *et al.* where dysphagia was a primary complaint in 36 of 44 dysphonia patients.[3]

Given that muscle tension dysphonia is caused primarily by an imbalance in laryngeal muscular activity, and that dysphagia is also caused by an imbalance in hypopharyngeal and oesophageal muscle activity, the high prevalence of dysphagia in muscle tension dysphonia patients is not surprising. Possible mechanisms for this high prevalence include: an increase in pharyngeal pressure or tension secondary to the increased laryngeal tension, and increased UOS pressure as hypothesised by Belafsky *et al.*[19] The abnormal laryngeal posture and movement of the laryngeal framework posteriorly can cause a 'squeeze' of the sphincter against the spine, with a subsequent increase in mechanical pressure. This hyperactivity or excessive contraction of the cricopharyngeal and thyropharyngeal muscles can affect: UOS pressure, resulting in dysphagia, and vocal fold length and tension, resulting in muscle tension dysphonia (by approximating the two thyroid laminae). These suggested mechanisms remain hypothetical given the lack of any manometric data on the UOS in this group of patients.

- This study compared dysphagia prevalence in muscle tension dysphonia patients versus non-dysphonia subjects
- It comprised 44 patients diagnosed with muscle tension dysphonia and 25 controls with no history of dysphonia and with normal laryngeal examination findings
- All subjects completed the Eating Assessment Tool ('EAT-10'), used as a primary outcome measure of dysphagia
- There was a significant difference in dysphagia prevalence between the two groups ($p < 0.05$), with a higher prevalence in muscle tension dysphonia patients

The results of this investigation carry clinical implications for the diagnosis and management of patients with dysphonia and dysphagia in the absence of an anatomical cause. Increased physician awareness of dysphagia as a secondary complaint in patients with a primary complaint of dysphonia is paramount in the management strategy. Aside from vocal hygiene and vocal resonant therapy, circumlaryngeal manual therapy might be a valuable addition to the treatment armamentarium for this subgroup of patients. The aim of therapy is to release the laryngeal contractures by lengthening the thyrohyoid laryngeal membranes, decreasing the tension within the constrictor muscles and restoring the symmetry.[5]

This study has two main limitations: namely, the relatively small size, and the lack of information on laryngopharyngeal reflux disease that is commonly reported in muscle tension dysphonia patients and which may accentuate dysphagia as a symptom. Nevertheless, this study provides further information on the significant interplay between dysphagia and dysphonia in patients with muscle tension dysphonia.

Conclusion

This study highlights the presence of an important obstructive symptom related to swallowing that is often underscored in patients with the presenting complaint of dysphonia who exhibit a laryngeal muscle tension pattern. The results indicate the high prevalence of dysphagia in patients with the

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presenting symptom of dysphonia and who have been diagnosed with muscle tension dysphonia. The pathogenic role of laryngeal muscle imbalance in dysphagia is suggested. Future studies using electromyography analysis of intrinsic and extrinsic laryngeal muscles in patients with muscle tension dysphonia and dysphagia may further elucidate the interplay between the two entities.

Competing interests. None declared

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