Clinical and Electromyographic Characteristics of Unilateral Vocal Fold Paralysis With Lower Cranial Nerve Injury

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Summary: Objectives. The aim was to investigate the clinical and electromyographic characteristics of patients with unilateral vocal fold paralysis (UVFP) combined with lower cranial nerve injury.
Study design. This is a case series with chart review.
Methods. Among 368 patients with idiopathic UVFP, 31 patients (8.4%) were eventually diagnosed with lower cranial nerve palsy after examinations of the head and neck, radiology, and electromyogram (EMG). The clinical and electromyographic characteristics of these patients were analyzed.
Results. Of the 31 patients, 27 patients exhibited obvious abnormal lower cranial nerve injury physical signs, and 4 patients showed atypical physical signs, identified by EMG. Ultimately, 41.9% (13/31) were diagnosed with idiopathic causes, 38.7% (12/31) with intracranial or skull-base lesions on radiology, 12.9% (4/31) with lower cranial neuritis, and 6.4% (2/31) with radiation-induced lower cranial nerve palsy. Among the cranial lesions, lesions of the jugular foramen region were the most common (50%, 6/12). All 26 patients who underwent EMG tests were confirmed to have vagus nerve impairments (11 complete and 15 incomplete) and accessory nerve impairments (16 complete and 10 incomplete), whereas only 13 patients (50%) exhibited hypoglossal nerve injuries (5 complete and 8 incomplete).
Conclusions. For patients with clinically "idiopathic" UVFP, physical examinations of the lower cranial nerves are essential screening procedures. For patients with abnormal or suspicious physical signs, radiology should be performed to detect possible cranial or cervical lesions. EMG tests were strongly recommended to identify suspicious lower cranial nerve injury and its severity, and may help to predict the prognosis.
Key Words: Vocal fold paralysis–Lower cranial nerve injury–Radiology–Laryngeal electromyogram–Nuchal electromyogram.

INTRODUCTION

The etiologies of unilateral vocal fold paralysis (UVFP) are very complicated and include neoplasms, iatrogenic factors, infections, trauma, and idiopathic causes. In addition to recurrent laryngeal nerve (vagus nerve) palsy, lesions located in the brain stem or skull base are often associated with impairments of the other lower cranial nerves (ie, cranial nerves IX, XI, and XII). The symptom of hoarseness caused by UVFP may be the easiest to perceive; however, the symptomatology resulting from impairments of the lower cranial nerves is often inconspicuous, and signs of lower cranial nerve palsy may easily be overlooked by otolaryngologists or misdiagnosed as "idiopathic" or "sole" UVFP.[1–5] Moreover, there are few published studies related to the detailed clinical and electromyographic features of UVFP combined with lower cranial nerve palsy. Thus, the objective of the present study was to investigate the clinical and electromyographic characteristics of the patients with UVFP combined with lower cranial nerve palsy.

MATERIALS AND METHODS

From April 2008 to October 2013, a consecutive series of 368 patients with initial diagnoses of idiopathic UVFP from the Department of Otorhinolaryngology—Head and Neck Surgery, Beijing Tongren Hospital, China, were included. Physical examinations of the head and neck including the lower cranial nerves and laryngostroboscopy were performed in all patients. The examined physical signs of the lower cranial nerves included the position and symmetry of the palate and uvula (the vagus nerve), shoulder shrugging and head rotation against resistance (the accessory nerve), and tongue position during protrusion and tongue atrophy or fasciculation (the hypoglossal nerve). Stroboscopic signs were evaluated by a laryngologist for the following characteristics: shape and movement of the vocal fold, glottal closure, mucosal wave, and supraglottic involvement. We conducted an interobserver study with blinded reviewers.

A total of 31 patients (8.4%) were eventually diagnosed with UFVP with lower cranial nerve palsy by the physical examination and electromyogram (EMG) tests. The patients ranged in age from 19 to 61 years, and there were 9 females and 22 males. All patients were examined with radiological scans, and neurologists were consulted to determine the possible causes of the patients' conditions.

The entire set of EMG tests was performed in 26 patients with lower cranial nerve palsy. A four-channel Nicolet VikingQuest Electromyographic Instrument (Nicolet Biomedical, Madison,

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WI, USA) was used for recording. For the laryngeal EMG (LEMG), concentric needle electrodes were placed percutaneously into the thyroarytenoid muscle, the cricothyroid muscle, and the posterior cricoarytenoid (PCA) muscle. The evoked response potentials (Eps) of the laryngeal muscles were investigated with monopolar needle electrodes that stimulated the recurrent laryngeal nerve, superior laryngeal nerve, and vagus nerve. The details of the LEMG techniques and interpretations have been described in our previous study.[6,7] The spontaneous potential activity patterns, motor unit potential (MUP) characteristics, and recruitment patterns of the involved laryngeal muscles were assessed. The amplitudes, latencies, durations, and waveforms of the Eps were evaluated. The measurement parameters for the lower cranial nerve-related EMG were similar to those used for LEMG. The recording electrodes were placed percutaneously into the following muscles: the sternocleidomastoid muscle (SCM), the trapezius muscle (TRA), and the genioglossus muscle. For the accessory nerve-evoked EMG of the SCM and TRA, the stimulating electrode was inserted percutaneously at the median point of the SCM posterior border. For the hypoglossal nerve-evoked EMG of the genioglossus muscle, the stimulating electrode was inserted percutaneously at the mandibular angle from the submandibular surface. The EMG data from the healthy sides were used as the normal controls.

