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REVIEW

Dysphagia in Lateral Medullary Syndrome: A Narrative Review

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Abstract

Dysphagia is a common clinical feature of lateral medullary syndrome (LMS) and is clinically relevant because it is related to aspiration pneumonia, malnutrition, increased mortality, and prolonged hospital stay. Herein, the pathophysiology, prognosis, and treatment of dysphagia in LMS are reviewed. The pathophysiology, prognosis, and treatment of dysphagia in LMS are closely interconnected. Although the pathophysiology of dysphagia in LMS has not been fully elucidated, previous studies have suggested that the medullary central pattern generators coordinate the pharyngeal phases of swallowing. Investigation of the extensive neural connections of the medulla oblongata is important in understanding the pathophysiologic mechanism of dysphagia in LMS. Previous studies have reported that most patients with dysphagia in LMS have a relatively good prognosis. However, some patients require tube feeding for several months, even years, due to severe dysphagia, and little has been reported about conditions associated with a poor prognosis of dysphagia in LMS. Concerning specific therapeutic modalities for dysphagia in LMS, in addition to general modalities used for dysphagia treatment in stroke patients, non-invasive modalities, including repetitive transcranial magnetic stimulation and transcranial direct current stimulation, as well as invasive modalities, such as botulinum toxin injection, balloon catheter dilatation, and myotomy for relaxation of the cricopharyngeal muscle, have been applied. For the appropriate application of therapeutic modalities, clinicians should be aware of the recovery mechanisms and prognosis of dysphagia in LMS. Further studies on this topic, as well as studies involving large numbers of subjects on specific therapeutic modalities, should be encouraged.

Keywords Lateral medullary syndrome · Dysphagia · Pathophysiology · Prognosis · Treatment

Abbreviations

LMS	Lateral medullary syndrome
NA	Nucleus ambiguus
NTS	Nucleus tractus solitarius
rTMS	Repetitive transcranial magnetic stimulation
tDCS	Transcranial direct current stimulation
CP	Cricopharyngeal
UES	Upper esophageal sphincter
MRI	Magnetic resonance imaging
BTX-A	Botulinum toxin type A
DOSS	Dysphagia Outcome and Severity Scale

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Introduction

Lateral medullary syndrome (LMS), also called Wallenberg's syndrome, is a neurological disease caused by ischemia in the lateral part of the medulla oblongata (medulla) due to an occlusion in a vertebral artery or posterior inferior cerebellar artery [1]. Clinical features of LMS vary according to lesion location and consist of dysphagia, cross body sensory deficits (ipsilateral face and contralateral trunk and extremities), ataxia, dizziness, and Horner's syndrome (ptosis, miosis, anhidrosis) [1].

Speech is a process that requires articulation and pronunciation, and spoken language involves the process of communicating thoughts. Dysarthria is a speech impairment caused by disturbance of oral musculature control, but dysphasia is a language impairment that results in difficulties in understanding or forming words and sentences. Dysarthria and dysphasia can often occur concurrently. However, speech impairments such as dysarthria, hoarseness, and dysphonia can occur in LMS patients, but language impairments such as aphasia or dysphasia have not been reported in LMS patients [2–4]. Swallowing

is an oral musculature control process that involves mastication, bolus formation, and the passing of food from the mouth to the stomach via the pharynx and esophagus. Swallowing-related structures, such as the nucleus ambiguus (NA) and the nucleus tractus solitarius (NTS), are located in the lateral medulla, and swallowing impairment (dysphagia) is a common clinical feature of LMS (51% to 94% incidence) [1, 5].

Dysphagia is clinically important because it is related to aspiration pneumonia, malnutrition, increased mortality, and prolonged hospital stay [6–11]. Although the pathophysiology of dysphagia in LMS has not been fully elucidated, previous studies have suggested that the swallowing center, which includes the NA and NTS, coordinates the pharyngeal phases of swallowing, and the extensive neural connections within the swallowing center are important in understanding the pathophysiologic mechanisms of dysphagia in LMS [12–15].

