The Prevalence of Undiagnosed Thyroid Disease in Patients With Symptomatic Vocal Fold Paresis

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Summary: Objective. Vocal fold paresis has a multifactorial etiology and is idiopathic in many individuals. The incidence of thyroid-related neuropathy in the larynx has not been previously described. The purpose of this study was to evaluate the prevalence of previously undiagnosed thyroid disease in patients with laryngeal neuropathy and to compare this prevalence with that in a cohort of patients with a neurotologic neuropathy.

Study Design and Setting. Case series with chart review; tertiary care, otolaryngology practice.

Subjects and Methods. Charts of 308 consecutive patients with dysphonia and vocal fold paresis and 333 consecutive patients with sensorineural hearing loss, who presented for evaluation during a 3-year period, were reviewed.

Results. One hundred forty-six of 308 (47.4%) patients with vocal fold paresis were diagnosed with concurrent thyroid disease, whereas 55 of 333 (16.5%) patients with sensorineural hearing loss were diagnosed with concurrent thyroid disease ($P < 0.001$, Pearson chi-square = 92.896; degrees of freedom = 5). Thyroid diagnoses among those with vocal fold paresis included benign growths (29.9%), thyroiditis (7.8%), hyperthyroidism (4.5%), hypothyroidism (3.6%), and thyroid malignancy (1.6%).

Conclusions. Thyroid abnormalities are more prevalent in patients with dysphonia and vocal fold paresis than in patients with symptomatic sensorineural hearing loss, suggesting a greater association between previously undiagnosed thyroid abnormalities and laryngeal neuropathy than that between neurotologic neuropathy and thyroid disease.

Key Words: Dysphonia–Vocal fold paresis–Thyroid disease–Recurrent laryngeal nerve–Superior laryngeal nerve.

INTRODUCTION

The etiology of vocal fold paresis can be multifactorial and remains unknown in most individuals who have voice complaints related to vocal fold paresis. Classically, the association between vocal fold paresis and thyroid disease has been in cases of thyroid cancer, and this association is usually a result of direct involvement of the laryngeal nerve in the neoplastic process.[1] Most studies in the literature describe findings of vocal fold paresis in patients who present with thyroid pathology and have the larynx evaluated as a part of the diagnostic evaluation of the thyroid.[2] However, the prevalence of asymptomatic thyroid disease in patients who present with a primary complaint of dysphonia and with findings of vocal fold paresis is unknown.

There have been case reports describing vocal fold paralysis in patients with goiter and/or thyroiditis; however, there is limited data evaluating the incidence of thyroid disease in patients with symptomatic vocal fold paresis.[3] One study that attempted to identify whether such an association exists found the presence of thyroid disease in 42.8% (21 of 49) of patients with vocal fold hypomobility who presented for evaluation of dysphonia.[4] However, that study was limited by sample size and the absence of confirmatory electrophysiological data.

Thyroid disease is known to cause peripheral neuropathy, affecting both sensory and motor nerves, including those in the respiratory system.[5–10] Thyroid-related neuropathies tend to reverse with treatment of the thyroid disease; however, associated myopathies do not always improve with treatment of the thyroid disease.[6,8,10,11] Thyroid disease has been closely associated with sensorineural hearing loss, and routine testing of thyroid function and antithyroid antibody production has been recommended in the evaluation of patients with sudden or asymmetric sensorineural hearing loss and in the evaluation of patients with sensory or motor peripheral neuropathies.[5,11–13]

The prevalence of previously undiagnosed thyroid disease in patients with dysphonia and vocal fold paresis is unknown. The purpose of this study was to evaluate the prevalence of previously undiagnosed thyroid disease in patients with a primary complaint of dysphonia and a clinical diagnosis of vocal fold paresis as a contributing factor to their dysphonia. We investigated whether the prevalence of thyroid disease in this group differs significantly from a cohort of patients with a different cranial neuropathy that has been shown to be highly associated with thyroid disease, namely, sensorineural hearing loss.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board at Graduate Hospital, Philadelphia, PA. The charts of 1645 patients who presented to a tertiary, private voice and otolaryngology practice to meet one of the senior authors (Y.D.H.-A. or R.T.S.) over the course of a 3-year period (January 2005 to December 2007) for the evaluation of dysphonia were reviewed. Inclusion criteria for the study group included a clinical diagnosis of vocal fold paresis as a primary cause of the dysphonia. The diagnosis of vocal fold paresis was made at the time of

