LARYNGOLOGY

Vocal fold paresis accompanying vocal fold polyps

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Abstract The aim of this study was to evaluate patients with vocal fold polyps using laryngeal electromyography (LEMG) for the presence of vocal fold paresis and to compare transnasal fiberoptic and rigid stroboscopic findings between polyp patients with normal LEMG and with vocal fold paresis. Thirty-five patients with a vocal fold polyp underwent transnasal fiberoptic laryngoscopy, rigid laryngostroboscopy, and LEMG. The findings were compared between the LEMG-confirmed vocal fold paresis patients and the normal LEMG patients. LEMG resulted in a diagnosis of unilateral or bilateral vocal fold paresis in 17 of 35 patients (48.6 %). More men than women with vocal fold paresis group had higher presence of axial rotation and hypomobility of vocal folds, higher asymmetry of vertical

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height of vocal folds, and less presence of longitudinal stretch of vocal folds (p < 0.05). Medial–lateral compression of the false vocal folds and anterior–posterior approximation of the larynx did not show any difference between the groups. No significant difference was found in vibratory wave characteristics between the groups through rigid laryngostroboscopy. Vocal fold paresis was present in almost half of the patients with vocal fold polyps. Paresis can only be accurately diagnosed with LEMG. Transnasal fiberoptic laryngoscopic examination is helpful to recognize vocal fold paresis in vocal fold polyp patients, while stroboscopic examination is not useful to identify it in vocal fold polyp patients.

Keywords Vocal fold paresis · Vocal fold polyp · Laryngeal electromyography · Laryngostroboscopy · Fiberoptic laryngoscopy

Introduction

Vocal fold polyps are benign, round, sessile or pedinculated lesions located on the free borders of the vocal folds. Several factors seem to influence the development of vocal fold polyps, such as vocal misuse or abuse, allergy, smoking, chronic infections of the upper airways, and laryngopharyngeal reflux [1–6].

Vocal fold paresis is a clinical condition involving partial paralysis of a vocal cord that requires greater clarification. The clinical presentation of vocal fold paresis may include subtle axial rotation of the posterior larynx, incomplete glottal closure, vibratory asymmetry, or prominent vocal fold hypomobility and bowing, depending on the laryngeal nerve involved and the degree of the neuromuscular deficit [7–9]. The wider use of laryngeal electromyography (LEMG) in clinical practice allows clinicians to more accurately identify vocal fold paresis.

There are very few reports which present paresis of one or more of the larvngeal nerves in the literature [9-14]. A detailed review of these retrospective case studies revealed that benign vocal fold lesions such as nodule, polyp, cyst, and granuloma were accompanied by paresis in some of the patients [10, 12, 13]. Comorbidity of vocal fold paresis with benign vocal fold lesions, such as a vocal fold polyp, can be especially important to achieve satisfactory treatment results. Because the presence of a vocal fold polyp is typically the prominent finding that requires treatment, an accompanying vocal fold paresis can easily be overlooked if it is not suspected as a result of clinical findings. This may result in an unsatisfactory voice outcome. The literature does not contain any study that evaluates the presence of vocal fold paresis accompanying vocal fold polyp or that tries to identify examination findings which could lead to a suspicion of comorbid vocal fold paresis.

The purposes of this study were to prospectively evaluate patients presenting with a vocal fold polyp using LEMG for a diagnosis of vocal fold paresis and also to identify transnasal fiberoptic and laryngostroboscopic findings that may help to differentiate vocal fold polyp patients with a normal LEMG from those with vocal fold paresis.

Methods

Institutional Review Board approval was obtained from our institution before the study commenced. Patients who had been diagnosed with a vocal fold polyp between October 2011 and November 2013 were asked to participate in this prospective study. The patients were informed about the study and their signed written consents were obtained. Exclusion criteria included age below 18 years, history of radiation therapy for neck, presence of complete immobility of the vocal fold (suggestive of paralysis or fixation), leukoplakia, and malignancy.

