Spasmodic Dysphonia

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ABSTRACT

Spasmodic dysphonia (SD) is focal dystonia involving intrinsic laryngeal muscles. It occurs because of a disorder in laryngeal motor neuron control during speech. Voluntary speech is affected, while involuntary actions such as loughing, crying, or coughing are not affected. An irregularity in organization in the central nervous system is observed in the pathophysiology of the disease. Some genetic mutations are found be responsible, and the disease is inherited in an autosomal dominant manner. There are two types of the dissease: adductor and abductor. The adductor type is more frequent. The diagnosis is usually delayed because of lack of exact objective criteria. Laryngeal electromyography (EMG) is the test that provides the most valuable diagnostic results. Injecting botulinium toxin into the densest muscle is the treatment of choice.

Keywords: Spasmodic dysphonia, laryngeal electromyography, Botulinium toxin

INTRODUCTION

Dystonia is a syndrome characterized by long-term and uncontrolled muscle contractions leading to abnormal and meaningless movements (1). Traditionally, dystonias are named according to the affected part or parts of the body. In focal dystonia, only a part of the body such as the larynx or eyelid is affected. In segmental dystonia, two lower facial muscles and the larynx or two adjacent muscle groups such as the neck and arm are affected. In multifocal dystonia, more than two muscle groups such as the larynx and the right foot or non-adjacent muscle groups are affected. Generalized dystonia affects both legs and at least one part of the body (2).

Spasmodic dysphonia (SD) is a focal dystonia in which intrinsic laryngeal muscles are affected (3). Although there is little information about the onset and formation, SD is a focal dystonia that develops secondary to a neurobiological mechanism that causes irregularity in laryngeal motor neuron control during speech (3). While voluntary speech is affected, involuntary movements such as crying, laughing, coughing, and yawning are not affected (3-5). Typically, the symptoms appear when the patient begins to speak. During speech, the vocal folds get very close to each other, and interruptions or pauses occur in the voice. The resulting sound is muffled, raucous, and effortful and often continuous, undamped, intermittent, or variable (6).

There is not much information about the natural course and epidemiology of SD. However, in light of existing information, no other part of the body is affected in the majority of patients with SD (7). The disease is usually observed in adults and in women (2, 5, 7). According to the study of Schweinfurth et al. (7), the disease is observed between the ages of 13–71 years. It was also reported that upper respiratory tract infection or stressful conditions may be triggered in some patients (7). Aronson (8) observed that the symptoms of SD remained stable at certain levels in some patients and worsened in other patients over time. In 29-disease series, Schaefer (9) observed intermittent symptoms within 2 years at the onset of the disease. Tanner et al. (10) specified that the symptoms of SD started slowly but worsened over time.

PATHOPHYSIOLOGY AND GENETICS

The pathophysiology of SD has not yet been fully explained. One of the reasons is that SD occurs only when talking, and emotional expressions are not affected (4). For this reason, it was previously believed that SD was only a psychogenic disease (3). However, it is currently believed that SD is caused by the difference between the mammalian speech system and the human speech system (3). Mammalian vocalizations are triggered by the cingulate cortex and periaqueductal gray matter to reach the pons and brainstem (11). This vocalization can be changed by environmental influences but is not acquired by learning (3). However, speech perception and production is acquired by learning. During communication, speech is produced as a cognitive process rather than by imitation (12). The links connecting the laryngeal cortex to the nucleus ambiguus have been found only in humans (13). For this reason, while the neural pathways related to speech are affected in patients with SD, emotional vocalization pathways are not affected (3). Simonyan et al. (14) attempted to examine the structural brain organization in patients with SD by combining neuroimaging and neuropathological methods (14). They found functional and structural changes in the corticobulbar and corticospinal tracts of 20 patients with

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SD in comparison to 20 healthy controls and found that these changes were significantly correlated with the clinical symptoms of SD. The authors concluded that these brain anomalies could affect the formation of voluntary vocalizations and thus determine the pathophysiology of the disease (14). Using functional MRI in patients with SD, Simonyan et al. (15) evaluated the neural activity in voluntary movements such as speech and in involuntary movements such as crying. Increased activity was detected in the primary motor cortex, insula, and superior temporal gyrus in both cases, whereas decreased activity during involuntary movements was detected in the basal ganglia, thalamus, and cerebellum (15). Through postmortem examinations of patients with SD, Simonyan (16) observed inflammation in the form of small clusters in the solitary tract, spinal trigeminal, ambiguous nuclei, inferior olive, and the reticular formation surrounding the pyramid and also detected mild neuronal degeneration and depigmentation in the substantia nigra and locus coeruleus. However, abnormal protein accumulation, demyelization, or axonal degeneration was not detected (16).