The severity and duration of dysphagia in LMS can vary widely from very mild and transient to extremely severe and prolonged. Regarding the prognosis of dysphagia in LMS, previous studies have reported that the majority of patients with LMS initially exhibit severe dysphagia and require non-oral feeding; however, they often recover rapidly and return to oral feeding within the first few months after onset [16–20]. In contrast, some patients with severe dysphagia require tube feeding for several months or years [14, 21–26]. Thus, prognosis prediction of dysphagia is clinically important to establish appropriate therapeutic strategies; however, there too few studies to allow precise prognosis prediction of dysphagia in LMS.

Regarding specific therapeutic modalities for application in dysphagia in LMS, in addition to general modalities, such as controlling bolus volume and viscosity, used for dysphagia treatment in stroke patients, both non-invasive modalities, such as repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), and invasive modalities, such as botulinum toxin injection, balloon catheter dilatation, and myotomy for relaxation of the cricopharyngeal (CP) muscle, have been applied [25–37]. Although the association of dysphagia with medical complications and reduced quality of life is considered important, there have been few studies on specific therapeutic modalities suitable for dysphagia in LMS.

Herein, we provide a review of the pathophysiology, prognosis, and treatment of dysphagia in LMS.

Pathophysiology of Dysphagia in LMS

Normal Physiology of the Pharyngeal Phase of Swallowing

The swallowing process consists of oral, pharyngeal, and esophageal phases with neural structures in the lateral medulla mainly involved in the pharyngeal phase [13, 38]. Once a bolus enters the pharynx, the pharyngeal phase of swallowing begins. Pharyngeal distention pressure, produced by the bolus passage, acts as a stimulus to trigger the involuntary and coordinated pharyngeal phase of swallowing. Presence of the bolus in the oropharynx elevates the soft palate, and contraction of the upper pharynx constrictor to prevent bolus backflow initiates pharyngeal peristalsis to move the bolus toward the esophagus. To protect the airway, respiration is temporarily inhibited as the larynx rises to close the glottis. Furthermore, the upper esophageal sphincter (UES) relaxes and opens the pharyngeal passage to allow movement of the bolus to the esophagus. After the bolus passes into the esophagus, the UES immediately closes to prevent bolus regurgitation.

The medulla contains several cranial nerve nuclei related to swallowing: the NTS, the spinal trigeminal nucleus, the NA, the vagal dorsal motor nucleus, and the hypoglossal nucleus [13, 20, 39]. The NTS is a purely sensory nucleus located in the dorsolateral medulla. The NTS accepts taste information and visceral sensations from the base of the tongue, as well as the epiglottis and pharyngolaryngeal areas through cranial nerves VII, IX, and X [20, 39–41]. The spinal trigeminal nucleus, located in the lateral medulla, accommodates tactile senses in oral structures, including the gums, tongue, and palate, as well as in facial areas through cranial nerves V, VII, IX, and X [20, 39, 41, 42]. The NA, a group of large motor neurons, is located in the lateral medulla posterior to the inferior olivary nucleus. The NA controls the ipsilateral muscles of the soft palate, pharynx, larynx, and upper esophagus through efferent motor fibers of cranial nerves IX, X, and XI [43, 44]. The vagal dorsal motor nucleus is a cranial nerve motor nucleus for the vagus nerve (CN X) and is located in the dorsal lateral area of the medulla. Along with the NA, it controls laryngeal and pharyngeal muscles [41, 45]. The hypoglossal nucleus, located in the dorsal medial area of the middle medulla, is a cranial nerve motor nucleus for the hypoglossal nerve (CN XII). It innervates all extrinsic and intrinsic muscles of the tongue, except for the palatoglossus, which is innervated by the vagus nerve [12, 20, 46]. Among the above five cranial nerve nuclei, four nuclei (the exception being the hypoglossal nucleus) are located in the lateral medulla.

