Journal of the Korean Dysphagia Society 2022:12:105-114 https://doi.org/10.34160/jkds.2022.12.2.004 Original Article

Differences in Dysphagia between Patients with Stroke or Amyotrophic Lateral Sclerosis

Jae Young Lee, M.D.[1], Yu Chan Park, M.D.[1], Jungsoo Lee, M.D.[2], Jinseok Park, M.D., Ph.D.[2], Ki Wook Oh, M.D., Ph.D.[2], Seung Hyun Kim, M.D., Ph.D.[2], Mi Jung Kim, M.D., Ph.D.[1]

[1]Department of Rehabilitation Medicine, Hanyang University College of Medicine, Seoul, [2]Department of Neurology, Hanyang University College of Medicine, Seoul, Korea

Objective: Dysphagia is a common symptom of stroke and affects 23-50% of such patients. In addition, bulbar involvement, which causes dysphagia, is the primary initial symptom in approximately 25-30% of amyotrophic lateral sclerosis (ALS) patients. The purpose of this study was to compare patterns of swallowing difficulties in stroke and ALS patients.

Methods: We retrospectively recruited 84 ALS patients with dysphagia and 294 stroke patients with dysphagia between January 2017 and December 2019. Swallowing processes were reviewed by videofluoroscopic swallowing studies (VFSSs). The presence of oral residues and oral transit times (OTTs) were measured in the oral phase, and the presence of penetration and aspiration and residues in valleculae or pyriform sinuses were evaluated. Statistical analysis was performed using SPSS 25.0 and comparisons using the Chi-square test.

Results: ALS patients more frequently had delayed OTTs and oral residues than stroke patients, and stroke patients more frequently experienced aspiration and had delayed thin liquid pharyngeal transit times (PTTs). However, no significant intergroup difference was observed for the presence of penetration, residues in valleculae or pyriform sinuses, or thick liquid PTTs.

Conclusion: The study shows that ALS patients exhibit slower food processing in the oral cavity and more significant bulbar muscle weakness than stroke patients. On the other hand, stroke patients had greater thin liquid aspiration rates than ALS patients. These findings should be considered when choosing treatments for ALS and stroke. **(JKDS 2022;12:105-114)**

Keywords: Stroke, Amyotrophic lateral sclerosis (ALS), Dysphagia

INTRODUCTION

Dysphagia is a common symptom of stroke, affecting 23-50% of stroke patients[1]. In addition, dysphagia after stroke increases the risk of pulmonary infection, with a threefold increased risk of patient mortality[2-4]. Dysphagia after stroke has a sevenfold increased risk of aspiration pneumonia and can be an independent predictor of mortality[1]. Swallowing problems in stroke patients are due to lesions affecting the corticobulbar tracts or ventral bulbar region. Furthermore, some evidence indicates that the swallowing center exists in

Received: April 18 2022, Revised: April 20 2022, Accepted: July 12 2022
Corresponding author: Mi Jung Kim, Department of Rehabilitation Medicine, Hanyang University College of Medicine, 222-1 Wangsimni-ro, Seongdong-gu, Seoul 04763, Korea
Tel: +82-2-2290-9353, Fax: +82-2-2282-0772, E-mail: kimmjreh@hanyang.ac.kr

the medullary reticular formation, and the association with brainstem diseases causes dysmotility in the pharyngeal phase of swallowing[5,6].

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by progressive loss of upper and lower motor neurons in the brainstem, spinal cord, and motor cortex[7,8]. Initial symptoms of ALS include muscle weakness, muscle stiffness, and dysarthria to dysphagia. When symptoms begin in the arms or legs, it is defined as "spinal onset" ALS, and when patients first notice dysarthria or dysphagia, it is defined as "bulbar onset" ALS. Dysphagia is a common symptom in ALS patients and is caused by involvement of bulbar motor neurons, especially in bulbar onset ALS patients. Bulbar involvement, which causes dysarthria, dysphonia, and dysphagia, is the primary initial symptom in approximately 25-30% of ALS patients. Also, nearly 80% of patients show bulbar involvement in later stage of ALS, regardless of type[9]. Dysphagia appears when the progressive loss of motor neurons exceeds their capacity to relieve the swallowing symptoms and refers to involvement of motor neurons in the brainstem that innervates the muscles of the face, tongue, pharynx, and larynx[10]. Dysphagia in ALS patients increases the risk of respiratory failure, aspiration pneumonia, dehydration, and mortality[11]. However, this symptom can be underestimated due to progressive adaptation to the slow deterioration of bulbar function[12]. Early recognition of dysphagia is important for administering adequate therapy, and a therapeutic target is important for effective treatment.