Studies on the genetic basis of the disease have recently been conducted. Some genetic mutations have been detected to cause various types of dystonia (2). While only SD is detected in patients with some genetic mutations, it can be seen as part of a large dystonia in some types of mutation (2). Dystonia is not found in the family history of the majority of patients with focal SD (17). At least 17 types of mutations that can cause dystonia were identified and named as DYT1-17 (2). While some syndromes are called as primary dystonia because they are related to only one muscle group, others are called dystonia-plus syndromes because multiple muscle groups are affected (2).

The DYT6 mutation results in dystonia that affects the cranial muscles and arms. Voice involvement is often the first finding in this type of dystonia (18). This syndrome was first identified via genetic analysis conducted in Amish–Mennonite families. Mutations in the THAP1 gene encoding a DNA-binding protein have been observed in these patients (19, 20). DYT6 mutations lead to an autosomal dominant genetic disorder (20). DYT6 mutations can cause a wide spectrum of diseases ranging from childhood-onset generalized dystonia to adult-onset focal dystonia including patients with SD (2). The detection of gene mutations or gene variations that cause dystonia can help us to understand the pathogenesis of SD, which has not yet been fully explained (2).

CLINICAL PRESENTATION

Two types of SDs have been defined: adductor SD and abductor SD (21). The adductor type has been observed in 90% of patients and is the more common type. Adductor SD is characterized by an intermittent, effortful, muffled, and tense voice. Abductor SD is characterized by an intermittent breathy voice due to contraction of the posterior cricoarytenoid muscle (22).

According to the severity, adductor SD is classified into the following three types: the spasm of only the true vocal folds, adductor spasm of the true and false vocal folds, and supraglottic constriction. Although adductor SDs occur due to the hyperabduction of the intrinsic laryngeal and pharyngeal muscles, upward (cephalic) movement of the larynx may accompany moderate-to-severe spasms. Thus, associated laryngeal movements also involve the extrinsic muscles. At the basis of these events, the vagal nerve is not the only one that innervates the intrinsic muscles. At the same time, the pharyngeal glossopharyngeal nerve and the cervical spinal nerves that innervate the extrinsic muscles can transmit the stimuli that are responsible for the spasm. For this reason, it is thought that the disease is a central nervous system disease rather than a peripheral nerve disease (6).

During the adductor SD attack, hidden lip movements are observed as a result of flushing and loss of voice. The patient makes articulator movements similar to stammering to make his/her voice heard. While doing this, the patient contracts the muscles of the neck, shoulder, and upper arm. Typically, these patients exhibit an angry and straight face, with a drooping mouth. Because speaking is exhausting, patients prefer to speak with a whisper or avoid speaking as much as possible and isolate themselves from society (6).

In abductor SD, the vocal folds are spasmodically exposed to hyperabduction, and sudden air leaks without phonation are detected. Its prevalence is less common (10%) than that of the adductor type. It starts with hoarseness or faintness that is not authentic in voice; after a few days or weeks, intermittent breathy air leaks become apparent. The patient speaks normally when he/she is comfortable and away from stress. While the leaks become apparent in consonants, the voice is normal when there are more vowels or when the patient speaks with a higher-pitch (6).

DIAGNOSIS

A thorough medical assessment must be conducted for every patient with SD. In otorhinolaryngology and head and neck examination, detailed laryngological examination (laryngeal videostroboscopy and objective sound analysis), neurolaryngologic and neurological examination (including laryngeal EMG and brain MRI), metabolic and radiological evaluation, and speech therapist evaluation should be additionally performed (23).

ENT examination and laryngoscopic examination should be performed for every patient complaining of SD. These patients are anxious about not being able speak properly, and they want to see their vocal folds. It is ensured whether or not there is a secondary pathology (such as a polyp or nodule) in the vocal folds of patients, and concerns about the presence of tumor are eliminated (6, 8).

