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REVIEW ARTICLE

Oro-Pharyngeal Dysphagia in Parkinson's Disease and Related Movement Disorders

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ABSTRACT

Oro-pharyngeal dysphagia is a common symptom in patients with Parkinson's disease (PD) and related disorders, even in their early stage of diseases. Dysphagia in these patients has been underdiagnosed, probably due to poor the self-awareness of the conditions and the underuse of validated tools and objective instruments for assessment. The early detection and intervention of dysphagia are closely related to improving the quality of life and decreasing the mortality rate in these patients. The purpose of this paper is to give an overview of the characteristics of dysphagia, including the epidemiology, pathophysiology, and clinical symptomatology, in patients with PD compared with other parkinsonian disorders and movement disorders. The management of dysphagia and future research directions related to these disorders are also discussed.

Key Words Dysphagia; Dystonia; Parkinsonism.

Oro-pharyngeal dysphagia is a common symptom in patients with neurological diseases. In particular, in neurodegenerative conditions such as Parkinson's disease (PD) and related disorders, the prevalence of dysphagia increases rapidly as the disease progresses. A systematic review showed that patients with PD developed oro-pharyngeal dysphagia approximately three times more than healthy elderly people.[1] Dysphagia can result in clinical complications, including malnutrition, dehydration, and aspiration pneumonia, affecting the quality of life and eventually increasing the mortality rate in these patients. However, the early detection of swallowing problems is not always easy because dysphagia may be asymptomatic and the self-awareness of patients is poor.[2]

In patients with Parkinson plus syndrome, dysphagia occurs significantly earlier from disease onset than in those with PD.[3] Other movement disorders, such as dystonia or myoclonus, can also present various swallowing difficulties depending on the affected muscles. Rarely, involuntary abnormal movements occur exclusively during swallowing.[4-6] This paper presents an overview of dysphagia, including its epidemiology, pathophysiology, and clinical symptomatology, and its management from the clinical perspective in patients with PD and related disorders.

PARKINSON'S DISEASE

Epidemiology

The symptoms of dysphagia were described in the first report of James Parkinson.[7] The prevalence of oro-pharyngeal dysphagia in patients with PD differs across studies, from 18.5% to 100% depending on their definition of dysphagia, assessment tools, and the assessment time point from the onset of the disease.[1,2,8-12] Usually, it is known that the prominent symptoms of dysphagia appear in the late stage of PD. A cohort study showed that severe dysphagia was reported approximately 10–11 years after the motor symptoms had appeared, and the prevalence was 68% even in the on-drug phase of patients with late-stage PD.[9] However, the poor awareness of swallowing problems in patients with PD may result in the underestimation of their presence.

Mild oro-pharyngeal symptoms of dysphagia were frequent in the early stage of PD, and dysphagia might be the first sign of

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the disease.[13-15] A recent meta-analysis showed that the pooled prevalence was 35% on the subjective or patient-related measure but increased up to 82% when the objective or clinician-related measure was employed.[1] Poor correlation between self-report and the water swallow test for dysphagia demonstrated that the subjective measure is not a good indicator for detecting dysphagia in patients with PD.[2] Other studies using a questionnaire or instrumental test, such as a videofluoroscopic swallowing study (VFSS) or fiberoptic endoscopic evaluation of swallowing (FEES), also showed that oro-pharyngeal dysphagia emerged in more than 50% of patients with PD who reported no problem with swallow.[10-12] Therefore, a patients' self-report or response to a simple question whether or not they have any swallowing problem can lead to an underestimation of dysphagia in patients with PD. The use of validated questionnaires or objective instrumental studies is essential for the early detection of dysphagia and proper management for those patients.

