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Neuroanatomical Basis of Swallowing disorders after Stroke: A Pilot Study

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Abstract

Background—Dysphagia is a common problem after stroke associated with significant morbidity and mortality. Except for patients with brain stem strokes, particularly lateral medullary strokes, it is difficult to predict which cases are likely to develop swallowing dysfunction based on their neuroimaging. Clear models of swallowing control and integration of cortico-bulbar input have not been defined and the role of subcortical structures is unclear.

Objective—To identify supratentorial regions of interest (ROIs) that might be related to clinically important dysphagia in acute stroke patients, focusing on subcortical structures.

Methods—We studied 29 acute supratentorial ischemic stroke cases admitted to our institution between 2001 and 2005 diagnoses with first ischemic stroke and without history of swallowing dysfunction. Subjects had magnetic resonance imaging within 24 hours. Cases were defined as those subjects who were diagnosed as dysphagic after clinical evaluation by a speech language pathologist (SLP) and whose dysphagia was considered clinically significant i.e., requiring treatment by diet modification. Controls were defined as those patients who: (1) passed the stroke unit's dysphagia screening, (2) had a clinical evaluation by SLP that did not result in a diagnosis of dysphagia or diet modifications, or (3) had no documented evidence of dysphagia evaluation or treatment during hospitalization and were discharged on a regular diet. A trained technician, blinded to case-control status, examined 12 ROIs for dysfunctional tissue in diffusion and perfusion-weighted images. The odds ratio (OR) of dysphagia was calculated for each ROI. Logistic regression models were used to adjust for stroke severity (NIHSS) and volume.

Results—Analysis of data on 14 cases and 15 controls demonstrated significant differences in the unadjusted odds of dysphagia for the following ROIs: 1) primary somatosensory, motor and motor supplementary areas (PSSM) (OR=10, p=0.009); 2) orbitofrontal cortex (OFC)(OR=6.5, p=0.04); 3) putamen, caudate, basal ganglia (PCBG)(OR=5.33, p=0.047); and 4) internal capsule (IC)(OR=26; p=0.005). Non-significant differences were found in the insula and temporopolar cortex. Adjusted OR of dysphagia for subjects with strokes affecting the IC was 17.8 (p=0.03). Adjusted odds ratios for the PSSM, OFC, and PCBG were not statistically significant.

Conclusion—Significantly increased odds of dysphagia were found in subjects with IC involvement. Other supratentorial areas that may be associated with dysphagia include the PSSM, OFC, and PCBG. Analysis of additional areas was limited by the number of subjects in our sample. Future studies with larger sample size are feasible and will contribute to the development of a full swallowing control model.

INTRODUCTION

Dysphagia is a common problem after stroke and has been identified as an independent predictor of morbidity and mortality. Except for patients with brain stem strokes, particularly lateral medullary strokes which are commonly associated with dysphagia, it is difficult to predict which cases are likely to develop swallowing dysfunction based on their neuroimaging.

Current assessments concentrate on identifying signs and symptoms of dysphagia that would cue for the need for further evaluation or intervention. Instruments used for bedside clinical assessments are not very sensitive and silent aspiration can be missed [1]. Except for a small minority of cases who experience brain stem strokes, it is not possible to predict if a particular stroke case is likely to have dysphagia.

Comprehensive models for neural control of swallowing and integration of cortico-bulbar input have not been defined and the role of subcortical structures is unclear. Early models described the role of the inferior precentral gyrus. Models developed by Daniels et al. suggest that subcortical structures such as the thalamus and basal ganglia as well as input from both cerebral hemispheres and the insula to and from the brain stem are critical in the swallowing network [2]. Their findings also suggest that lesions disrupting cortical-subcortical connectivity are more likely to increase the risk of aspiration in stroke patients as compared to isolated cortical or subcortical lesions. Other models suggest parallel networks between cortical areas and the cerebellum [3].

With current magnetic resonance imaging (MRI) techniques, including diffusion (DWI) and perfusion weighting (PWI), we can not only delineate areas of infarction but areas that are dysfunctional as a result of hypoperfusion in the tissues surrounding the stroke (Figure 4.1)[4]. This area, the ischemic penumbra, is important in stroke not only for research, but for clinical purposes since this area has been associated with response to treatment 5. Deficits disproportionate to the diffusion abnormalities but proportionate to perfusion abnormalities have been found to correlate with potential for recovery 6. Traditional lesion analysis did not allow analysis of dysfunctional or ischemic brain, but only infarcted brain. Dysphagia is particularly difficult to study because it improves quickly, thus studying it requires early assessment of both stroke and swallowing. Determining what areas of the brain are dysfunctional allows for clearer stroke-to-deficit assessments thus allowing us to build on the previously proposed models to develop a comprehensive model of swallowing control.