During the study period 35 patients diagnosed with a vocal fold polyp agreed to participate in this study. This included 13 (37.1 %) female and 22 (62.9 %) male. The mean age was 38.9 years (range 22–59 years). The mean time interval from the onset of symptoms to presentation at the clinic was 2 years (range 1 month–10 years). Three (8.6 %) patients had a giant polyp, 7 (20 %) patients had a contact lesion on the contralateral side, 2 (5.6 %) patients had contralateral side, 2 (5.6 %) patients had contralateral vocal fold edema, and 1 (2.8 %) patient had sulcus vocalis on the same side as the polyp.

Patients were asked to complete the Glottal Function Index (GFI) and Reflux Symptom Index (RSI) [15, 16]. The GFI is a 4-item symptom index used to assess the four main symptoms of glottal dysfunction: effortful speaking, vocal pain after talking, vocal fatigue, and pitch breaks. The patient is asked to rate his/her voice symptoms during the past month on a scale from 0 (no problem) to 5 (severe problem). A score of 20 represents maximum symptom severity, and a total score greater than four is considered to be abnormal [15]. The RSI is a 9-item self-administered outcomes questionnaire for evaluating symptoms of laryngopharyngeal reflux. Each item is scored between 0 (no problem) and 5 (severe problem), with a maximum total score of 45. An RSI of greater than 13 is considered to indicate laryngopharyngeal reflux [16].

All patients underwent the following evaluations: (1) a complete medical history and review of systems; (2) transnasal fiberoptic laryngoscopy; (3) rigid laryngostroboscopy; and (4) LEMG.

The larynx was first visualized through a transnasal fiberoptic endoscope with illumination provided by the halogen light, then with a 70° transoral rigid endoscope (Karl Storz, Tuttlingen, Germany) attached to a CCD camera and Storz Pulsar Stroboscope (Karl Storz, Tuttlingen, Germany). All laryngoscopic examinations were made by the same otolaryngologist (S.A.) with the same protocol and later reviewed and rated by her on standard protocol sheets. The examination recordings were played back using the same equipment used for the original recording and viewed on the same monitor.

The transnasal fiberoptic examination included speech and nonspeech tasks for assessing glottal closure, supraglottic hyperfunction, and degree of abduction and adduction. Patients were asked to sniff repetitively, to alternate between a sniff and */i/* vowel repeatedly, to alternate between */i/* and */hi/* vowels repeatedly, and also to repeat the phrase */pa/-/ta/-/ka/*. The glissando maneuver, from low to high tone and then from high to low tone, was performed to evaluate longitudinal stretching of the vocal cords and axial rotation. Supraglottic hyperfunction was assessed during counting from '1' to '10' and also during running speech [11, 17].

During transoral rigid laryngoscopy, patients were instructed to phonate */i/* vowel at low, modal, and high frequencies with a stroboscopic light (Pulsar Stroboscope, Karl Storz, Tuttlingen, Germany). Glottal closure configuration and vibratory wave characteristics (amplitude, symmetry, periodicity, and mucosal wave) were evaluated. The stroboscopic signs and rating categories are shown in Table 1. The ratings were based on visualization of the patients' modal pitch, at comfortable loudness phonation according to the criteria found in Hirano's book on videostroboscopy [18]. Amplitude of the vocal fold vibration,

Table 1	Stroboscopic	signs	and	category	ratings

Glottal closure configuration	Amplitude (each cord separately)
Hourglass	Normal
Complete	Slightly decreased
Incomplete	Moderately decreased
Irregular	Severely decreased
Posterior chink	No visible movement
Vibratory behavior (each cord separately)	Mucosal wave (each cord separately)
Always fully present	Normal
Partial absence sometimes	Slightly decreased
Partial absence always	Moderately decreased
Complete absence sometimes	Severely decreased
Complete absence always	Absent
	Increased
Phase symmetry	Periodicity
Regular	Regular
Irregular	Irregular

mucosal wave and vibratory behavior were rated separately for each vocal fold.