In several previous studies, the characteristics of dysphagia in stroke patients were associated with the location of the stroke lesion. Swallowing problems are common in middle cerebral artery (MCA) strokes as well as brainstem strokes[13,14]. In addition, left cortical stroke tends to be associated with oral dysfunction and right cortical stroke with pharyngeal dysfunction[15]. Kim et al.[16] compared territorial anterior infarcts (TAIs), territorial posterior infarctions (TPIs), and white matter disease (WMD) and found that TAIs are more relevant to oral phase dysfunction, while TPIs are associated with pharyngeal dysfunction.

Furthermore, previous research has been conducted regarding dysphagia in ALS patients. Solazzo et al.[12] revealed characteristics of swallowing problems in ALS patients using videofluoroscopic swallowing study (VFSS), which showed increase in pharyngeal contraction time and residual pressure after relaxation of the upper esophageal sphincter (UES). In addition, a study using VFSS revealed that bulbar onset ALS patients showed more significant dysfunction than spinal onset ALS in the oral phase[17].

As mentioned above, the relationships between stroke lesions and characteristics of dysphagia have been reported. However, to the best of our knowledge, characteristics of swallowing problems have not been compared between stroke and ALS patients. If the characteristics of dysphagia are dissimilar in the two patient groups, the treatment strategy should be different.

Accordingly, in the present study, differences of dysphagia characteristics in the oral phase and pharyngeal phase between stroke and ALS patients were identified using VFSS. Furthermore, brainstem stroke and ALS patients were compared to discover dysphagia characteristics due to the association of brainstem diseases.

In addition, because there were the differences in dysphagia for each type of stroke and ALS, we investigated the characteristics of each type of ALS (bulbar/spinal onset ALS) and compared them by type of stroke (brainstem lesion/other brain area lesion)[16,17].

MATERIALS AND METHODS

1. Subjects

The electronic medical records of VFSS examinations were reviewed retrospectively. We retrospectively searched for patients complaining of swallowing difficulties due to stroke or ALS between January 2017 and December 2019. The study inclusion criteria were (1) first-ever ischemic stroke confirmed using magnetic resonance imaging (MRI) or hemorrhagic stroke

confirmed using computed tomography (CT), (2) swallowing difficulty and/or signs of aspiration after stroke onset, (3) evaluated using VFSS, and (4) >20 years of age at VFSS examination. Exclusion criteria for stroke patients were (1) poor head and neck control, (2) recurrent infarction or hemorrhage, (3) poor cognitive status causing inability to obey commands for the VFSS, and (4) medical illness due to aspiration pneumonia.

In addition, the study inclusion criteria for ALS patients were (1) definite or probable ALS according to the El Escorial World Federation of Neurology criteria, presenting with swallowing difficulty and/or signs of aspiration at time of first diagnosis, (2) evaluated using VFSS, and (3) >20 years of age at VFSS examination. Exclusion criteria for ALS patients included poor head and neck control and medical illness due to aspiration pneumonia.

Between January 2017 and December 2019, 294 stroke patients and 84 ALS patients were identified for this study. Approval was obtained from the Institutional Review Board of Hanyang University Hospital (approval No. 2020-07-039-004).

2. VFSS

VFSS was performed using the modified Logemann protocol[18]. Patients were seated, fluoroscopic video were recorded in the lateral projection, and images were stored. For screening the presence of liquid aspiration, 2 ml of thin liquid containing barium solution was swallowed. If there was massive aspiration of the thin liquid, the evaluation was stopped. If aspiration of thin liquid was not massive or no aspiration occurred, further evaluation was continued with 5 ml each of thin liquid and thick liquid.

Two physiatrists analyzed the swallowing process in the oral and pharyngeal phases. In the oral phase, presence of oral residue and oral transit time (OTT) were evaluated. OTT is defined as the time from onset of bolus movement in the mouth until the bolus reaches the point at which the lower rim of the mandible passes the tongue base. Oral residue was defined as greater than 10% of the bolus remaining in the oral cavity[19]. More than 1.5 seconds of OTT was defined as delayed oral phase[20]. In the pharyngeal phase, the presence of residue in the valleculae and pyriform sinus, presence of penetration and aspiration, and pharyngeal transit time (PTT) were assessed. Residues in the valleculae and pyriformis sinus was measured as the ratio of residue area divided by the area of the valleculae and pyriformis sinus, respectively[21]. The presence of residues in valleculae and pyriform sinus was defined as when the residues were greater than 10%[19]. PTT is defined as the time from the onset of pharyngeal entry, where the leading edge of the bolus has reached the anterior border of the vertical ramus of the mandible to the end of the pharyngeal exit where the trailing edge of the bolus had passed the superior border of the UES[22].