Although symptoms of dysphagia are frequent even in the early stage of PD, compensation mechanisms may occur at the cortical level,[16] and a severe form of dysphagia is more associated with the late stage of the disease. Clinical predictors highly correlated with severe dysphagia in PD are the Hoehn & Yahr stage, the motor part (III) of the Unified Parkinson's Disease Rating Scale (UPDRS), the body mass index (BMI), dementia, depression, saliva control, dysarthria, and disease duration.[17]

Pathophysiology

The pathophysiology of the preparation and execution of swallowing is complex and has not yet been fully understood. Functional magnetic resonance imaging (fMRI) studies have shown that multiple brain areas are involved during the process of swallowing, such as the lateral pericentral areas, anterior cingulate cortex, insula, thalamus, caudate, putamen, and cerebellum,[18-24] in addition to the brainstem, including the medulla and the central pattern generator.[25-27] In particular, volitional swallow is closely related to the basal ganglia and frontal lobe as well as other cortical structures.[19,21,24] Dysfunctions of the dopaminergic neural network seem to affect the supramedullary swallowing system and cause dysphagia in PD.[16,28,29]

Lewy bodies in nondopaminergic brain areas[30] and alpha-synuclein in peripheral motor and sensory nerves innervating the pharyngeal muscles[31] have also been discussed in relation to dysphagia in patients with PD. Recently, alpha-synuclein pathology was found to cause gastrointestinal dysfunction in colon tissues even before the patients with PD developed their motor symptoms and was discussed as a possible biomarker for premotor PD.[32] A study using manometry also showed esophageal impairment in very early stages of PD.[14] These results suggest that enteric nervous system involvement may precede the progression in the central nervous system in PD and possibly affect the pharyngeal muscles responsible for swallowing as well.[33]

Another pathological factor related to dysphagia in patients with PD is the reduced concentration of substance P, a neuropeptide associated with cough and the swallow reflex, in their sputum. It was lower in advanced stages of PD, causing disturbances of airway protection and silent aspiration.[34,35] A recent study also demonstrated that saliva substance P concentrations were significantly lower in PD patients with pharyngeal dysphagia than in those with a normal pharyngeal swallowing function, suggesting a reduction in substance P levels in the saliva as a possible biomarker for the early detection of pharyngeal dysphasia in patients with PD.[36]

Clinical symptomatology

Dysphagia is one of the crucial nonmotor symptoms of PD that can lead patients to malnutrition, dehydration, and aspiration pneumonia. Patients with PD can show problems in any of three or four stages of swallow: the oral preparatory and transportation stage, the pharyngeal stage, and the esophageal stage (Table 1). In the oral stage of swallow, the typical repetitive backward-forward movement of the bolus is frequently observed in patients with PD. This rocking-rolling festination phenomenon may be related to some rigidity in the muscle responsible for lowering the back of the tongue to allow for the bolus passage to the pharynx.[15] As a result, difficulties in the initiation of swallow, piecemeal swallow and premature falling of the food can occur. Reduced tongue pressure and abnormal patterns of tongue-palatal pressure also contribute to difficulties with bolus management (Supplementary Videos 1-3 in the online-only Data Supplement).[37,38] It can cause severe residue in the oral cavity and may be involved in problems of oral hygiene or medication

Table 1. Characteristics of dysphagia in patients with parkinsonism

Phases of swallow	Frequent findings
Oral phase	Repetitive pumping movement of the tongue
	Rocking-rolling festination movement of the tongue
	Oral residue
	Piecemeal swallow
	Difficulty of bolus formation
	Premature falling
Pharyngeal phase	Reduced tongue retraction and pharyngeal constriction
	Regurgitation of food into the nasal cavity or upper pharynx
	Difficulty in initiating and completing airway closure
	Penetration/aspiration
	Residue in valleculae and pyriform sinuses
	Reduced pharyngeal and laryngeal sensitivity
Esophageal phase	Reduced esophageal peristalsis

intake.

Delays in the swallowing reflex are relatively mild in patients with PD,[15] but reduced tongue base retraction and pharyngeal wall constriction are frequently involved in pharyngeal residue and aspiration during/after swallow when the pharyngeal stage of swallow is triggered. The regurgitation of food to the upper pharynx and the nasal cavity can be observed in these patients. Dysfunctions in vocal cords and insufficient respiratory supports interfere with the ejection of aspirated materials out of the larynx.