Laryngeal electromyography was performed within 2-3 weeks after the laryngoscopic examination. The needle electrodes were inserted by the otolaryngologist (S.A.), and the operation of the electromyography machine and interpretation of the signal were done by the neurologist (R.A.I.). The neurologist was blinded to the findings of the laryngoscopic examination in all cases. Electromyography was performed with a Nihon Kohden-Neuropack (Tokyo, Japan) MEB-5504K electromyography machine with 45 mm \times 26 G concentric needle electrodes (Myoline[®]), Spes Medica, Genoa, Italy). Needle placement and position were confirmed with standard percutaneous laryngeal electromyographic techniques [19-21]. The left cricothyroid, right cricothyroid, left thyroarytenoid, and right thyroarytenoid muscles were evaluated routinely. Placement into the cricothyroid muscle was confirmed by the presence of increased recruitment during a high-pitched /i/ and less recruitment during a low-pitched /i/. Placement into the thyroarytenoid muscle was confirmed by the presence of recruitment during speech or sustained phonation of the vowel /i/, combined with the presence of silence during active inspiration and sniffing. Phonation was tested at moderate intensity (normal speaking level).

Three to five different sites were evaluated within each muscle. The placement of needle near individual motor units was confirmed with the characteristic crisp sound and fine biphasic motor unit potentials. The low-frequency filter was set at 20 Hz, and the high-frequency filter was set at 10 kHz. For insertional and spontaneous activity a sweep speed of 10 ms per division and gain of 50 μ V per division

were used. Motor unit action potential (MUAP) morphology, amplitude, duration, and recruitment were assessed at sweep speeds of 10–20 ms and at gains of 200–500 μ V.

The analysis of EMG data included assessment of insertional activity, spontaneous activity, amplitude of MUAP, MUAP duration, MUAP morphology during volunteer muscle activity, and recruitment pattern. Presence of full recruitment and normal MUAP, absence of spontaneous activity and of reinnervation MUAPs or polyphasic MUAPs was defined as "Normal" LEMG (Fig. 1). The diagnosis of neuropathy (i.e., paresis) was confirmed when there was one or more of the following "abnormal" LEMG findings in the muscle: presence of decreased recruitment, increased MUAP duration and amplitude, polyphasic MUAPs, spontaneous activities such as fibrillation potentials, or positive sharp waves [13, 21–23] (Figs. 2, 3).

The transnasal fiberoptic evaluation and rigid stroboscopic examination findings were compared between the LEMG-confirmed vocal fold paresis patients (Polyp with Paresis Group) and the normal LEMG patients (Polyp with Normal LEMG Group).

Statistical analysis

The statistical data were conducted using Number Cruncher Statistical System (NCSS) 2007 (NCSS LLC, Kaysville, UT, USA) and Power Analysis and Sample Size (PASS) 2008 for Windows (NCSS LLC, Kaysville, UT, USA). Metric data are presented as mean \pm SD. Descriptive statistics were calculated for numeric variables. The comparison of quantitative data was done using the Mann–Whitney *U* test. In qualitative analyses, Fisher's exact test, Fisher–Freeman–Halton exact test, and Yates' continuity correction test were used. A *p* value <0.05 was considered to be statistically significant.