Several studies have described the central pattern generator as the swallowing center, which mainly consists of two anatomic regions within the lateral medulla: (1) a dorsal region consisting of the NTS and surrounding neurons and (2) a ventral region corresponding to the NA and the reticular formation around the NA [12–15]. This central pattern generator is located on each side of the brainstem. The neural pathways originating from the premotor neurons are connected in and around the NTS, the NA, and the surrounding reticular formation, and they interconnect the

ipsilateral cranial nerves' sensory and motor nuclei in the medulla. These neural structures are also connected to the peripheral afferents and the cerebral cortex. Furthermore, their neural fibers are known to cross the midline of the brainstem and interconnect the bilateral central pattern generators. Consequently, the central pattern generator on either side coordinates the pharyngeal phase of swallowing reciprocally. The dorsal region of the central pattern generators integrates cortical input with peripheral afferents and transmits the integrated input to the ventral region of the central pattern generators. Subsequently, the ventral region produces the coordinated, sequential muscle activities of swallowing through its projections to the ipsilateral and contralateral cranial nerve motor nuclei.

Pathophysiology of Dysphagia in LMS

Occurrence and severity of dysphagia in LMS depend on the extent of involvement of the swallowing-related structures in the infarct lesion. Because the swallowing-related NA, NTS, vagal dorsal motor nucleus, and spinal trigeminal nucleus are located in the lateral medulla, dysphagia is a common symptom of LMS (Fig. 1) [47].

The NA controls the muscles of the soft palate, the pharynx, the larynx, and the upper esophagus; therefore, an LMS lesion involving the NA can result in pharyngeal peristaltic weakness, vocal cord palsy, or abnormal UES relaxation [20, 48, 49], which could lead to a moderate to severe bolus residue in the pyriform sinuses after swallowing and the possibility of subsequent aspiration. In addition, speech impairment such as dysarthria and dysphonia could result from an LMS lesion involving the NA, although aphasia and dysphasia are not associated with LMS [2–4]. An LMS lesion containing the vagal dorsal motor nucleus can also result in pharyngeal and laryngeal muscle weakness because, along with the NA, the vagal dorsal motor nucleus is involved in controlling the pharyngeal and laryngeal muscles [20, 48, 49]. A few studies have reported on muscle weakness due to injuries of these cranial motor nuclei in LMS. Aydogdu et al. reported various weaknesses among 20 patients with LMS; 7 patients (35%) with palatal paresis, 3 patients (15%) with facial weakness, 12 patients (60%) with slow laryngeal elevation, and 20 patients (100%) with vocal cord paresis [13]. Moreover, Kwon et al. investigated 37 patients with LMS 10 patients (27%) with facial palsy, 5 patients (14%) with tongue weakness, and 17 patients (46%) with soft palate weakness [50].

Additionally, because the NTS accepts visceral sensations from the base of the tongue, epiglottis, and pharyngolaryngeal areas, a LMS lesion involving the NTS can, before swallowing, lead to premature spillage of the bolus into the pharynx and can decrease the patient's awareness of a pharyngeal residue after swallowing [20, 39]. An LMS lesion of the spinal trigeminal nucleus can cause decreased bolus control and reduced awareness of oral bolus residue or pooling because that nucleus is involved in the tactile senses of oral structures [20, 39].

Because the hypoglossal nucleus is located in the dorsal medial area of the middle region of the medulla, tongue dysfunction is rare in LMS. However, in lateral lower medullary infarction cases, hypoglossal nerve palsy without the involvement of the anteromedial part of the medulla has been reported [51]. In addition, animal studies have reported that the rhythmic movements of the tongue, which participate in swallowing and mastication, driven by the hypoglossal nucleus receive inputs from the dorsal medullary reticular formation and the NTS [52, 53]. Therefore, tongue dysfunction could occur in the absence of direct injury of the hypoglossal nucleus or nerve.

Nuclei in the central pattern generator for swallowing are extensively interconnected; therefore, the swallowing-associated neural connections are, initially after the onset of LMS, affected collectively in LMS [12–15]. Aydogdu et al. described dysphagia in LMS as an acute disconnection syndrome resulting from a sudden disconnection between the bilateral central pattern generators, and the unaffected central pattern generator cannot produce the required coordinated sequential muscle activity during the pharyngeal phase of swallowing during the acute stage, thus resulting in bilateral dysfunction of the swallowing muscles [13]. Subsequently, the unaffected premotor neurons and the central pattern generator may eventually begin to function so that recovery from dysphagia can occur over time. On that basis, the authors suggested that the extent of a lesion involving the central pattern generator and its associated connections is important in determining the severity and duration of dysphagia in LMS [13]. However, the pathophysiology and recovery mechanisms of dysphagia in LMS have not been fully elucidated.