Laryngeal videostroboscopic examination should be performed continuously and with flexible transnasal endoscopy and laryngeal telescopes under stroboscopic light. Supraglottic hyperfunction and stenosis in the anterior/posterior and lateral diameters can be detected with flexible fiberoptic laryngoscopy. An accurate diagnosis can be made owing to this compensatory hyperfunction. With repeated phonatory maneuvers, the adductor type and the abductor type can be distinguished from each other. For example, continuously and repeatedly producing the sounds like / i / - / hi /, / I / - / hi / may lead to the formation of abductor spasms. Likewise, in patients with abductor SD, the onset of voice is delayed after silent phonemes such as / f /, / sh /, / ch /, / h /, / k /, / p / and / s /. Vocalization of the sentences containing silent phonemes may also cause symptoms in patients with adductor SD. Adductor spasms can be generated

by repeated phonation of / pa /, / ta /, / ka / sounds, with the repetition of sentences containing many vowel phonemes or by reading the "diet passage" (22, 23).

Conventional objective sound measurements may not always be useful for diagnosing SD. However, these measurements may be useful for detecting compensatory mechanisms developed against spasms in the patient, for determining the pretreatment ground, and for determining the success of treatment. Objective measurements can help with the detection and digitization of the vibrations that can coexist in patients with SD. Rarely, these vibrations are associated with neurogenic dystonia and can be mistakenly diagnosed as SD in patients without laryngeal dystonia (22, 23).

Electromyography (EMG) is useful for SD evaluation. It is primarily useful for detecting the development of dysphonia depending on the peripheral nervous system, neuromuscular junction, and muscle disorders. Second, it finds the specific values in patients with SD. If spectrogram analysis and EMG are performed simultaneously, the delay between the electrical signal and the formation of audible phonation can be detected. Normally, this delay is 0–200 milliseconds. In SD, this delay can be between 500 milliseconds and 1 second (22,24). Furthermore, the determination of the more active muscle group with EMG is important in deciding the type of botulinum toxin to be administered.

The most important issue associated with the diagnosis of SD is that there is no objective criterion in the diagnosis and that the above mentioned tests cannot be performed everywhere. For this reason, early diagnosis is not established owing to patients' late visits to several doctors. According to a survey conducted by Creighton et al. (25) with 107 patients with SD in 2015, the average diagnosis period of patients was 4.5 years, and these patients were examined by four different doctors on average. Some patients (30%) received medical treatment other than botulinum toxin, whereas other patients (30%) received alternative treatment methods. The authors think that this situation is caused by the fact that there are few physicians with a sufficient level of knowledge to make an SD diagnosis and that the objective diagnostic criteria have not been fully determined (25).

TREATMENT

Once the diagnosis of SD is made, the treatment should be started. There are three basic options for treatment: speech therapy, nerve destruction, and neuromuscular blockade.

a. **Speech Therapy:** It facilitates the differentiation between psychogenic and functional dysphonia. It lessens the struggle, power, and fatigue of the patients during conversation. In addition, it decreases compensatory hyperfunction. It can provide disease control, particularly in mild cases. Depending on the patient's situation, traditional voice therapy methods, inhalation methods during speech, or singing therapy may be useful. These methods reduce the excessive pressure on the patient's speech. Baclofen or phenytoin (Dilantin) may also be effective in some patients (22, 23). When used together with medical or invasive treatment, it yields more successful results than a single treatment method. Although SD is not a psychogenic disease, speech problems cause stress in the patient, and this stress affects the complaints and treatment success (22, 23). For this reason, it is recommended that there should be a psychologist or psychiatrist in the treatment team (22, 23).