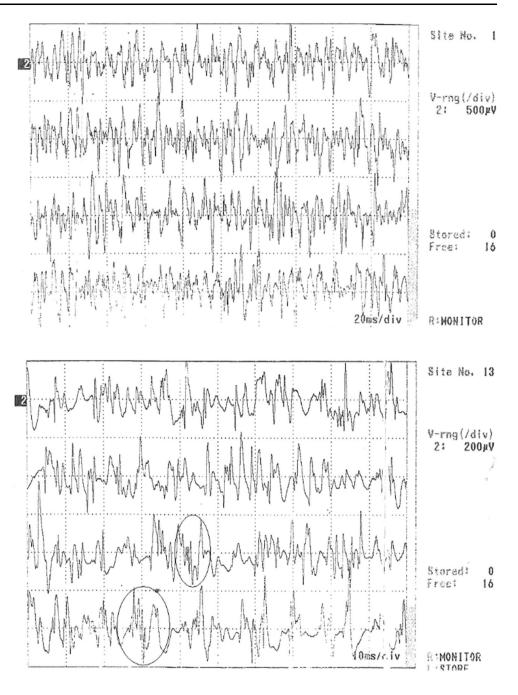
Results

Laryngeal electromyography resulted in a diagnosis of unilateral or bilateral vocal fold paresis in 17(48.6 %) of the 35 patients with a vocal fold polyp. Unilateral neuropathic findings were found in 11(31.5 %) patients and bilateral findings in 6 (17.1 %) patients. Unilateral neuropathies included both "ipsilateral" (i.e., on the side of the vocal fold with a polyp) and "contralateral" (i.e., on the side of the vocal fold without a polyp) neuropathies.

Of the 11 patients with unilateral neuropathy, ipsilateral combined recurrent laryngeal nerve (RLN) neuropathy and superior laryngeal nerve (SLN) neuropathy were found in 5 (14.3 %) patients; ipsilateral SLN neuropathy in 3 (8.5 %) patients; contralateral RLN neuropathy in 2 (5.6 %) patients; and ipsilateral RLN neuropathy in 1 (2.8 %)

Fig. 1 Normal motor unit action potential waveform and full interference pattern at maximal contraction (patient 3, recorded from left cricothyroid muscle) (sweep speed 20 ms/ div, sensitivity 500 μV/div)

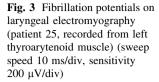
Fig. 2 Polyphasic motor unit action potentials with long duration and high amplitude (*marked with circle*) and decreased recruitment pattern on laryngeal electromyography (patient 7, recorded from right thyroarytenoid muscle) (sweep speed 10 ms/div, sensitivity 200 μV/div)



patient. Neuropathy was found in three nerves in 5 (14.3 %) patients and in four nerves in 1 (2.8 %) patient. Table 2 summarizes the LEMG findings.

Comparison of the patients with vocal fold paresis and with normal LEMG revealed that more men than women with vocal fold polyps also had vocal fold paresis (p = 0.049; Table 3). The mean RSI of the patients with normal LEMG was significantly higher than that of the patients with vocal fold paresis (p = 0.024; Table 3). Other than these there were no differences in age, polyp side, duration of the symptoms, and GFI between the groups (p > 0.05).

The distributions of transnasal fiberoptic laryngoscopic examination findings of the groups are displayed in Table 4. All the patients in the Polyp with Paresis Group had medial-lateral compression of the false vocal folds while 22.2 % (4/18) of the patients in the Polyp with Normal LEMG Group revealed no medial-lateral compression of the false vocal folds, but the difference between the distribution of medial-lateral compression of the groups was not found to be statistically significant (p = 0.098). The distribution of anterior-posterior approximation of the larynx also did not show any difference between the groups (p = 0.621). The presence of



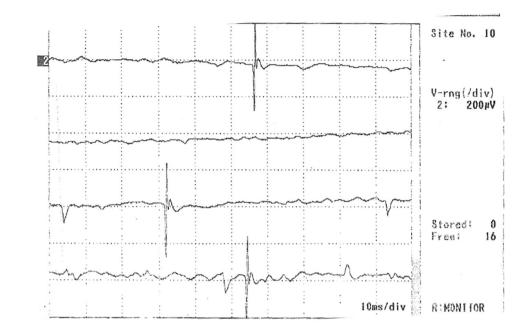


Table 2 Laryngeal electromyography findings

Electromyographic findings	n	%
Normal	18	51.4
Paresis		
Unilateral findings	11	31.5
Ipsilateral combined RLN & SLN	5	14.3
Ipsilateral SLN	3	8.5
Ipsilateral RLN	1	2.8
Contralateral RLN	2	5.6
Bilateral findings	6	17.1
3 nerves involvement	5	14.3
4 nerves involvement	1	2.8
Total	35	100