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TABLE 1.
Laryngeal Nerve Involvement in Patients With Dysphonia and Vocal Fold Paresis

Nerve Involvement	Unilateral Nerve Involvement	Bilateral Nerve Involvement	Total
Superior laryngeal nerve	189	65	254
Recurrent laryngeal nerve	3	1	4
Both superior and recurrent laryngeal nerves	30	20	50
Total	222	86	308

the initial evaluation using a combination of indirect mirror laryngoscopy, flexible laryngoscopy, complex voice evaluation, and rigid laryngeal videostroboscopy, and confirmed by laryngeal electromyography (EMG) in all cases. All of the patients with diagnoses of vocal fold paresis had mobile, but slightly sluggish, vocal folds. The degree of sluggishness in most patients was a subtle asymmetry or decrease in the degree of abduction, adduction, longitudinal tension, and/or agility of the vocal fold. In no patient with a diagnosis of paresis was the vocal fold immobile, and patients with immobile and/or paralyzed vocal folds were excluded from this study. Details of the laryngoscopic and EMG examinations and diagnostic criteria used have been described elsewhere.[14–16] Briefly, it is the standard practice of the physicians in this institution to refer patients to a neurologist (S.M.M.) for laryngeal EMG when laryngeal movement abnormalities or asymmetries are noted during laryngeal examination. Laryngeal EMG is performed using monopolar electrodes and either Teca Synergy (Oxford Instruments Medical, Oxfordshire, UK) with Toshiba Satellite Pro 3400 Series (Toshiba Corporation, Tokyo, Japan) laptop computer or Dantec Keypoint (Dantec Dynamics, Bristol, UK) with Toshiba Satellite 330 CDT (Toshiba Corporation, Tokyo, Japan) laptop computer. Criteria for the diagnosis of paresis include abnormalities in the recruitment pattern; the waveform morphology, amplitude, or duration; evidence of spontaneous activity; and/or abnormal insertional activity. Of the 1645 patients who presented for evaluation of dysphonia, 308 (18.7%) had a clinical diagnosis of vocal fold paresis and met the eligibility requirements for the study group.

In accordance with the guidelines for the evaluation of peripheral neuropathy, all patients who presented to this facility with an unexplained cranial neuropathy undergo testing for metabolic, infectious, inflammatory, autoimmune, and neoplastic abnormalities that are known to cause cranial neuropathy.[5,11] Included in this battery of testing is an evaluation of thyroid disease and autoimmune disorders that can affect thyroid function. The two most common cranial neuropathies seen in our practice are vocal fold paresis and sensorineural hearing loss. The charts were evaluated for the prevalence of thyroid disease, which was defined as an abnormality in the thyroid hormone levels (total T_3 [triiodothyronine], free T_4 [free thyroxine], and TSH [thyroid stimulating hormone]), thyroid antibody titers (antithyroglobulin antibody, thyroid peroxidase antibody, anti-TSH receptor antibody, thyroid-stimulating antibody, and/or thyroid-binding inhibitory antibody), thyroid ultrasonography, and/or computed tomography or magnetic resonance imaging of the neck. The exclusion criteria for the study group included a history of previously diagnosed thyroid disease or a prior history of thyroid, neck, mediastinal, or chest surgery. Patients in whom laryngeal EMG did not confirm vocal fold paresis, those who did not complete laryngeal or thyroid evaluation, and those patients who were lost to follow-up were also excluded. The prevalence of thyroid disease in this study group was then compared with the prevalence of thyroid disease in patients with sensorineural hearing loss who presented to the same clinical practice during the same time period for evaluation of hearing loss. In accordance with the guidelines for the evaluation of unexplained sensorineural hearing loss, a detailed thyroid evaluation is a part of the diagnostic evaluation of unexplained neurotologic symptoms in this practice, and patients who presented for such an evaluation were used as the comparison group.[12,13] Exclusion criteria for the control group included a concurrent history of neurotologic symptoms and vocal fold paresis; a concurrent history of neurotologic symptoms and dysphonia; any motion abnormality noted during laryngoscopy; failure to perform a laryngoscopic examination at the time of the initial evaluation of the neurotologic complaints; previously diagnosed thyroid disease; or a prior history of thyroid, neck, mediastinal, or chest surgery. Of the 2128 patients who presented for