3. Statistical analysis

Statistical analysis was performed using SPSS 25.0 for Windows version 25.0 (IBM, Armonk, NY, USA). This study analyzed differences in dysphagia characteristics between stroke and ALS patients. The Chi-square test was used to compare the contents of oral and pharyngeal phases between the two groups. Statistical significance was set at P=0.05.

RESULTS

The study included 294 stroke patients and 84 ALS patients. The age of stroke patients ranged from 20-94 years, with a mean age of 66.4 years. Mean duration of disease was 100.6 days. The stroke group included 69 (29.4%) patients with tube feeding and 225 (70.6%) patients with oral feeding. The stroke group included 122 (41.5%) patients with ischemic stroke and 172 (58.5%) with hemorrhagic stroke. In addition, this group was classified based on the location of the stroke lesion (brainstem involved or not); 34 (11.5%) patients had brainstem involvement. The age of ALS patients ranged from 23-79 years, with a mean age of 59.1 years. Mean duration of disease was 1168.5 days. The ALS group included 10 (11.9%) patients with tube feeding and 74 (88.1%)

patients with oral feeding. The ALS group included 31 (36.9%) patients with bulbar-type ALS and 53 (63.1%) with spinal-type ALS.(Table 1)

In stroke patients, 294 ingested the 2 ml of thin liquid. However, 30 patients showed massive aspiration of 2 ml of thin liquid and could not be evaluated using thick liquid. However, all 84 ALS patients could be examined using thick liquid without massive aspiration of thin liquid.

1. Differences in the oral phase of the swallowing process

Among the 294 stroke patients, 80 (27.2%) showed delayed OTT. Among the 84 ALS patients, 33 (39.2%) showed delayed OTT. More frequent delay of OTT was observed in the ALS group (P=0.033).

In addition, excessive oral residue was observed most frequently in the ALS group (P=0.027). Among 294 stroke patients, 206 (70.0%) showed the presence of oral residue. Among the 84 ALS patients, 69 (82.1%) showed the presence of oral residue.(Table 2) However, when comparing brainstem stroke and ALS patients, differences were not prominent in OTT (P=0.188) and oral residue (P=0.292).(Table 3) In addition, when comparing each type of stroke (brainstem lesion/other brain area lesion) with each type of ALS (bulbar/spinal onset ALS), more prominent oral phase delay was noted in bulbar ALS than in stroke of brainstem lesion and stroke of other brain area lesion (P<0.01).(Table 3)

Table 1. Demographic and clinical characteristics of study participants.

Variable	Stroke (n=294)	ALS (n=84)
Age (mean±SD), y	66.4±14.72	59.1±12.17
Range, y	20-94	23-79
Sex (m/f), n	166/128	35/49
Type of feeding (tube/oral), n	69/225	10/74
Duration of disease (mean), days	100.6	1168.5
Stroke type (ischemic/hemorrhagic), n	121/173	
Site of stroke (brainstem/non-brainstem), n	34/260	
ALS type (bulbar/spinal), n		31/53

SD: standard deviation, ALS: amyotrophic lateral sclerosis.

These findings are suggestive of more frequent dysfunction of the oral phase in ALS patients than stroke patients; however, brainstem stroke patients had similar characteristics to ALS patients in the oral phase. Considering subgroup analysis, bulbar ALS had more prominent oral phase dysfunction compared to each type of stroke.

2. Differences in the pharyngeal phase of the swallowing process

Significant difference was not observed in valleculae residue (P=0.445) or pyriform sinus residue (P=0.171) between the two groups. However, more frequent delay of PTT existed in the stroke group, especially for swallowing of thin liquid (P<0.01). Among the 294 stroke patients, 96 (32.7%) showed delayed PTT with thin liquid. Among the 84 ALS patients, 10 (11.9%) showed delayed PTT with thin liquid.

Regarding PTT with thick liquid, statistically significant difference was not observed between the two groups. However, stroke patients tended to have delayed PTT with thick liquid (P=0.096).(Table 4)

Similar findings were observed in brainstem stroke and ALS patients. Statistically significant difference was not observed between the two groups regarding valleculae residue (P=0.389), pyriform sinus residue (P=0.291), and delayed PTT with thick liquid (P=0.087). However, more frequent delay of PTT with thin liquid was observed in patients with brainstem stroke (P<0.01).(Table 5) In addition, when comparing each type of stroke (brainstem lesion/other brain area lesion) with each type of ALS (bulbar/spinal onset ALS), more

Table 2. Oral phase dysfunctions in each group.