In conjunction with problems in oro-pharyngeal dysphagia, salivary pooling[39] and an abnormal breathing-swallowing pattern, which is frequent inhalation instead of exhalation right before and after swallow, may contribute to aspiration in patients with PD.[40] Cognitive dysfunction also appears as the disease progresses. In particular, frontal executive dysfunctions are closely associated with difficulties in the oral stage of swallow, even in patients in the early stage of PD.[41]

Table 2. Clinical scales related to dysphagia for patients with parkinsonism

	Scale
Sialorrhea scale	Drooling severity and frequency scale (DSFS)
	Drooling rating scale
	Sialorrhea clinical scale for PD (SCS-PD)
Dysphagia scale	Swallowing disturbance questionnaire (SDQ)
	Swallowing quality of life (SWAL-QOL)
	Functional dysphagia scale (FDS)
General scale	Scales for outcomes in PD-autonomic (SCOPA-AUT)
	Nonmotor symptoms questionnaire for PD (NMSQuest)
	Nonmotor symptoms scale (NMSS)
	Parkinson's disease questionnaire (PDQ)
	Activities-specific balance confidence (ABC)
	Schwab and England activities of daily living (S-E ADL)
	Northwestern university disability scale (NUDS)
	Stanford self-efficacy for managing chronic disease 6-item scale (SSE)

Adapted from Evatt, et al.[39] PD: Parkinson's disease.

Assessment

The early detection and individualized treatment of dysphagia for each patient with PD is crucial to prevent complications of the disease and improve the quality of life of the patient. A screening test for dysphagia in patients with PD by simply asking them whether they have swallowing problems or may not be sensitive enough due to their poor awareness of the problems and their reduced sense of the oro-pharynx as well as larynx. The task force team of the Movement Disorders Society has assessed clinical rating scales for PD and made recommendations.[39] Through their systematic reviews, two dysphagia scales, the Swallowing Disturbance Questionnaire (SDQ) and the Dysphagia-Specific Quality of Life (SWAL-QOL) scale, met the "suggested" but not the "recommended" criteria. There was no scale for the "recommended" criteria that is proven to be valid, reliable, and sensitive in clinical studies. A recent study showed that the functional dysphagia scale (FDS) and the Schwab and England activities of daily living (S-E ADL) scale can effectively predict the occurrence of aspiration pneumonia in patients with PD.[42] Clinical scales of nonmotor symptoms relating to dysphagia for patients with PD are summarized in Table 2.[39]

Instrumental swallowing studies, such as VFSS or FEES, are essential to patients with suspected problems in the pharyngeal stage of swallow.[15] In particular, the VFSS provides temporal parameters related to both oral and pharyngeal dysphagia, including the etiology of aspiration. The studies using VFSS made it clear that dysphagia began with the reduced tongue retraction and repetitive rocking-rolling movement of the tongue. Additionally, food residue in valleculae and pyriform sinuses and piecemeal swallow were significantly associated with penetration/aspiration in patients with PD.[15,43] The study for developing the PD VFSS scale (PDVFS) showed that problems with mastication, lingual motility prior to transfer, aspiration, and total swallow time were the predictors of aspiration pneumonia.[44]

Management

Treatments of dysphagia for patients with PD include various rehabilitative treatments, compensatory techniques, and medical treatments. Exercises to increase the strength and range of motion of the oropharynx and larynx and to enhance bolus control ability have been frequently used for the rehabilitative treatment of those patients. Not only direct approaches to oro-pharyngeal and laryngeal structures but also strategies such as consolidating the abilities of speech, coordination between breathing and swallowing or airway protection have also shown some improvement in VFSS and SWAL-QOL.[45,46] Studies on the correlation between voice function and dysphagia also demonstrated positive results. Lee Silverman Voice Treatment (LSVT) showed effects on pharyngoesophageal swallow and involuntary cough functions as well as voice in patients with PD.[47-49] The maximum phonation time (MPT) also showed a significant correlation with oro-pharyngeal swallowing functions.[50] Expiratory muscle strength training (EMST) to increase the ability to voluntarily cough to eject foreign material from the airway was also effective in decreasing penetration/aspiration in these patients.[46,51] Considering the function of the vocal cords in the swallowing process, it was suggested that the use of vocal augmentation may be beneficial for these patients with glottal insufficiency.[52]