TABLE 2.
Distribution of Multiple Nerve Involvement in Paretic Laryngeal Nerves

Nerve Involvement	Ipsilateral Unilateral Recurrent Laryngeal Nerve	Contralateral Unilateral Recurrent Laryngeal Nerve	Bilateral Recurrent Laryngeal Nerve
Unilateral superior laryngeal nerve	30	0	0
Bilateral superior laryngeal nerve	11 (unilateral recurrent laryngeal nerve involvement)		9

TABLE 3.
Thyroid Diagnoses in Patients With Vocal Fold Paresis Versus Those Without Vocal Fold Paresis

	Vocal Fold Paresis Group (n = 308)	Control Group (n = 333)	Level of Significance
Thyroid Diagnosis	n (%)	n (%)	*P*
Benign growth	92 (29.8)	18 (5.4)	<0.00001
Thyroiditis	24 (7.8)	10 (2.4)	0.007
Hyperthyroidism	14 (4.5)	16 (4.8)	0.877
Hypothyroidism	11 (3.6)	11 (3.3)	0.852
Thyroid malignancy	5 (1.6)	0 (0)	0.025
Total	**146 (47.4)**	**55 (16.5)**	**<0.001**

Pearson chi-square = 92.896; degrees of freedom = 5; $P < 0.0001$.

evaluation of hearing loss during this time period, 333 (15.6%) met the eligibility criteria for the comparison group.

RESULTS

In the vocal fold paresis group, there were 88 males and 220 females (male:female ratio of 1:2.5). The mean age was 50.9 ± 18.04 years (range: 15–90 years). The distribution of laryngeal nerve involvement is presented in Tables 1 and 2. Of the 308 patients with vocal fold paresis, 222 (72.1%) patients had unilateral vocal fold paresis, whereas 86 (27.9%) had bilateral vocal fold paresis. Of the 222 patients with unilateral vocal fold paresis, 189 patients had only superior laryngeal nerve involvement, and three patients had recurrent laryngeal nerve involvement only. The remaining 30 patients had both superior and recurrent laryngeal nerve involvement. Of the 86 patients with bilateral vocal fold paresis, 65 patients had bilateral superior laryngeal nerve involvement, one patient had bilateral recurrent laryngeal nerve involvement, and 20 patients had mixed nerve involvement.

The thyroid diagnoses are presented in Table 3. Thyroid disease was found in 146 of 308 (47.4%) patients with vocal fold paresis. Thyroid diagnoses included benign growths (92 of 308 or 29.8%), thyroiditis (24 of 308 or 7.8%), hyperthyroidism (14 of 308 or 4.5%), hypothyroidism (11 of 308 or 3.6%), and thyroid carcinoma (5 of 308 or 1.6%). Among the patients with thyroiditis, 17 had Hashimoto's thyroiditis and seven had autoimmune thyroiditis.

Benign growths on the thyroid were defined as nodules, cysts, thyromegaly or goiter, or adenomas. Hypothyroidism was defined as an elevated TSH or a low free T_4. Hyperthyroidism was defined as a low TSH with a normal or elevated free T_4. Hashimoto's thyroiditis was defined as an elevated thyroid peroxidase antibody level. Autoimmune thyroiditis was defined as an elevated antithyroglobulin antibody. All cases of thyroid malignancy and benign thyroid neoplasms had been diagnosed by fine needle aspiration biopsy and confirmed by surgical excision of the thyroid gland when clinically indicated.