	Stroke (n=294)	ALS (n=84)	P-value
OTT			0.033*
Intact	214 (72.8%)	51 (60.8%)	
Delayed	80 (27.2%)	33 (39.2%)	
Oral residue			0.027*
None	88 (30.0%)	15 (17.9%)	
Present	206 (70.0%)	69 (82.1%)	

*P-value<0.05.
OTT: oral transit time, ALS: amyotrophic lateral sclerosis.

Table 3. Oral phase dysfunctions in subgroups.

	Stroke (n=294)		All ALS (n=84)	Bulbar ALS (n=31)	Spinal ALS (n=53)
	Brainstem lesion (n=34)	Other brain area lesion (n=260)			
OTT					
Intact	25 (73.5%)†	189 (72.7%)†	51 (60.8%)	14 (45.2%)	37 (70.0%)
Delayed	9 (26.5%)†	71 (27.3%)†	33 (39.2%)	17 (54.8%)	16 (30.0%)
Oral residue					
None	9 (26.5%)†	79 (30.4%)†	15 (17.9%)	1 (3.3%)	14 (26.4%)
Present	25 (73.5%)†	181 (69.6%)†	69 (82.1%)	30 (96.7%)	39 (73.6%)

†Significant difference compared to bulbar ALS group ($P<0.05$).
ALS: amyotrophic lateral sclerosis, OTT: oral transit time.

Table 4. Pharyngeal phase dysfunctions in each group.

	Stroke (n=294)	ALS (n=84)	P-value
PTT with thin liquid			<0.01**
Intact	198 (67.3%)	74 (88.0%)	
Delayed	96 (32.7%)	10 (12.0%)	
PTT with thick liquid			0.096
Intact	204 (77.3%)	72 (85.7%)	
Delayed	60 (22.7%)	12 (14.3%)	
Vallecula residue			0.445
None	62 (21.1%)	21 (25.0%)	
Present	232 (78.9%)	63 (75.0%)	
Pyriform sinus residue			0.171
None	102 (34.7%)	36 (42.9%)	
Present	192 (65.3%)	48 (57.1%)	

**P-value<0.01.
ALS: amyotrophic lateral sclerosis, PTT: pharyngeal transit time.

delayed PTT with thin liquid was observed in brainstem stroke compared to bulbar ALS (P=0.03). Each type of stroke (brainstem lesion/other brain area lesion) had more prominent delayed PTT with thin liquid compared to spinal ALS (P<0.01 and P<0.01) and PTT with thick liquid compared to spinal ALS (P<0.01 and P=0.038). In addition, more prominent pyriform sinus residue was shown in stroke of other brain area lesion compared to spinal ALS (P=0.029).(Table 5)

3. Penetration and aspiration rates

The frequency of penetration with thin (P=0.274) and thick liquid (P=0.243) and aspiration with thick liquid (P=0.311) were not significantly different between the two groups. However, stroke patients showed more frequent aspiration with thin liquid compared with ALS patients (P=0.026).(Table 6)

However, comparing brainstem stroke and ALS patients, significant difference was not observed even in aspiration with thin liquid (P=0.073). In addition, each type of stroke had more prominent aspiration of thin liquid (P=0.023, P=0.015) and aspiration of thick liquid compared to spinal ALS (P=0.023, P=0.018).(Table 7)

DISCUSSION

The characteristics and mechanisms of swallowing problems in stroke and ALS patients have been identified in many studies. However, to the best of our knowledge, the present study is the first in which the swallowing problem characteristics were compared in stroke and ALS patients. The results of this study showed that ALS patients had more frequent oral residue and delayed OTT, indicating more frequent dysfunction of the oral phase in ALS patients. Stroke patients had more frequent aspiration with thin liquid and tended to have delayed PTT with thick liquid. Fig. 1 depicts representative examples of an ALS patient who has oral residue and delayed OTT and a stroke patient who had thin liquid aspiration. However, significant difference was not observed in vallecula residue or pyriform sinus residue. These findings show that ALS patients had delayed food processing, especially in the oral cavity, and had more significant bulbar muscle weakness compared with stroke patients. In addition, stroke patients had more severe pharyngeal dysfunction, indicating

Table 5. Pharyngeal phase dysfunctions in subgroups.