The effects of surface electrical stimulation (SES) applied to

the neck in patients with PD seem to be unclear. Through the systematic review,[53] three studies on SES were included in their qualitative analysis, and the results showed no treatment effects of SES exceeding those of traditional behavioral treatment[54,55] or a demonstrated placebo effect.[56] Only a recent study on SES applied to the infrahyoid area accompanied with effortful swallow demonstrated effects for increasing hyoid movement and decreasing aspiration in nine patients with PD.[57] Further studies on SES with randomized, double-blind, placebo-controlled trials including a larger population to elucidate the exact mechanism of SES in the neural pathway of swallow are necessary.

As medical treatments for patients with PD, dopaminergic medications such as levodopa have been primarily considered to improve their motor symptoms. However, the effects of levodopa or apomorphine on patient dysphagia remain controversial.[58-62] Some studies have reported significant reductions in oral and pharyngeal delay time for swallow after levodopa or apomorphine.[58,63] A meta-analysis also showed large evidence of levodopa effects on improving swallowing function.[60] On the other hand, other studies showed that there were no or even negative effects of levodopa on swallowing or the coordination between swallowing and respiration.[59,64-66] However, it is hard to deny that the mortality rate in patients with PD has been reduced after the use of levodopa, which may be due to the improvement in dysphagia,[60] which is the leading cause of aspiration pneumonia. Recent studies on non-oral dopaminergic treatment, which can measure dysphagia without the influence of fluctuating drug effects due to dysphagia, have also shown improved swallowing functions in these patients.[29,67] Accumulating evidence to confirm the effects of the new medical treatments should be addressed in future studies.

Deep brain stimulation (DBS) is another option for treating patients with advanced PD who have difficulties taking oral medication. However, the effect of DBS on dysphagia is not yet clear.[68] The positive effects of DBS on some parameters of pharyngeal swallow have been reported, but there was no clinical impact on dysphagia.[69-71] A recent study revealed that a low frequency of 60 Hz DBS of the bilateral STN showed some improvement in the aspiration rate in VFSS compared to the routine frequency of 130 Hz DBS.[72] Disappointingly, however, there was no long-term effect on dysphagia despite persistent benefits on gait or other motor symptoms of patients with PD.[73] Moreover, there are no relevant data for the comparison of STN vs. globus pallidus internus (GPi) or unilateral vs. bilateral STN DBS and the effects on dysphagia. Additionally, the therapeutic effects of botulinum toxin therapy on crico-pharyngeal dysfunction[74] or repetitive transcranial magnetic stimulation (rTMS) on dysphagia in these patients have yet to be investigated.[17]

PARKINSON PLUS SYNDROMES

Dysphagia as well as dysarthria appear earlier in patients with Parkinson plus syndromes than PD overall. However, studies including large subjects and valid instruments for assessing dysphagia in these patients were not many. A postmortem study[3] showed that the median latencies of subjective dysphagia were 42 months, 64 months, 67 months, and 130 months in patients with progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), multiple system atrophy (MSA), and PD, respectively, and that dysphagia or dysarthria that appeared within one year of disease onset was a prominent feature for atypical parkinsonian syndrome with 100% specificity. However, the survival time from the onset of dysphagia was similar (15–24 months) among the disease groups.