In patients with thyroid masses (n = 97), the relationship between the side of paresis and the side of the thyroid mass was evaluated (Table 4). Twenty-six (26.8%) patients had unilateral paresis ipsilateral to the thyroid mass, whereas 16 (16.5%) patients had unilateral paresis contralateral to the thyroid mass. Thirteen (13.4%) patients showed bilateral vocal fold paresis with bilateral thyroid mass. Nineteen (19.6%) patients had unilateral vocal fold paresis with bilateral mass, and nine (9.3%) patients had bilateral vocal fold paresis with unilateral thyroid mass. Of the 92 benign masses detected, five were thyromegaly and 22 were in patients with bilateral paresis. Among the patients with unilateral vocal fold paresis, the location of the thyroid lesion relative to the side of the vocal fold paresis was not statistically significant (ipsilateral = 42.6%, contralateral = 26.2%, and bilateral = 31.2; $P = 0.264$, chi-square test).

In the comparison group, there were 333 patients, 171 males and 162 females, (male:female ratio of 1.05:1) with a mean age of 57.9 ± 16.88 years (range: 17–97 years). Fifty-five (16.5%) patients in this group were diagnosed with thyroid disease. Thyroid diagnoses included benign growths (18 of 333 patients or 5.4%), thyroiditis (10 of 333 patients or 2.4%), hyperthyroidism (16 of 333 patients or 4.8%), and hypothyroidism (11 of 333 patients or 3.3%). There were no patients diagnosed with thyroid

TABLE 4.
Side of Thyroid Mass (Benign and Malignant) Relative to Side of Vocal Fold Paresis

Side of Paresis	Mass Same Side as Paresis	Mass Opposite Side of Paresis	Bilateral Mass	Total
Unilateral	26	16	19	61
Bilateral	N/A	N/A	13	22 (9 are unilateral masses)

Abbreviation: N/A, not applicable.
Chi-square goodness of fit test, $P = 0.274$.

TABLE 5.
Age and Gender Characteristics of the Study and Control Groups

Characteristics	Vocal Fold Paresis (n = 308)	Control (n = 333)
Gender ($P < 0.001$, chi-square)		
Male, n (%)	88 (28.6%)	171 (51.4%)
Female, n (%)	220 (71.4%)	162 (48.6%)
Age ($P = 0.119$, *t* test)	50.87 ± 18.04	57.91 ± 16.88

malignancy in this group. Of the patients with thyroiditis, eight had Hashimoto's thyroiditis and two had autoimmune thyroiditis. There were statistically significantly more patients with vocal fold paresis and thyroid disease (146 of 308 or 47.4%) than there were patients in the comparison group with thyroid disease (55 of 333 or 16.5%) (chi-square = 70.914; $P < 0.001$). The 95% confidence interval for the difference in percentage is 23.86–37.52%. The odds ratio is 4.55 (95% confidence interval of 3.16–6.57).

There was no statistically significant difference between the average ages of the subjects in each group ($P = 0.119$). However, there were significantly more females (71.4%) in the vocal fold paresis group than those in the comparison group (48.6%; $P < 0.001$—Table 5).

DISCUSSION

This study reveals a prevalence of thyroid disease in patients with symptomatic vocal fold paresis of 47.4% (146 of 308), which is significantly higher than the prevalence of thyroid disease in the comparison group with sensorineural hearing loss (16.5%; 55 of 333). This suggests a strong association between the presence of symptomatic vocal fold paresis and thyroid disease. Of the patients with thyroid disease and vocal fold paresis, the presence of a benign mass in the thyroid was the most common cause of the thyroid disease, accounting for 63% of all thyroid pathologies in this group of patients. Furthermore, the presence of a benign mass in the thyroid was significantly more common in patients with symptomatic vocal fold paresis (29.8%) than it was in the comparison group (5.4%), as were both thyroiditis (7.8% vs. 2.4%, respectively) and thyroid malignancy (1.6% vs. 0%, respectively). Hyperthyroidism (4.5% and 4.8%, respectively) and hypothyroidism (3.6% and 3.3%, respectively) had similar levels of prevalence in the vocal fold paresis and the comparison groups.