"Ipsilateral" refers to the side of the vocal cord with a polyp

"Contralateral" refers to the side of the vocal cord without a polyp *RLN* recurrent laryngeal nerve, *SLN* superior laryngeal nerve

axial rotation, asymmetry of vertical height of vocal folds, hypomobility of vocal folds, and presence of longitudinal stretch of vocal folds were the findings that showed statistically significant differences between the groups (Table 4). The Polyp with Paresis Group had a higher presence of axial rotation (ipsilateral or contralateral), higher asymmetry of vertical height of vocal folds, more patients with hypomobility of vocal fold (ipsilateral or contralateral), and fewer patients with presence of longitudinal stretch of the vocal folds (p < 0.05).

Laryngeal electromyography revealed one patient who presented ipsilateral vocal fold fatigue with paresis on the same side. LEMG also revealed a patient with contralateral recurrent laryngeal nerve neuropathy who showed bowing of the contralateral vocal fold. That patient also revealed hypomobility of the contralateral vocal fold and axial rotation to the contralateral side, as well as medial–lateral compression and anterior–posterior approximation in the fiberoptic examination. The other patient with contralateral vocal fold paresis had only medial–lateral compression of the false vocal folds and anterior–posterior approximation of the larynx.

In 12 (70.6 %, 12/17) patients of the Polyp with Paresis Group there was at least one of the fiberoptic examination findings other than medial–lateral compression of the false vocal folds and anterior–posterior approximation of the larynx. Five (29.4 %, 5/17) of the Polyp with Paresis Group exhibited only supraglottic hyperfunction and did not have any other fiberoptic examination findings giving rise to a suspicion of paresis.

Transoral rigid stroboscopic evaluation showed no significant differences between the groups in distribution of glottal closure configuration or of any vibratory wave characteristics including amplitude, mucosal wave, vibratory behavior, phase symmetry, and periodicity (p > 0.05) (Table 5).

Discussion

Vocal fold paresis is incomplete paralysis in which some gross vocal fold mobility is preserved. Many inexplicable patient symptoms and indefinite laryngoscopic examination findings have been associated with mild neurologic deficit

Table 3 Characteristics of normal LEMG and paresis		Normal LEMG	(n = 18)	Paresis $(n = 17)$	р	
groups		Mean \pm SD	Min-Max (median)	Mean \pm SD	Min-Max (median)	
	Age	36.67 ± 8.47	22.00-48.00 (38.00)	41.35 ± 9.96	26.00-59.00 (43.00)	0.180 ^a
	Duration	17.56 ± 18.83	2.00-66.00 (12.00)	31.53 ± 41.02	1.00-120.00 (9.00)	0.703 ^a
	GFI	13.00 ± 3.60	4.00-19.00 (13.00)	11.76 ± 4.22	3.00-20.00 (12.00)	0.336 ^a
	RSI	20.11 ± 9.29	4.00-34.00 (20.00)	13.12 ± 8.13	2.00-30.00 (11.00)	0.024* ^{, a}
		n	%	п	%	р
	Sex					
GFI Glottal Function Index, RSI	Female	10	55.6	3	17.6	0.049* ^{, b}
Reflux Symptom Index	Male	8	44.4	14	82.4	
* $p < 0.05$ ^a Mann–Whitney U test	Polyp side					
	Right	7	38.9	11	64.7	0.234 ^b
^b Yates's continuity correction test	Left	11	61.1	6	35.3	

of the vocal folds [7]. Subtle laryngeal asymmetries observed with laryngoscopic examination may be defined as paresis, but LEMG is used to confirm the diagnosis [7, 11, 14].

In a study by Heman-Ackah and Batory, 33 patients had benign vocal fold lesions, such as a polyp, cyst, edema, granuloma etc., in 61 patients with clinically defined vocal fold hypomobility. But the confirmation of vocal fold paresis with LEMG was not done in this study [12]. The coexistence of vocal fold mucosal lesions was found in 11 of 19 patients with clinical- and LEMG-confirmed vocal fold paresis in the case series of Heman-Ackah and Barr [13]. Dursun et al. [10] reported that vocal fold mucosal lesions such as polyp, cyst, nodule, etc. were observed in 27.7 % of 126 patients with LEMG-confirmed superior laryngeal nerve paresis or paralysis.