	Stroke (n=294)		All ALS (n=84)	Bulbar ALS (n=31)	Spinal ALS (n=53)
	Brainstem lesion (n=34)	Other brain area lesion (n=260)			
PTT with thin liquid					
Intact	19 (55.9%)†,†††,††††	179 (68.8%)††	74 (88.0%)	25 (80.6%)	49 (92.4%)
Delayed	15 (44.1%)†,†††,††††	81 (31.2%)††	10 (12.0%)	6 (19.4%)	4 (7.6%)
PTT with thick liquid					
Intact	19 (70.4%)††	185 (78.1%)††	72 (85.7%)	24 (77.4%)	48 (90.6%)
Delayed	8 (29.6%)††	52 (21.9%)††	12 (14.3%)	7 (23.6%)	5 (9.4%)
Vallecula residue					
None	6 (17.6%)	56 (21.5%)	21 (25.0%)	3 (9.7%)	18 (34.0%)
Present	28 (82.4%)	204 (78.5%)	63 (75.0%)	28 (90.3%)	35 (66.0%)
Pyriform sinus residue					
None	11 (32.4%)	91 (35.0%)††	36 (42.9%)	9 (29.0%)	27 (51.0%)
Present	23 (67.6%)	169 (65.0%)††	48 (57.1%)	22 (71.0%)	26 (49.0%)

†Significant difference compared to bulbar ALS group ($P<0.05$).
††Significant difference compared to spinal ALS group ($P<0.05$).
†††Significant difference compared to all ALS group ($P<0.05$).
ALS: amyotrophic lateral sclerosis, PTT: pharyngeal transit time.

Table 6. Presence of penetration and aspiration.

	Stroke (n=294)	ALS (n=84)	P-value
Penetration (thin liquid)			0.274
None	67 (22.8%)	24 (28.6%)	
Present	227 (77.2%)	60 (71.4%)	
Penetration (thick liquid)			0.243
None	145 (54.9%)	40 (47.6%)	
Present	119 (45.1%)	44 (52.4%)	
Aspiration (thin liquid)			0.026*
None	138 (46.9%)	51 (60.7%)	
Present	156 (53.1%)	33 (39.3%)	
Aspiration (thick liquid)			0.311
None	203 (76.9%)	69 (82.1%)	
Present	61 (23.1%)	15 (17.9%)	

*P-value<0.05.
ALS: amyotrophic lateral sclerosis.

greater pharyngeal muscle weakness.

Furthermore, the differences of swallowing characteristics between brainstem stroke and ALS patients were analyzed because the brainstem is the swallowing center, especially the medullary reticular formation, and is associated with dysmotility in the pharynx. The dysfunctions of the oral phase were similar between brainstem stroke and ALS subjects. However, more prominent dysfunctions of the pharyngeal phase were noted in brainstem stroke. These findings revealed that brainstem stroke had more prominent oral phase dysfunction than stroke of other brain areas. A study revealed that the trigeminal nucleus and reticular formation in the brainstem contain the oral phase pattern-generating neural circuitry[23]. On the basis of the above study, we posited that oral phase dysfunction was more common in brainstem stroke patients, similar to ALS patients, than other patients experiencing stroke in other brain areas.

In addition, we compared each type of stroke (brainstem lesion/other brain area lesion) with each type of ALS (bulbar/spinal onset ALS) because the characteristics of dysphagia vary by location of stroke lesion and type of ALS[16,17]. In subgroup analysis, bulbar ALS had more prominent oral phase dysfunction compared to each type of stroke. However, each type of stroke had more prominent pharyngeal dysfunction compared to each type of ALS, particularly in spinal ALS patients. Therefore, this study revealed that ALS patients had more prominent oral phase dysfunction, particularly in bulbar ALS patients, than stroke patients. Also, stroke patients had more prominent pharyngeal phase dysfunctions than ALS patients, especially than spinal ALS patients.

Several studies have identified the mechanism of dysphagia in stroke patients. A study revealed that

Table 7. Presence of penetration and aspiration in subgroups.

	Stroke (n=294)		All ALS (n=84)	Bulbar ALS (n=31)	Spinal ALS (n=53)
	Brainstem lesion (n=34)	Other brain area lesion (n=260)			
Penetration (thin liquid)					
None	7 (20.6%)	60 (23.1%)	24 (28.6%)	9 (29.0%)	15 (28.3%)
Present	27 (79.4%)	200 (76.9%)	60 (71.4%)	22 (71.0%)	38 (71.7%)
Penetration (thick liquid)					
None	14 (51.9%)	131 (55.2%)	40 (47.6%)	14 (45.2%)	26 (49.1%)
Present	13 (48.1%)	106 (44.8%)	44 (52.4%)	17 (54.8%)	27 (50.9%)
Aspiration (thin liquid)					
None	14 (41.1%)††	124 (47.7%)††	51 (60.7%)	16 (51.6%)	35 (66.0%)
Present	20 (58.9%)††	136 (52.3%)††	33 (39.3%)	15 (48.4%)	18 (34.0%)
Aspiration (thick liquid)					
None	19 (70.3%)††	184 (77.6%)††	69 (82.1%)	21 (67.7%)	48 (90.6%)
Present	8 (29.7%)††	53 (22.4%)††	15 (17.9%)	10 (32.3%)	5 (9.4%)

†† Significant difference compared to spinal ALS group ($P<0.05$).
ALS: amyotrophic lateral sclerosis.