Progressive supranuclear palsy

Dysphagia in patients with PSP appeared earlier from the disease onset than other Parkinson plus syndromes or PD.[3] In several studies, however, there was no significant difference in the onset of dysphagia or alternative feeding between PSP and MSA.[75,76]

Two clinical phenotypes of PSP, Richardson syndrome (PSP-RS) and PSP-parkinsonism (PSP-P), have been identified based on early symptoms. The disease progression was also different between the two phenotypes.[75,77] Patients with PSP-RS reached their clinical milestones, including severe dysphagia, frequent falls, cognitive impairments, and gait and speech dysfunctions, earlier than those with PSP-P. The shorter time to the milestones from the disease onset was an unfavorable predictor of survival. A longitudinal study also regarded gait disturbance, dysarthria, and severe dysphagia as the first key motor impairments in patients with PSP, although severe dysphagia was the least frequent as the first sole milestone.[78] Only one patient needed a nasogastric feeding tube without other motor symptoms. The use of feeding tubes in other patients was more closely associated with speech than the gait milestone.

The early development of dysphagia as well as the severity of dysphagia were reported as poor prognostic predictors.[79,80] Swallowing problems that occurred within 2 years were associated with a shorter survival time. The onset of falls, speech problems, diplopia, cognitive dysfunction, and older age at onset were also identified as negative factors for prognosis in these patients.

Although the characteristics of dysphagia in patients with PSP were not significantly different from those with PD, the time of transferring a bolus from the oral cavity to the pharynx or the time of initiating swallow was longer in PSP.[76,81,82] This observation may be caused by pathological changes in the cerebral cortex and brainstem as well as the basal ganglia in PSP. A treatment

for dysphagia specifically developed for patients with PSP is difficult to find.

Multiple system atrophy

Patients with MSA showed various clinical symptoms, mainly including cerebellar dysfunctions, parkinsonism, and autonomic dysfunctions, and were subdivided into MSA-C and MSA-P according to their predominant symptoms. The presence or absence of early autonomic dysfunction has also been identified as an important predictor for the prognosis of MSA. In particular, if dysphagia or gait problems appeared in patients with early autonomic dysfunction, the survival interval was shorter from the onset.[75,83]

Swallowing dysfunctions in MSA were generally similar to those in PD, but different characteristics of dysphagia have been reported in patients with MSA-C and MSA-P.[84-87] The most frequent symptom of dysphagia was aspiration (approximately 90%) in MSA-C, whereas aspiration and difficulty in swallowing were reported to be approximately 58% and 45%, respectively, in MSA-P.[87] A comparison of two groups in VFSS revealed that apraxia of swallow and residue in valleculae were more frequent in MSA-P. Another study using VFSS showed that cerebellar dysfunction as well as parkinsonism affect the muscle coordination for managing a bolus in the oral phase of swallow from the early stage of MSA-C.[86] On the other hand, in MSA-P and PSP, incomplete relaxation of the crico-pharyngeal muscle was found on electromyography even in the early stage of the disease.[84] Crico-pharyngeal dysfunction has also been noticed on the manometry of 60% of patients with more than 5 years since the onset of MSA.[85] It may be associated with neuronal loss of the nucleus ambiguous in these patients.[88]

The effects of levodopa were mild and limited in MSA as well as PSP, although it was more beneficial for patients with MSA than PSP when levodopa was started.[75] Furthermore, the effects of levodopa on dysphagia are controversial. Surgical procedures of laryngeal closure following tracheostomy can be considered a viable treatment option for patients with vocal cord abductor paralysis and dysphagia due to MSA.[89]

Corticobasal degeneration

Clinical studies addressing dysphagia in patients with CBD are few. A previous study reported that the prevalence of dysphagia in these patients was as low as 13%.[90] The occurrence of dysphagia in a postmortem study was 31% after a median disease duration of 64 months in patients with CBD.[3] However, in a study using VFSS, 86% of the patients showed dysphagia, and their mean disease duration was approximately 55 months.[91] A recent study using the National Institutes of Health (NIH)-Speech Pathology swallowing questionnaire, ultrasound, and VFSS[92] in patients with a mean disease duration of 46 months showed that 96% had complaints of swallow, 61% showed a longer duration of oro-pharyngeal swallow on ultrasound examination, and 27% showed abnormal swallow on VFSS. It increased up to 100% when the cases of mild to moderate dysphagia in VFSS were included.