In our prospective study 48.6 % (17) of 35 patients with a vocal fold polyp revealed vocal fold paresis with LEMG and 31.5 % of patients (11 patients, including 6 bilateral paresis and 5 unilateral SLN and RLN paresis) showed paresis of more than one laryngeal nerve. There is no other study available in the literature which investigated the presence of vocal fold paresis in vocal fold polyps.

Actually the incidence of vocal fold paresis has not been well demonstrated, but the few reports from tertiary laryngology practices suggest that vocal fold paresis is not common. Merati et al. [8] reported 29 patients of vocal fold paresis in a year. Heman-Ackah and Barr [13] identified 19 patients over a 13-month period. Simpson et al. [14] presented 13 patients of paresis over 4 years and Koufman et al. [11] reported 50 patients over 4 years. It should be emphasized that all these data are from retrospective chart reviews. Taking into account these studies, the considerably high percentage of incidence of vocal fold paresis in patients with a vocal fold polyp in our study is noteworthy. Not all the patients with a vocal fold polyp agreed to participate in the LEMG study because of apprehension of the procedure, so we may have had patient selection bias on the part of the potential participants. It is possible that more patients with vocal fold polyp and comorbid vocal fold paresis agreed to have a LEMG study resulting in a higher incidence of vocal cord paresis in our study. Our results suggest that further study of vocal fold paresis and its association with vocal fold polyps is essential.

Patients with vocal fold paresis may generate asymmetrical excessive muscular forces to achieve glottal closure which may lead to trauma to the vocal folds. This may be a contributing factor for polyp development in some patients with vocal fold paresis, or for recurrence of the lesion after excision. It has been stated that underlying "hypofunctional" glottal disorder sometimes demonstrates as "hyperfunctional" compensation [11, 17]. Thus, LEMG is an essential procedure to test neuromuscular integrity in these patients. Koufman and Belafsky [24] proposed that midfold localized Reinke's edema or pseudocysts are a consequence of underlying paresis. Increased sheering trauma from effortful glottic closure is their hypothesized mechanism responsible for these lesions. It should be kept in mind that patients with voice disorders often have more than one underlying disorder, and each problem should be identified and corrected for a better voice outcome [11, 25].

Laryngopharyngeal reflux has been reported to be associated with vocal fold polyps as a causative factor [3, 6]. In our study RSI was found to be lower in the patients with vocal fold paresis than the patients with normal LEMG. It was reported that there is a slight predominance of vocal fold polyps in women [5]. Contrarily, we found that more men than women with vocal fold polyps accompanied vocal fold paresis in our study. These may suggest that clinicians should be more attentive to the possibility of coexisting vocal fold paresis with vocal fold

 Table 4 Distribution of the fiberoptic laryngoscopic examination findings in the groups

Table 5	Distribution	of the	rigid	stroboscopic	examination	findings
in the gr	oups					

	Normal LEMG $(n = 18)$		Paresis $(n = 17)$		р	
	п	%	n	%	-	
Medio-lateral c	ompress	ion of false v	ocal cord			
None	4	22.2	0	0.0	0.098 ^a	
Bilateral	12	66.7	16	94.1		
Contralateral	2	11.1	1	5.9		
A-P approxima	tion					
None	11	61.1	8	47.1	0.621 ^b	
Present	7	38.9	9	52.9		
Axial rotation						
None	17	94.4	9	53	0.012* ^{, a}	
Present-I	1	5.6	4	23.5		
Present-C	0	0	4	23.5		
Fatigue						
None	18	100	16	94.1	0.486 ^c	
Present-I	0	0	1	5.9		
Bowing						
None	18	100	16	94.1	0.486 ^c	
Present-C	0	0	1	5.9		
Vertical height	of vocal	cord				
Symmetric	18	100	13	76.5	0.045* ^{, c}	
Asymmetric	0	0	4	23.5		
Hypomobility						
None	18	100	12	70.6	0.019* ^{, a}	
Ipsilateral	0	0	4	23.5		
Contralateral	0	0	1	5.9		
Longitudinal str	retch					
None	0	0	3	17.6	0.018* ^{, a}	
Present	17	94.4	9	53		
Doubtful	1	5.6	5	29.4		