b. **Surgical Treatment:** In the 1970s and 1980s, Recurrent Laryngeal Nerve (RLN) destruction was considered by many surgeons as a surgical procedure that could be attempted (22, 23). Dedo et al. (26) developed this method and had an opportunity to apply it on a 300-disease series in those years. They followed up the patients for 5–14 years. They noted that patients showed a decrease in spastic speech and found an effective speech voice. They reported that they did not provide voice therapy to their patients in the preoperative period, but the correctly performed therapy accelerated the best sound reception in the postoperative period (26). By this means, thyroarytenoid (TA), posterior cricoarytenoid (PCA) and lateral cricoarytenoid (LCA) muscles are unilaterally denervated. The dissection is made on the left side because it is longer and more sensitive to damage (27). However, in the following years, Aronson and De Santo (28) reported that the success in voice decreased over time and that 2/3 patients had worse voice outcomes after 3 years. Although the detection and destruction of the selective branch of the RLN to the thyroarytenoid muscle was suggested, this method was usually conducted by experienced surgeons (22, 23). In 2005, the destruction of the branch of the RLN to the thyroarytenoid muscle was attempted using radiofrequency thermocoagulation (29). In 2008, TA myectomy was performed using a radiofrequency device. Although the results were initially good, complaints recurred in half of the patients who were then injected with botulinum toxin (30). In Japan, laryngologists have focused on surgical treatment, particularly because botulinum toxin is not state approved in the treatment of SD. Nowadays, thyroid cartilage relaxation surgeries are being attempted (31-33).

c. **Botulinum Toxin Injection**: Today, it is the most recommended treatment method for SD due to its potency, specificity, and low antigenicity (22). Botulinum toxin has seven different serotypes, which are structurally similar but immunologically different and are named as A, B, C, D, E, F, and G. Only type A neurotoxin has been approved for clinical use, and further studies are continuously being conducted for other serotypes. This toxin prevents the release of acetylcholine from the cholinergic extremities. The complex action mechanism has not yet been fully clarified. Clinically, the effect is observed with a delay of 48 hours (22).

   Adductor SD: Botulinum toxin injections into the TA muscle have been administered since 1984 and have been the preferred treatment method. Injections can be administered percutaneously or perorally with the assistance of EMG (34). A perforated, Teflon-coated, 27 gauge EMG needle is used to pass through the cricothyroid membrane. After being inserted, the needle is turned toward the superior and lateral side. An increase in electrical activity in the EMG when the patient phonates indicates that the needle has reached the TA. The disadvantage of this method is that it requires a per-

son who has a good command of the EMG (35). One of the percutaneous methods is performing the injection through the transcartilaginous, thyrohyoid, or cricothyroid membrane while the vocal fold image is generated through flexible laryngoscopy. In the peroral method, laryngeal topical anesthesia is administered before the procedure. The larynx is observed with flexible nasolaryngoscopy or indirect laryngoscopy. With a curved laryngeal needle, botulinum toxin is injected up to the top of the vocal folds. The advantage of this method is that it is known by laryngologists, and they do not require EMG. The disadvantage is that special needles are used, the injection of the toxin lasts longer, and the toxin remains unused in the catheter (35). One of the most important benefits of using botulinum toxin in therapy is that the dosage can be adjusted. The dosage may vary from 1 MU to 30 MU depending on the patient's response to the treatment, the level of side effects, and the technique used. Blitzer et al. (36) recommend initiating the treatment with an average of 1 MU and then adjusting the dose according to the patient's response. According to Blitzer et al (36), 90% of patients with adductor SD benefit from the botulinum toxin treatment and have a symptom-free period of 15 weeks on average.

Abductor SD: In these patients, botulinum toxin injections have been administered into the PCA muscle since 1989 (36). As a treatment protocol, Blitzer et al. (36) recommend that, first of all, a more active PCA should be determined through EMG and a 3.75 MU Botulinum toxin should be injected into the muscle; then, botulinum toxin at lower doses should be injected into the contralateral PCA muscle of patients with ongoing symptoms during the follow-ups. They found that botulinum toxin effects started within a minimum of 4 days, reached the maximum level on the 10th day, and that the patients benefited from it for an average of 10 weeks (36). After botulinum toxin injection, mild side effects such as breathy phonation in 35% of patients, breath obstruction following fluid intake in 15% of patients, and pain and sensitivity at the injection site in 1% of patients can be seen (36).

CONCLUSION

Spasmodic dysphonia is a type of dystonia that can be observed in laryngeal structures with other muscle groups of the body as a result of a type of irregularity in the central nervous system. It is observed as adductor SD in 90% of patients. It is essential to diagnose the disease before treatment. Despite the use of voice therapy and surgical methods, recurrent botulinum toxin injections are the gold standard of treatment.

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