All subjects, including the patients and control subjects, agreed to participate in this study and provided written informed consent. The study was approved by our local ethics committee and institutional review board.

The SPSS/PC 11.5 package (SPSS Inc., Chicago, IL, USA) was used for the statistical analyses of the data. The EMG results from all groups were tested with one-way analyses of variance. All tests were two sided, and a P value of <0.05 was considered statistically significant.

RESULTS

Clinical characteristics

A total of 31 patients (31/368, 8.4%) were eventually diagnosed with UVFP combined with lower cranial nerve palsy by physical examination and EMG tests.

The main complaints of the patients were moderate to severe persistent hoarseness with breathiness, vocal fatigue, effortful phonation, and aspiration. The durations ranged from 20 days to 38 years (median 10.5 months). The durations of the 12 patients with intracranial or skull-base lesions ranged from 1 month to 9 years (median 18 months), and these patients exhibited longer overall durations than the other patients. On laryngostroboscopy, 14 patients exhibited left-side VFP, and 17 patients exhibited right-side VFP. The affected vocal folds of all of the patients were found to be completely fixed in the paramedian or abduction positions with accompanying glottic insufficiency.

Based on the physical examinations and EMG, all 31 of the patients were found to have vagus nerve involvement (100%). A total of 27 patients (80.6%) were found with obvious abnormal physical examination of the accessory nerve injury. However, four patients (12.9%) who showed suspicious accessory nerve involvement had the atypical history and physical examination: two of them had no obvious causes, one had a history of a cold, and one may had congenital causes. And their SCM and TRA muscles atrophy was not so obvious. But the definite diagnoses of them were still neuropathic injury by EMG. The hypoglossal nerve involvement was observed in 13 cases (41.9%).

TABLE 1.
The Intracranial or Skull-base Lesions Caused Lower Cranial Nerve Palsy

Location	Lesions	Cases
Skull-base region	Glomus jugular tumors	4
	Schwannoma	3
	Meningioma	1
Intracranial region	Vertebral artery aneurysm	1
	Brainstem meningioma	1
	Cerebellopontine angle tumor	1
	Medulla oblongata infarction	1

Further radiological examinations revealed intracranial or skull-base lesions in 12 cases (38.7%; Table 1). Four patients (4/31, 12.9%) were ultimately diagnosed with infectious lower cranial neuritis by the neurologist. Two cases (6.4%) had previous histories of radiotherapy due to the malignant tonsil tumor 28 years prior and a malignant parotid gland tumor 2 years prior. No causes were identified in the other 13 cases (41.9%). The clinical characteristics of the patients with lower cranial nerve palsy are summarized in Table 2.

EMG characteristics

Based on our previous LEMG study,[6] the involved nerve impairments were grouped into complete and incomplete injuries. The EMG characteristics of incomplete impairments included reduced MUP amplitudes without abnormal MUP patterns or spontaneous electrical activity, mixed or pathological interference recruitment patterns with decreased amplitudes, and abnormal Eps of the target muscles (ie, delayed latency, prolonged duration, or decreased amplitude). The EMG characteristics of the complete impairments included denervation potential patterns (ie, electrical silence, fibrillation potentials, or positive sharp waves) or regeneration potential, mixed or simple recruitment patterns, and the absence of Eps in the target muscles. The general and evoked electromyography parameters of the SCM, TRA, and genioglossus muscles are listed in Tables 3 and 4. The fibrillation potentials or positive sharp waves can be observed in 7 patients with the course of 20 days to 2 months. The denervation potentials gradually reduce with the time. The regeneration potential of laryngeal muscles and SCM can be first seen in the patient with duration of 2 months. And most patients (14 patients) with regeneration potential had the duration of more than 6 months. The EMG patterns were showed in Figure 1. Among the 26 patients who underwent EMG tests, all (100%) exhibited the vagus nerve and the accessory nerve involvement, and 13 patients (50%) also exhibited hypoglossal nerve involvement. All of the patients with accessory nerve impairments exhibited both SCM and TRA muscle involvement. The synkinesia was only seen in the PCA muscles, in which the vagus nerves