Previous Studies on Prognosis of Dysphagia in LMS

Prognosis prediction for dysphagia in stroke patients is clinically important because it can provide information useful in determining the need to install a percutaneous endoscopic gastrostomy tube, estimating the duration of the dysphagia rehabilitation period, and establishing therapeutic strategies for dysphagia. For example, when nasogastric tube feeding is deemed necessary for more than one month, gastrostomy can be considered [54]. Furthermore, the application of more effective therapeutic modalities for dysphagia, including non-invasive and invasive modalities, can be considered when a poor prognosis is predicted. However, there have been few investigations reporting on prognosis prediction for dysphagia in LMS (Table 1) [16–20].

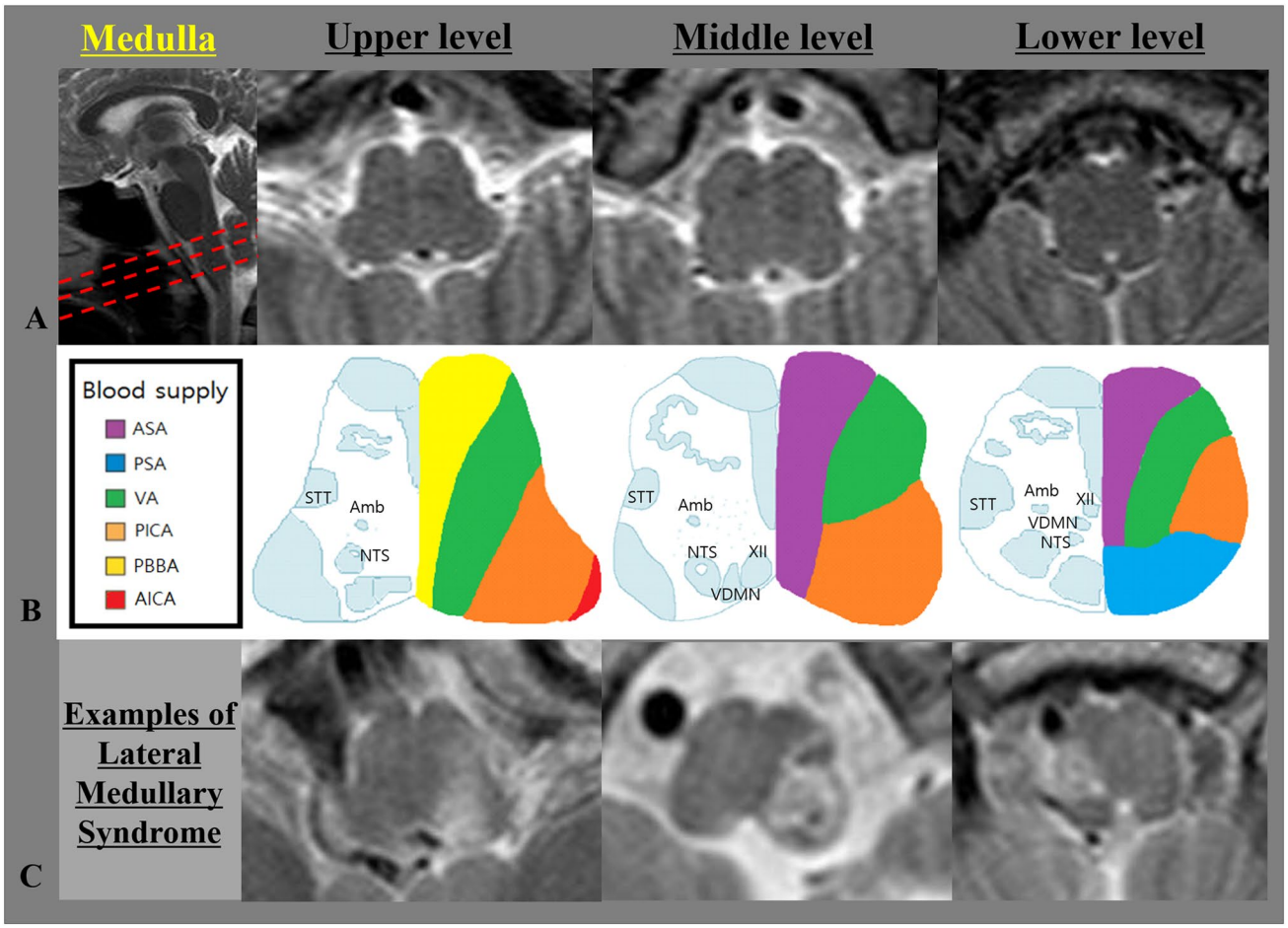

Fig. 1 Anatomy of swallowing-related structures in the medulla oblongata. **a** The medulla can be divided into upper, middle, and lower levels, which can be distinguished based on their characteristic shapes in T2-weighted brain magnetic resonance images. The upper medulla level is seen as a massive bulge near the restiform body in the dorsolateral area of the medulla; the middle medulla level shows a rugged lateral surface due to the large olive nucleus forming a prominent bulge; and the lower medulla level is smaller than the upper parts and has a rounded shape. **b** Anatomic location of swallowing-related structures and blood supply region: diagrams at the upper, middle, and lower medulla levels. There are four vessels to the medulla: ASA, PSA, VA, and PICA. The ASA is responsible for blood supply to the anterior and medial areas of the medulla, while the PSA is responsible for the posterior area blood supply. VA and PICA are responsible for blood supply to the lateral and dorsolateral areas of the medulla, respectively. **c** Lateral medullary syndrome (LMS) is caused by ischemia in the lateral part of the medulla due to an occlusion in the VA or PICA. Example lesions of LMS in the T2-weighted magnetic resonance images at the upper, middle, and lower medulla levels are shown. *ASA* anterior spinal artery, *PSA* posterior spinal artery, *VA* vertebral artery, *PICA* posterior inferior cerebellar artery, *PBBA* paramedian branch of basilar artery, *AICA* anterior inferior cerebellar artery, *STT* spinal trigeminal tract and nucleus, *Amb* nucleus ambiguus, *XII* hypoglossal nucleus, *VDMN* vagal dorsal motor nucleus, *NTS* nucleus tractus solitarius