The most common features of dysphagia in patients with CBD were piecemeal swallow in the oral phase and residue pooling in valleculae in the pharyngeal phase of swallow,[91,92] whereas a delay of swallow initiation was the most common in patients with PSP syndrome.[81] The involvement of the neural pathways from the anterolateral cortex and subcortical structures to the brainstem may affect the swallowing function in these patients.[91] Symptomatic treatments, including various rehabilitative treatments and compensatory techniques, can be applied in patients with CBD as well as other parkinsonism.

DYSTONIA

Focal dystonia, especially involving the orofacial and cervical areas, can cause dysphagia. The performances of the patients vary across tasks, but the symptoms are usually relieved by sensory tricks.[93] The underlying etiology of idiopathic dystonia is not yet clearly known. Pathological changes in the brainstem, basal ganglia, and cerebellum were reported in patients with dystonia.[94] Rarely, dystonic storms, which are fast and furious aggravations of dystonia involving tachycardia, tachypnea, hypertension, and autonomic instability, can also accompany the rapid progress of bulbar dysfunction, such as dysarthria, dysphagia, and respiratory failure.[95] It usually occurs in patients with dystonia due to known hereditary causes such as Wilson's disease, DYT1 dystonia, or cerebral palsy, but the mechanism is not clearly understood.

Oromandibular dystonia

Oromandibular dystonia (OMD) is a focal dystonia characterized by sustained involuntary movement in the masticatory, lower facial, labial, and lingual muscles.[96] The etiology of OMD is poorly understood, although peripheral mechanisms and abnormal sensorimotor integration or somatosensory dysfunctions were suggested to occur and aggravate OMD.[97] Patients with OMD usually demonstrate dysphagia as well as dysarthria, particularly difficulties in the oral phase of swallow.[98,99] They showed difficulties in chewing solid food and in forming and transferring a bolus into the pharynx (Supplementary Video 4 in the online-only Data Supplement). However, the characteristics of dysphagia were different depending on the affected muscles of each patient.[99,100]

OMD is often associated with blepharospasm (Meige syn-

drome) or other types of dystonia, and dysphagia is more frequent in these patients than pure OMD. Nine (64%) out of 14 patients with OMD reported dysphagia, and 5 of the patients had OMD with cervical dystonia.[99] A study using VFSS in patients with blepharospasm and Meige syndrome showed that 18 (90%) out of 20 patients presented dysphagia.[101] The most frequent abnormal findings were premature falling of food and residue in pharyngeal spaces.

Botulinum toxin injection has been considered for treating patients with OMD. However, injection into the tongue muscle can aggravate dysphagia. The effects of the drug were not consistent.[102,103]

Cervical dystonia

Cervical dystonia characterized by sustained involuntary neck muscle contraction can affect swallowing functions mainly due to the abnormal head and neck postures.[103] A delayed swallowing reflex and reduced tongue base retraction were the main findings in these patients on VFSS. Not only cervical dystonia itself but the medical treatments applied for cervical dystonia, such as botulinum toxin injection or surgical procedures, can aggravate the dysphagia of the patients. Ultrasound with electromyography-guided injection of botulinum toxin injection or surgery for selective peripheral denervation have been recommended for better outcomes in reducing the incidence of dysphagia.[104-106]

Lingual protrusion dystonia

Lingual protrusion dystonia (LPD) often appears with OMD but can occur in isolation. Tardive dystonia, which can be observed as a side effect of neuroleptic medication, is the most common form (41%) of LPD, followed by idiopathic causes (29%).[107] It interferes with speaking and swallowing as well. A cessation of or a change in neuroleptic medication should be the first consideration for tardive dystonia. For the treatments for other LPD, if symptomatic pharmacological treatment is unresponsive, botulinum toxin injection in the genioglossus muscles may be effective. However, it can also cause severe dysphagia and breathing difficulties as well.[107,108]