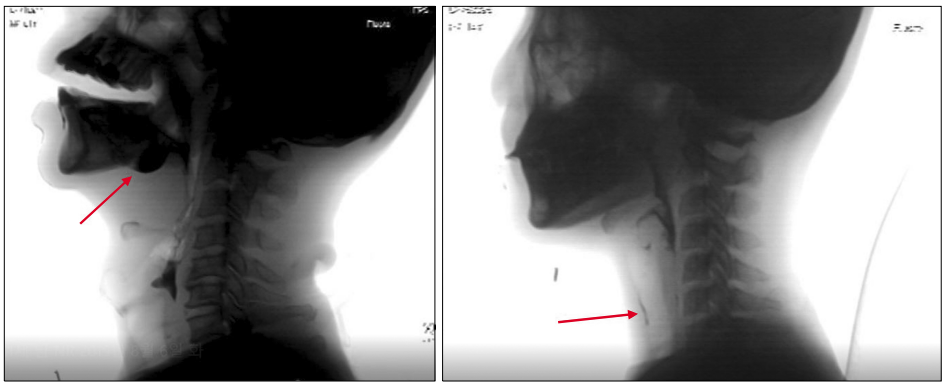

Fig. 1. A representative example of VFSS findings of ALS and stroke patients. The ALS patient (left side) has oral residue and delayed OTT. The stroke patient (right side) has aspiration of thin liquid.

cerebral lesions interrupt bolus movement and voluntary control of mastication during the oral phase, and cortical lesions involving the precentral gyrus can produce impairment in lip, facial, tongue, and pharyngeal muscles. Daniels et al.[24] used the anatomic model of swallowing function and found that the neural mechanisms that mediate lingual coordination might be independent of the neural systems that mediate buccofacial, limb, and speech functions. In addition, they showed the anterior insula to be connected to the primary and supplementary motor cortices, the ventroposterior medial nucleus of the thalamus, and to the nucleus tracus solitaries, and a lesion in the anterior insula can disrupt these connections and produce dysphagia[24]. In addition, the characteristics of dysphagia in stroke patients were different depending on the location of the stroke lesion. Kim et al.[16] compared the anterior infarction group, posterior infarction group, and white matter disease group and showed that the posterior infarction group was associated more highly with pharyngeal dysfunction. Considering the results of above studies, dysphagia of stroke patients can be considered as a result of various brain structure lesions.

In addition, the characteristics and mechanisms of dysphagia in ALS patients have been reported. Solazzo et al.[12] found that increased pressure of the UES is a characteristic of swallowing problems in ALS patients. Also, dysphagia of ALS is the result of mixed type because it involves both the central motor neuron and the second motor neuron located in the

motor nuclei of the brainstem. Also, hyposthenia can be present with atrophy of the tongue and facial muscles, particularly in bulbar onset ALS patients[25]. We thought this is why ALS patients in this study have more severe oral phase dysfunction than stroke patients. Especially in bulbar ALS patients, the weakness of the tongue and facial muscles can be initial symptoms and lead to the oral phase dysfunction, and nearly 80% of patients show bulbar involvement in later stage of ALS, regardless of type[9]. As mentioned above, the characteristics and mechanisms of dysphagia in each stroke and ALS have been studied, but no study has compared the characteristics of swallowing difficulties in stroke and ALS patients.

Treatments for dysphagia have been described in several studies and include texture-modified diets, medications, non-oral feeding, general dysphagia therapy, physical and olfactory stimulation, postural change, enhancing preswallow sensory input, voluntary swallow maneuvers, and exercise[26,27]. Furthermore, surface electromyographic feedback can be used for treatment of stroke patients with chronic dysphagia. In a previous study, patients suffering from dysphagia after stroke were treated for swallowing problems with surface electromyography as biofeedback with normal exercise and experienced superior results to standard therapy for swallowing disorders[28]. For ALS patients, the goals for treating swallowing problems are to maximize function and safety through the use of compensatory strategies, energy conservation, and patient and caregiver education and counseling[29].

Although treatment of dysphagia should be based on the individualized pattern of dysphagia, physicians can focus on the oromotor process in ALS patients and the pharyngeal process in stroke patients, considering the results of this study. If these treatment methods are established, rehabilitation strategies and other treatment methods can be more effective. Based on the present research findings, further studies are necessary to determine how to focus treatment of dysphagia of ALS and stroke patients.