I ipsilateral, which refers to the side of the vocal cord with a polyp, C contralateral, which refers to the side of the vocal cord without a polyp

* p < 0.05

- ^a Fisher-Freeman-Halton exact test
- ^b Yates' continuity correction test

^c Fisher's exact test

polyps in men and in patients who have few symptoms for laryngopharyngeal reflux.

The neurolaryngeal examination of the larynx is performed best using transnasal fiberoptic laryngoscopy under continous light as subtleties can be observed easily. Presence of axial rotation, asymmetry in vocal cord longitudinal stretch, hypomobility, fatigue or bowing of

	Normal LEMG $(n = 18)$		Paresis $(n = 17)$		p ^a
	n	%	n	%	
Glottal closure configuration					
Hourglass	8	44.4	6	35.3	0.97
Complete	1	5.6	1	5.9	
Incomplete	7	38.8	7	41.2	
Irregular	1	5.6	2	11.7	
Posterior chink	1	5.6	1	5.9	
Amplitude-I					
Normal	1	5.6	1	5.9	0.95
Slightly decreased	6	33.3	4	23.6	
Moderately decreased	4	22.2	3	17.6	
Severely decreased	5	27.8	6	35.3	
None	2	11.1	3	17.6	
Amplitude-C					
Normal	12	66.6	6	35.3	0.52
Slightly decreased	2	11.1	4	23.5	
Moderately decreased	2	11.1	3	17.6	
Severely decreased	1	5.6	2	11.8	
None	0	0	1	5.9	
Increased	1	5.6	1	5.9	
Mucosal wave-I					
Normal	1	5.6	1	5.9	0.25
Slightly decreased	1	5.6	0	0	
Moderately decreased	2	11.1	5	29.4	
Severely decreased	7	38.8	5	29.4	
Invisible	6	33.3	2	11.8	
Increased	1	5.6	4	23.5	
Mucosal wave-C					
Normal	12	66.7	8	47	0.68
Slightly decreased	3	16.7	3	17.6	
Moderately decreased	1	5.6	2	11.8	
Severely decreased	0	0	1	5.9	
Invisible	0	0	2	11.8	
Increased	1	5.6	1	5.9	
Nonassessed	1	5.6	0	0	
Vibratory behavior-I					
Always fully present	3	16.7	0	0	0.16
Partial absence sometimes	1	5.6	0	0	
Partial absence always	7	38.8	12	70.5	
Complete absence sometimes	2	11.1	2	11.8	
Complete absence always	5	27.8	2	11.8	
Nonassessed	0	0	1	5.9	
Vibratory behavior-C					

Table 5 continued

	Normal LEMG $(n = 18)$		Paresis $(n = 17)$		p ^a	
	n	%	n	%		
Always fully present	13	72.2	9	52.9	0.48	
Partial absence sometimes	1	5.6	4	23.5		
Partial absence always	2	11.1	3	17.7		
Complete absence sometimes	2	11.1	1	5.9		
Phase symmetry						
Regular	7	38.8	4	23.5	0.59	
Irregular	10	55.6	11	64.7		
Nonassessed	1	5.6	2	11.8		
Periodicity						
Regular	6	33.3	3	17.6	0.57	
Irregular	11	61.1	12	70.6		
Nonassessed	1	5.6	2	11.8		

I ipsilateral, which refers to the side of the vocal cord with a polyp, C contralateral, which refers to the side of the vocal cord without a polyp

^a Fisher-Freeman-Halton exact test

vocal fold, and asymmetry of vertical height of vocal folds are findings that support the clinical suspicion of vocal fold paresis [12, 17, 25]. Our study also confirms that these laryngoscopic findings are observed significantly more in vocal fold polyp patients with vocal fold paresis than in vocal fold polyp patients with normal LEMG (p < 0.05). More than 70 % of the vocal fold polyp patients with LEMG-confirmed paresis revealed at least one of these fiberoptic examination findings in our study.