In 1995, Crary et al. reported on the prognoses of six patients with LMS [16]. Three and one patients were able to have a total oral diet within one month and three months after onset, respectively. In addition, one patient was able to eat semi-solid food within three weeks after onset, making it unnecessary to maintain a nasogastric tube. However, the remaining patient could not have a total oral diet until seven months after onset. In 2000, Robbins et al. investigated the dysphagia prognoses of 23 patients with medullary infarction (LMS: 13 patients) [20]. Ten of the 23 patients had no dysphagia symptoms from onset (6 patients, posterior spinal artery infarction; 2 patients, anterior spinal artery infarction [medial medullary syndrome]; and 2 patients with LMS). By contrast, among the 13 patients with initial dysphagia, 12 patients were able to have an oral diet within 1–2 months; however, in one patient with bilateral medullary infarctions due to two stroke incidents, it was not possible to remove the tube after 110 days. During the same year, Meng et al. investigated dysphagia outcomes in 36 patients with LMS [17]. Eight (22%) of the 36 patients relied on nasogastric tube feeding at discharge. After discharge, 27 of the 36 patients were contacted via follow-up phone calls, and three (12%)

Table 1 Previous studies related to the prognosis of dysphagia in lateral medullary syndrome

Authors	Publication year	Number of patients (total)	Number able to have an oral diet within the first few months	Number with continued tube feeding for several months to years
Crary et al.	1995	6 (6)	3: Total oral diet within 1 month 1: Total oral diet at 3 months 1: Partial oral diet within 3 weeks	1: Total oral diet at 7 months
Robbins et al.	2000	13 (23)[a]	12: Oral diet within 1–2 months	1: Tube feeding at 3 months
Meng et al.	2000	27 (36)[b]	24: Oral diet within 1–4 months	3: Tube feeding at 4 months
Chun et al.	2017	40 (40)	20: Full oral diet within 1–6 months 17: Partial oral diet within 1–6 months	3: Tube feeding at 6 months
Kim et al.	2018	11 (11)	11: Full oral diet within 1–2 months	–