1. Study limitations

The present study had several limitations. First, the sample sizes of the groups differed due to the different prevalence rates of the diseases. In addition, this was a retrospective study based on electronic medical records. If further prospective studies can be conducted, the groups can be paired at a 1:1 ratio, and comparison of the groups can be more meaningful. However, compared with other studies for dysphagia, the sample size in the present study was large. In particular, ALS is a rare motor neuron disease, and our sample size in the ALS group was particularly large compared with other studies. Second, stroke patients were not classified based on affected site, with the exception of brainstem involvement. In some studies, the characteristics of dysphagia were reportedly different based on lesion site[16,30]. In further studies in which stroke and ALS are compared, classification of stroke type (ischemic, stroke), and stroke lesion site (anterior, posterior, left, and right) should be performed. Third, we could not apply the disease severity scale of ALS in this study. The characteristics of dysphagia in ALS patients are heterogeneous according to disease severity. However, we used the Revised ALS Functional Rating Scale (ALSFRS-R) only in outpatients for identifying the indications of hospitalization.

CONCLUSION

Dysphagia is a common symptom in stroke and ALS patients, and treatment is important because dysphagia has a significant impact on nutrition, other medical conditions, and quality of life. In the present study, dysphagia in stroke and ALS patients was different. Stroke patients had more frequent pharyngeal dysfunction, while ALS patients had more frequent oral dysfunction, especially in bulbar ALS patients. Because pathophysiology differs by disease, these findings were expected. Thus, in treatment of dysphagia in stroke or ALS patients, these differences should be determined along with the rehabilitation

method of focus by patient group.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

ACKNOWLEDGEMENTS

There are no financial conflicts of interest to disclose.

AUTHORS' CONTRIBUTIONS

Conceptualization: Kim MJ, Park J, Oh KW, Kim SH. Data curation: Lee JY, Park YC, Lee J. Formal analysis: Lee JY, Park YC. Investigation: Lee JY, Lee J. Resources: Park J, Oh KW, Kim SH. Validation: Kim MJ, Kim SH. Supervision: Kim MJ, Kim SH. Writing – original draft: Lee JY. Writing – review & editing: Kim MJ.

REFERENCES

1. Singh S, Hamdy S. Dysphagia in stroke patients. Postgrad Med J. 2006;82:383-91. https://doi.org/10.1136/pgmj.2005.043281.
2. Holas MA, DePippo KL, Reding MJ. Aspiration and relative risk of medical complications following stroke. Arch Neurol. 1994;51:1051-3. https://doi.org/10.1001/archneur.1994.00540220099020.
3. Kidd D, Lawson J, Nesbitt R, MacMahon J. The natural history and clinical consequences of aspiration in acute stroke. QJM. 1995;88:409-13.
4. Katzan IL, Cebul RD, Husak SH, Dawson NV, Baker DW. The effect of pneumonia on mortality among patients hospitalized for acute stroke. Neurology. 2003;60:620-5. https://doi.org/10.1212/01.wnl.0000046586.38284.60.
5. Meadows JC. Dysphagia in unilateral cerebral lesions. J Neurol Neurosurg Psychiatry. 1973;36:853-60. https://doi.org/10.1136/jnnp.36.5.853.
6. Barer DH. The natural history and functional consequences of dysphagia after hemispheric stroke. J Neurol Neurosurg Psychiatry. 1989;52:236-41. https://doi.org/10.1136/jnnp.52.2.236.
7. Wijesekera LC, Leigh PN. Amyotrophic lateral sclerosis. Orphanet J Rare Dis. 2009;4:3. https://doi.org/10.1186/1750-1172-4-3.
8. Roche JC, Rojas-Garcia R, Scott KM, Scotton W, Ellis CE, Burman R, et al. A proposed staging system for amyotrophic lateral sclerosis. Brain. 2012;135(Pt 3):847-52. https://doi.org/10.1093/brain/awr351.
9. Tandan R, Bradley WG. Amyotrophic lateral sclerosis: part 1. Clinical features, pathology, and ethical issues in management. Ann Neurol. 1985;18:271-80. https://doi.org/10.1002/ana.410180302.
10. Worms PM. The epidemiology of motor neuron diseases: a review of recent studies. J Neurol Sci. 2001;191:3-9. https://doi.org/10.1016/s0022-510x(01)00630-x.
11. Solazzo A, Del Vecchio L, Reginelli A, Monaco L, Sagnelli A, Monsorrò M, et al. Search for compensation postures with videofluoromanometric investigation in dysphagic patients affected by amyotrophic lateral sclerosis. Radiol Med. 2011;116:1083-94. https://doi.org/10.1007/s11547-011-0698-1.
12. Solazzo A, Monaco L, Vecchio LD, Reginelli A, Iacobellis F, Capasso R, et al. Earliest videofluoromanometric pharyngeal signs of dysphagia in ALS patients. Dysphagia. 2014;29:539-44. https://doi.org/10.1007/s00455-014-9542-9.
13. Horner J, Massey EW. Silent aspiration following stroke. Neurology. 1988;38:317-9. https://doi.org/10.1212/wnl.38.2.317.
14. Paciaroni M, Mazzotta G, Corea F, Caso V, Venti M, Milia P, et al. Dysphagia following stroke. Eur Neurol. 2004;51:162-7. https://doi.org/10.1159/000077663.
15. Robbins J, Levine RL, Maser A, Rosenbek JC, Kempster GB. Swallowing after unilateral stroke of the cerebral cortex. Arch Phys Med Rehabil. 1993;74:1295-300. https://doi.org/10.1016/0003-9993(93)90082-l.
16. Kim SY, Kim TU, Hyun JK, Lee SJ. Differences in videofluoroscopic swallowing study (VFSS) findings according to the vascular territory involved in stroke. Dysphagia. 2014;29:444-9. https://doi.org/10.1007/s00455-014-9525-x.
17. Park YC, Lee JY, Lee JS, Park JS, Oh KW, Kim SH, et al. Characteristics of dysphagia based on the type of ALS in Korean patients evaluated using videofluoroscopic study: a retrospective analysis. Dysphagia. 2022. https://doi.org/10.1007/s00455-022-10430-8. [Epub ahead of print].
18. Logemann JA. The need for clinical trials in dysphagia. Dysphagia. 1998;13:10-1.
19. Han TR, Paik NJ, Park JW, Kwon BS. The prediction of persistent dysphagia beyond six months after stroke. Dysphagia. 2008;23:59-64. https://doi.org/10.1007/s00455-007-9097-0.
20. Rademaker AW, Pauloski BR, Logemann JA, Shanahan TK. Oropharyngeal swallow efficiency as a representative measure of swallowing function. J Speech Hear Res. 1994;37:314-25. https://doi.org/10.1044/jshr.3702.314.
21. Yoon JA, Kim SH, Jang MH, Kim SD, Shin YB. Correlations between aspiration and pharyngeal residue scale scores for fiberoptic endoscopic evaluation and videofluoroscopy. Yonsei Med J. 2019;60:1181-6. https://doi.org/10.3349/ymj.2019.60.12.1181.
22. Hamlet SL, Muz J, Patterson R, Jones L. Pharyngeal transit time: assessment with videofluoroscopic and