Another laryngeal finding which may suggest clinical suspicion of vocal fold paresis is supraglottic hyperfunction, such as medial-lateral compression of false vocal folds or supraglottic anterior-posterior approximation. These supraglottic hyperfunction patterns may result from an attempt to compensate the underlying glottal insufficiency [11, 25, 26]. But the presence of a vocal fold polyp complicates the situation as benign vocal fold lesions usually exhibit some supraglottic hyperfunction [27]. In our study, patients with vocal fold paresis and with normal LEMG did not reveal any difference in medial-lateral compression of false vocal folds or in anterior-posterior approximation. It thus can be concluded that supraglottic hyperfunction may not help to differentiate the presence of vocal fold paresis in vocal fold polyp patients. In fact, approximately 30 % (5/17) of the patients with LEMGconfirmed paresis exhibited only supraglottic hyperfunction during their fiberoptic examination.

In the setting of no readily apparent differences in vocal fold mobility, it can be difficult to recognize vocal fold paresis. Asymmetry of vibration, and incomplete glottal closure during laryngostroboscopic examination were reported to be associated with vocal fold paresis in this situation [9, 14]. But the presence of vibratory asymmetry is a common finding in patients with a vocal fold polyp [4, 28]. In our study vocal fold polyp patients with paresis and with normal LEMG showed a similar distribution of stroboscopic findings including vibratory characteristics and glottal closure pattern (p > 0.05). Our study suggests that laryngostroboscopic examination does not help to identify paresis in vocal fold polyp patients.

One of the limitations of our study is small sample size. Further study including more patients would be worthwhile to support our results. Second, the LEMGs and laryngoscopic examinations in our study were performed by the same neurologist and the same otolaryngologist. As both interpretation of LEMG findings and evaluation of laryngoscopic examinations are subjective in nature, having additional reviewers would have strengthened our study. To overcome the subjectivity problem in LEMG, another alternative would have been using a quantitative LEMG analysis [29]. Finally, it should be kept in mind that vocal fold paresis is not a single clinical condition but is more like different conditions lying along a spectrum, depending on the laryngeal nerve or nerves involved and the degree of the neuromuscular deficit. As a result, laryngoscopic examination findings can be expected to occur in a spectrum as well. This was not taken into account in our study when comparing examination findings. The evaluation of different subgroups of paresis could be the subject of future studies.

Conclusion

When dealing with patients who have been diagnosed with a vocal fold polyp, the possibility of coexisting vocal fold paresis should be kept in mind. LEMG confirmed the diagnosis of unilateral or bilateral vocal fold paresis in almost half of the patients with a vocal fold polyp. Vocal fold paresis accompanied vocal fold polyp more in men and in patients with few symptoms of laryngopharyngeal reflux. More than 70 % of the vocal fold polyp patients with LEMG-confirmed paresis had at least one of the transnasal fiberoptic examination findings, i.e., the presence of axial rotation, asymmetry in vocal cord longitudinal stretch, hypomobility, fatigue or bowing of vocal fold, and asymmetry of vertical height of vocal folds. Supraglottic hyperfunction did not help to differentiate the presence of vocal fold paresis in vocal fold polyp patients. Stroboscopic examination was not useful to identify paresis in vocal fold polyp patients.

It should be emphasized that vocal fold paresis can only be accurately diagnosed with LEMG even when laryngoscopic examination suggests the presence of it. Further research is needed to clarify the relationship between vocal fold paresis and vocal fold polyps and to determine the clinical significance of LEMG in patients with vocal fold polyps.

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Conflict of interest None.

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