[a]Ten patients were excluded due to a lack of dysphagia symptoms since onset

[b]Nine patients were excluded due to a lack of response to follow-up phone calls

of the 27 said they still were tube-fed at four months after onset. In 2017, Chun et al. reported on dysphagia outcomes after six months from onset in 40 patients with LMS; of those, 21 patients (52.5%) were able to resume a full oral diet, 3 patients (7.5%) still needed nasogastric tube feeding, and the remaining 16 patients (40%) had achieved a partially oral diet [18]. Recently, Kim et al. (2018) performed videofluoroscopy every two weeks in 11 patients who needed tube feeding due to severe dysphagia following LMS and reported that all patients had resumed a full oral diet at an average of 52.2 ± 21.8 days after onset [19]. In addition to the above studies, several case studies have reported on patients with LMS who failed to resume an oral diet for several months to years after onset [14, 21–26]. Based on the abovementioned studies, the majority of LMS patients with dysphagia can resume an oral diet within one to three months after infarct onset; regardless, approximately 10% of LMS patients may continue tube feeding for several months or years after onset. However, those studies did not describe several conditions (e.g., demographic factors and location and extent of lesions) of the patients with prolonged dysphagia, except for one patient with bilateral medullary infarction. Therefore, further prospective studies to clarify the factors related to a poor prognosis of dysphagia in LMS are needed.

Treatment for Dysphagia in LMS

Because therapeutic modalities for dysphagia vary with its pathophysiology and prognosis, precise evaluation of the characteristics of the patient's dysphagia by using videofluoroscopy and brain magnetic resonance imaging (MRI) is necessary during the early stage after onset. For dysphagia patients, the following general treatment modalities have been performed: controlling bolus volume and viscosity; using a variety of swallowing techniques, maneuvers, and exercises for strengthening the swallowing muscles and improving expectoration; and applying neuromuscular electrical stimulation to the suprahyoid and/or infrahyoid muscles to increase anterior hyoid motion [55–66]. In addition to the above general treatment modalities, the following several specific modalities have been applied for dysphagia in LMS: (1) non-invasive modalities, including rTMS and tDCS, and a novel swallowing maneuver called vacuum swallowing; and (2) invasive modalities including botulinum toxin injection, balloon catheter dilatation, and myotomy of the CP muscle for UES opening [25–37].

Non-invasive Modalities

Recently, non-invasive neuromodulation using rTMS and tDCS has been applied for dysphagia treatment in stroke patients [26–28, 67–74]. Both rTMS and tDCS can increase cortical excitability through non-invasive transcranial brain stimulation by facilitating the secretion of several neurotransmitters in the brain [75–78]. Consequently, these modalities can induce a change in the excitability of the stimulated region of the brain cortex and affect the entire associated network. Regarding rTMS, few studies have reported on its effect on dysphagia in LMS patients [26–28]. In 2010, Khedr et al. investigated the effect of rTMS on dysphagia in 22 patients with brainstem infarction (LMS: 11 patients) [27]. The rTMS group (11 patients; application area, esophageal motor cortex of both hemispheres; frequency, 3 Hz; intensity, 130% of the resting motor threshold with 300 pulses for 10 min/session and one session/day for 5 consecutive days) showed significant dysphagia improvement compared to that of the sham-treated group (11 patients, no changes in dysphagia grade during the study). Subsequently, Rhee et al. reported on a patient with LMS who exhibited dysphagia improvement and achieved oral diet capability after rTMS application (application area, esophageal motor cortex of both hemispheres; frequency, 5 Hz; intensity, 120% of the resting motor threshold and 500 pulses for 10 min/session with one session/day and

five sessions/week for 2 weeks) [28]. In 2016, Verin et al. reported on the treatment of two patients with LMS who showed dysphagia until three years after onset. The treatment included rTMS (application area, mylohyoid cortical area of both hemispheres; frequency, 1 Hz; intensity, 20% above the threshold value and 100 pulses for 10 min/session with one session/day for 5 consecutive days and 5 times at 3-month intervals), electrical stimulation of the submental area during rTMS, and myotomy of the inferior pharyngeal constrictor and CP muscles [26]. After surgical healing, both patients showed rapid improvement in swallowing, and both patients resumed an oral diet at six months after surgery.