The mechanism of nonmalignant thyroid masses causing vocal fold paresis is not completely understood. In other studies, suggested mechanisms have included nerve compression, neural stretch, perineural vascular insufficiency, and perineural inflammation.[17–19] However, most of the benign lesions seen in the patients in the present study did not cause gross enlargement of the involved thyroid lobe, making neural stretch, vascular insufficiency, or nerve compression unlikely causes of the pareses seen in this study. Furthermore, there was no statistically significant difference in the location of the mass and the side of the paresis, suggesting that compression may not be the causative factor in the development of paresis and that other, yet undefined, local factors may play more of a role. It is possible that autoimmune and/or local inflammatory factors are more likely to be causally related to the pareses seen, especially given the higher incidence of thyroiditis in the study group. Local inflammation in reaction to a benign thyroid mass and perhaps even a local hormonal effect may also explain the higher incidence of vocal fold paresis in the group of patients with benign thyroid lesions. The incidence of systemic hypothyroidism and hyperthyroidism were the same in the comparison and the study groups, making the role of systemic thyroid hormone levels in the development of vocal fold paresis unclear. In other peripheral neuropathies, hyperthyroidism and hypothyroidism are clearly the contributing factors in their development, as it has been observed that these neuropathies reverse when the metabolic abnormality is reversed.[7,8,10,20] Perhaps, a local metabolic derangement in thyroid hormone levels related to relative decreases or increases in hormone production from the benign lesions may play a role in the increased incidence of vocal fold paresis in these patients. Or, perhaps, the development of vocal fold paresis in patients with thyroid masses is related to a lesser extent to local hormonal production and to a greater extent to local inflammatory or mass effect-associated factors.

Another observation of this study was that there were significantly more women with vocal fold paresis than there were in the comparison group. It is known that thyroid disease is more prevalent in females than in males, and the greater prevalence of thyroid disease in the study group may, at first glance, be secondary to these known gender differences. However, when one looks more closely at the prevalence of thyroid disease only in the female subjects with paresis versus those without paresis, it is seen that the prevalence of thyroid disease is still greater among those

TABLE 6.
Prevalence of Thyroid Disease in Female Subjects

Disease State	Vocal Fold Paresis Group	Sensorineural Hearing Loss Group (Control)	Total
Thyroid disease, n (%)	120 (54.5)	30 (18.5)	150
No thyroid disease, n (%)	100 (45.5)	132 (81.5)	232
Total	220	162	382

Chi-square = 50.778; Yates value = 49.279.
Degrees of freedom = 1; $P < 0.0001$.

with vocal fold paresis, eliminating gender as a cause of the difference in vocal fold paresis seen in the present study (Table 6). Furthermore, the higher prevalence of thyroid disease among the female patients with vocal fold paresis versus those without paresis may further support the notion that thyroid disease may be an important cause of symptomatic vocal fold paresis.

The limitations of our study include the inability to observe directly the anatomical and histological associations between the thyroid gland and the laryngeal nerves. Such an observation might enable a better understanding of the relationship between thyroid disease and the observed laryngeal nerve pathology. Because this study was primarily interested in understanding the relationship between thyroid disease and peripheral neuropathy, as it relates to the laryngeal nerves versus other cranial nerves, a cohort of patients with sensorineural hearing loss was chosen as the comparison group. It would also be interesting to evaluate the prevalence of thyroid disease in patients with dysphonia and no evidence of vocal fold paresis on laryngeal examination or laryngeal EMG. Such a cohort of patients was not available for the present study, but may represent an interesting comparison group for a prospective study.

CONCLUSIONS

Our results suggest that thyroid abnormalities are more prevalent in patients with symptomatic vocal fold paresis than they are in a cohort of patients with sensorineural hearing loss, suggesting a stronger association between symptomatic vocal fold paresis and thyroid disease than that between sensorineural hearing loss and thyroid disease. This association is clinically relevant to the treatment of patients who present with voice complaints and findings of vocal fold hypomobility. In these cases, a detailed thyroid evaluation is warranted as a part of the diagnostic evaluation. Further investigation through prospective human and experimental animal studies may provide more information about the nature of the association between dysphonia, vocal fold paresis, and thyroid disease, and whether treatment of the thyroid disease has any significant effect on the voice and/or the degree of vocal fold paresis.

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