scintigraphic techniques. Dysphagia. 1989;4:4-7. https://doi.org/10.1007/BF02407396.
23. Lang IM. Brain stem control of the phases of swallowing. Dysphagia. 2009;24:333-48. https://doi.org/10.1007/s00455-009-9211-6.
24. Daniels SK, Brailey K, Foundas AL. Lingual discoordination and dysphagia following acute stroke: analyses of lesion localization. Dysphagia. 1999;14:85-92. https://doi.org/10.1007/PL00009592.
25. Fattori B, Siciliano G, Mancini V, Bastiani L, Bongioanni P, Caldarazzo Ienco E, et al. Dysphagia in amyotrophic lateral sclerosis: relationships between disease progression and fiberoptic endoscopic evaluation of swallowing. Auris Nasus Larynx. 2017;44:306-12. https://doi.org/10.1016/j.anl.2016.07.002.
26. Logemann JA. Treatment of oral and pharyngeal dysphagia. Phys Med Rehabil Clin N Am. 2008;19:803-16, ix. https://doi.org/10.1016/j.pmr.2008.06.003.
27. Foley N, Teasell R, Salter K, Kruger E, Martino R. Dysphagia treatment post stroke: a systematic review of randomised controlled trials. Age Ageing. 2008;37:258-64. https://doi.org/10.1093/ageing/afn064.
28. Bogaardt HC, Grolman W, Fokkens WJ. The use of biofeedback in the treatment of chronic dysphagia in stroke patients. Folia Phoniatr Logop. 2009;61:200-5. https://doi.org/10.1159/000227997.
29. Palovcak M, Mancinelli JM, Elman LB, McCluskey L. Diagnostic and therapeutic methods in the management of dysphagia in the ALS population: issues in efficacy for the out-patient setting. NeuroRehabilitation. 2007;22:417-23. https://doi.org/10.3233/NRE-2007-22603.
30. Cola MG, Daniels SK, Corey DM, Lemen LC, Romero M, Foundas AL. Relevance of subcortical stroke in dysphagia. Stroke. 2010;41:482-6. https://doi.org/10.1161/STROKEAHA.109.566133.