In LMS, swallowing maneuvers and exercises can be used to compensate for pharyngeal peristalsis weakness or abnormal UES relaxation [55–59, 64, 65]. When pharyngeal peristalsis is weak, turning the head to the weaker side during swallowing may help accomplish closure of the hemipharynx of the weaker side and help the bolus move to the stronger side of the pharynx [55, 56]. Furthermore, lingual exercises for improving tongue base action or the effortful swallowing practice can be helpful in cases with weak pharyngeal contraction [64, 65]. The UES opening is associated with contraction of the suprahyoid muscle and relaxation of the CP muscle. Hence, the Mendelsohn maneuver, which is a method of holding the larynx in the elevated position for several seconds during swallowing, and the Shaker exercise, which is a method of repetitive head raising movements from a supine position, have been commonly used to increase the extent and duration of suprahyoid muscle contraction [57–59]. Kunieda et al. reported on a novel alternative swallowing maneuver for dysphagia in LMS, "vacuum swallowing", in which a negative pressure in the esophagus is created via voluntary contraction of the diaphragm before swallowing, thereby allowing the bolus to pass down to the esophagus [60]. The authors reported on a patient with LMS who used this maneuver and in whom oral feeding became possible at three months from onset.

Invasive Modalities

For relaxation of the CP muscle, invasive treatments such as botulinum toxin injection, balloon catheter dilatation, and myotomy have been applied [79]. Many studies have demonstrated the effects of such procedures on dysphagia in patients with CP dysfunction due to various brain pathologies including parkinsonism, progressive supranuclear palsy, amyotrophic lateral sclerosis, multiple sclerosis, post-traumatic encephalopathy, and stroke [25, 29–37, 79–108]. However, few studies have reported on the effects of these modalities in patients with LMS [25, 26, 29–32, 34–37].

The use of botulinum toxin type A (BTX-A) for treatment of CP dysfunction was first reported in 1994 by Schneider et al. [33]. Since then, a few studies have reported on the effect of BTX-A injection on the CP muscle in patients with dysphagia in LMS [34–37]. In 2006, Kim et al. investigated the therapeutic effectiveness of BTX-A (100 units into both CP muscles) in eight stroke patients with CP dysfunction (LMS in 2 patients) [34]. Although six (75%) of the eight patients showed significant improvement after BTX-A injection, the two patients with LMS did not show significant improvements. Subsequently, Lee et al. recruited eight patients with CP dysfunction (LMS in four patients), and 100 units of BTX-A or 750 units of Dysport were injected into both CP muscles [35]. Although some improvement (statistically insignificant) was observed functional dysphagia scale results, six of the eight patients showed improvement on videofluoroscopy; however, no patient was able to change from tube feeding to oral feeding. In 2017, Alfonsi et al. investigated the therapeutic effectiveness of BTX-A in 67 patients with CP dysfunction (LMS in 14 patients) [36]. In each patient, 15–20 units of BTX-A was injected into the unilateral CP muscle, and dysphagia was quantified using the Dysphagia Outcome and Severity Scale (DOSS; range 1–5 with a lower score indicating more severe dysphagia). Among 14 patients with LMS, 4 patients were classed as high responders (DOSS score increase >2 levels), 7 patients were low responders (DOSS score increase ≤ 2 levels), and, of those 11 responsive patients, the beneficial clinical effect of BTX-A lasted more than two months. However, the remaining three patients with no DOSS response showed transient worsening of dysphagia for one to three weeks after treatment. During the same year, Battel et al. reported on a patient with LMS who had effective saliva control, mouth opening, and UES relaxation after BTX-A injection (100 units into both parotid and submandibular salivary glands, 40 units into both temporalis muscles, 80 units into both masseter muscles, and 15 units into the right CP muscle) [37]. Among those four previous studies, small sample sizes prevail with only 21 patients among those studies. In addition, the injection schemes were not unified and the effects of botulinum toxin injection were inconsistent. Therefore, further studies involving larger numbers of subjects and including well-defined injection approaches, subject indications, and outcome definitions should be undertaken.