Task-specific dystonia

Task-specific dystonia (TSD) is a focal dystonia of a body part that is associated with a specific activity. For example, focal dystonia of the arms and hands or oromandibular muscles was observed most frequently in players of acoustic guitar or brass instruments during their performance, which is often called musician's dystonia.[109] Isolated lingual dystonia induced by speaking[110] or OMD induced by praying[111] are also found in TSD. Since dystonia usually does not occur during other tasks, dysphagia in TSD is less frequent even in patients whose oromandibular muscles are affected. However, the symptoms can progress and involve swallowing functions over time.[109]

On the other hand, dystonia that occurs exclusively during eating (eating dystonia) has been reported.[4,5] The cases were all related to neuroleptic medications. Transfer dysphagia has also been reported as a focal dystonia, which is the difficulty of transferring a bolus from the posterior oral cavity to the pharynx.[6] However, the symptom may not be clearly distinguished from the difficulty of the initiation of swallow or the repetitive pumping motion of the tongue in patients with parkinsonism. Although the authors argued that there was no effect of antidepressants and the symptom was improved by approximately 50−70% with trihexyphenidyl/tetrabenazine, the possibility of functional dysphagia cannot be ruled out completely as the authors mentioned.

OTHER DISORDERS

Palatal myoclonus, the rapid rhythmic spasm of velopharynx, may extend to the pharyngeal and laryngeal muscles. In these cases, a disruption of bolus transportation in the oral phase and difficulty initiating swallowing and airway protection can be presented.[93] Other involuntary movements involving oral, pharyngeal, and laryngeal muscles, such as spasmodic dysphonia, essential voice tremor or tongue tremor, can also be associated with dysphagia depending on the level and extension of the abnormal movements.[93,112-114]

CONCLUSION

Dysphagia is very common and a major clinical concern in patients with PD from the early stage of the disease. Poor self-awareness of the symptoms may hamper early detection and proper management in these patients.[1,2,10-12] Therefore, early intervention employing validated screening tools and instruments would be recommended from the initial stage of the disease to improve the quality of life and mortality rate of the patients. Diagnosis and intervention for dysphagia are very complex and require multidisciplinary collaborations for high-quality clinical practice.

The pathophysiology of dysphagia in PD has not yet been clearly identified. The effects of medical treatments on dysphagia, such as dopaminergic medication or DBS, are also controversial. Further research is needed to clarify the underlying mechanism of dysphagia and the treatment effects using randomized, double-blinded, placebo-controlled trials. Individualized rehabilitation programs for each patient should also be developed based on the findings of an objective assessment such as VFSS.

In patients with Parkinson plus syndrome, the early development of dysphagia might be a prominent feature for a differential diagnosis from PD.[3] It was also associated with the poor prognosis of the disease. The characteristics of dysphagia or survival after the onset of dysphagia were not much different among those patients with parkinsonian disorders. Focal dystonia or other movement disorders involving the oral, pharyngeal, and laryngeal muscles can cause various forms of dysphagia depending on the affected structures. For these patients, oral medication may be considered prior to botulinum toxin injection or surgery, which might cause or aggravate dysphagia or breathing difficulties.[107,108] Future research for optimal treatment for these patients depending on the nature of their symptoms is warranted.

Supplementary Video Legends

Video 1. Videofluoroscopic image of a 78-year-old man showing normal oro-pharyngeal stages of swallow.

Video 2. Videofluoroscopic image of a 73-year-old man with Parkinson's disease showing the repetitive pumping movement of the tongue.

Video 3. Videofluoroscopic image of a 74-year-old man with Parkinson's disease (S/P bilateral STN DBS) showing the repetitive pumping movement of the tongue followed by the rocking-rolling festination movement of the tongue.

Video 4. Videofluoroscopic image of a 70-year-old man with oro-mandibular dystonia showing involuntary movements during rest, followed by difficulties in triggering the swallowing reflex, and the effects of a sensory trick with holding an object in the mouth finally.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.14802/jmd.19048.

Conflicts of Interest

The authors have no financial conflicts of interest.

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