Concerning balloon catheter dilatation, a few studies have reported on its effect on dysphagia in patients with LMS [29–31]. In 2000, Katoh et al. reported on a patient who was able to have an oral diet after undergoing balloon catheter dilatation of the CP muscle at three months after onset [29]. In 2014, Miyamoto et al. reported on a patient who showed rapid improvement of dysphagia after intermittent air stretching of the CP muscle via balloon catheter dilatation [30]. Recently, Ogata et al. demonstrated that early post-onset induction of balloon catheter dilatation for the CP muscle might be useful for achieving early improvement of swallowing dysfunction in LMS [31]. The authors performed

balloon catheter dilatation of the CP muscle six times per month in an elderly patient (77 years old) who suffered from recurrent aspiration pneumonia, and the patient was able to recover swallowing function without recurrence of aspiration pneumonia. However, the above three studies have limitations because all are case reports. Therefore, further studies involving large numbers of subjects are warranted. Furthermore, the associated indications, complications, and therapeutic outcomes of balloon catheter dilatation should be clarified through further comparative studies.

A few studies have reported on the effect of CP myotomy in patients with LMS [25, 26, 32]. Myotomy of the CP muscle has been performed on patients who did not show improvement or had complications following botulinum toxin injection or balloon catheter dilatation [22, 29]. In 2016, Sruthi et al. reported that a patient with severe dysphagia (persisting for more than 1.5 years) and no improvement after BTX-A injection (40 units into both CP muscles) was able to have an oral diet after CP myotomy [25]. In 2019, Osamu et al. reported that three patients with severe dysphagia and no improvement after balloon catheter dilatation showed rapid improvement of dysphagia after CP myotomy [32]. Among the pathologic findings from the examination of the resected CP muscle of the three patients, fibrosis due to mechanical damage associated with the balloon catheter dilatation was observed. In all three cases, dysphagia has not recurred during several years following the CP myotomies, but one patient experienced esophagopharyngeal reflux as a complication. It has been reported that the presence of esophagopharyngeal reflux or pneumonia after CP myotomy may be related to improper closing of the UES; moreover, vocal hoarseness may occur due to recurrent laryngeal nerve injury [108]. Because patients with dysphagia with LMS can recover rapidly, often within 1–3 months, further studies into the indications for and complications of CP myotomy are necessary.

Conclusion

In this article, the pathophysiology, prognosis, and treatment of dysphagia in LMS were reviewed. There are close interconnections among the pathophysiology, prognosis, and treatment of dysphagia in LMS. Because dysphagia in LMS is known to have a relatively good prognosis, clinicians need to concentrate on patients who have a poor dysphagia prognosis, particularly during the early post-onset period. However, very little has been reported on the conditions associated with a poor prognosis for dysphagia in LMS. Hence, further studies on this topic should be encouraged.

For the selection of an application-precise therapeutic modality, clinicians should be aware of the recovery mechanisms of dysphagia in LMS; however, as far as we are aware, there is only one report describing a possible recovery mechanism for dysphagia in LMS. That paucity may be related to the shortage of studies on the pathophysiologic mechanisms of dysphagia in LMS. One previous study indicated that restoration of transient dysfunction of an unaffected central pattern generator for swallowing was related to recovery from dysphagia, thus suggesting a potential recovery mechanism for dysphagia in LMS [13]. When central pattern generator restoration is demonstrated as a recovery mechanism of dysphagia in LMS, we believe that clinicians would be able to apply appropriate therapeutic modalities that can facilitate stimulation of the unaffected central pattern generator for swallowing, such as by the precise application of rTMS or tDCS during the early stage of LMS. Therefore, further studies on this topic, as well as studies aimed at elucidating other potential recovery mechanisms of dysphagia in LMS, should be undertaken. Furthermore, studies that include large numbers of subjects with various indications and complications associated with invasive therapeutic modalities for patients who are refractory to non-invasive therapeutic modalities are also necessary.

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Compliance with ethical standards

Conflict of interest The authors report no conflict of interest.

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