The lack of a comprehensive swallowing control model has limited our ability to design therapies geared toward the specific dysfunction a particular patient might be experiencing as determined by their stroke. Ideally, neuroimaging obtained for stroke ascertainment should function as a tool to risk-stratify patients as to the possibility of dysphagia and dysphagia complications. Developing such a model is critical in the understanding of swallowing dysfunction and will provide the framework for the development of evidence-based dysphagia interventions.

The purpose of this study was two-fold: 1) To identify supratentorial regions of interest (ROIs) that might be related to clinically significant dysphagia (requiring intervention) in acute stroke patients, with a focus on subcortical structures, and 2) to determine if the data obtained as part of clinical practice allow the recruitment of a large cohort to study in detail those brain locations that might be associated with dysphagia.

METHODS

Study Design and sample

We conducted a case-control study by reviewing the medical charts of patients previously enrolled in other stroke studies in our group from 2001–2005. The subjects were admitted to our institution’s stroke unit with a diagnosis of first ischemic stroke. Exclusion criteria included previous history of stroke or swallowing disorders, contraindication for MRI, reduced level of consciousness, ongoing sedation, or stroke limited to the brainstem. Individuals with history of other neurological disorders were also excluded since many neurological diseases are also associated with swallowing disorders. MRI was obtained

within 24 hours of onset. The study was approved and informed consent was waived by the Institutional review Board.

The sample size required to detect a significant odds ratio of 10 or higher with a=0.05, 90% power and a probability of exposure in the controls of 40% is 20 subjects. Conversely, with a sample size of 29 and the aforementioned parameters we can detect odds ratios of 6.08 or higher.

Inclusion Criteria

Cases were defined as those subjects who were both: 1) diagnosed as dysphagic after clinical evaluation by a speech language pathologist (SLP); and 2) whose dysphagia was clinically significant in that it required treatment by diet modification.

Controls were defined as those patients who: (1) passed the stroke unit's dysphagia screening, (2) had a clinical evaluation by SLP that did not result in a diagnosis of dysphagia or need for diet modifications, or (3) had no documented evidence of dysphagia evaluation or treatment during hospitalization and were discharged home on a regular diet.

Other study variables

Demographic characteristics such as age, gender, and race were abstracted from the medical records. To ascertain the effect of stroke severity we abstracted the NIH stroke scale (NIHSS) from the medical chart whenever available. The NIHSS is recorded upon admission by the treating physicians. For those cases where the NIHSS was not available it was calculated using the protocol validated by Williams, et al.[7] using the admission history and physical examination.

Volumes of infarct were computed from the admission MRI - DWI images using Image J software (http://rsb.info.nih.gov/ij). Abnormal tissue was manually traced on each separate slice, multiplied by the thickness, and computed across the entire brain to assess the total volume of stroke.

Image Acquisition and Processing

All imaging was performed on a 1.5 Tesla scanner using a standard quadrature transmit-receive head coil. In addition to time-of-flight MRA, conventional T1- and T2-weighted images, and FLAIR MRI, isotropic DWI images were obtained (b_{max} =1000 s/mm^2; TR/TE of 10,000 msec/120 msec). PWI images were recorded during bolus injection of 20 cc Gadopentate dimeglumine (GdDTPA) at 5 cc/sec using single-shot gradient-echo echo-planar imaging (TR/TE of 2000/60 msec, 30 dynamics). PWI scans were post-processed to generate maps of time to peak (TTP) arrival of the GdDTPA in each voxel. Hypoperfusion was defined as ≥4 s mean delay in TTP arrival of contrast across voxels in the region of interest (ROI) relative to the homologous region in the contralateral hemisphere/region. This threshold was based on evidence that tissue with this degree of hypoperfusion is dysfunctional, although it may not be at risk for progressing to infarction, whereas a delay of <2.5 s is not associated with dysfunction[8,9].

Image Analysis

The reported analyses used DWI (after confirming the acuity of the lesion as dark on absolute diffusion coefficient (ADC) maps) and PWI (co- registered to T2 to provide anatomical boundaries less visible on PWI). Areas were considered dysfunctional if they were bright on DWI and dark on ADC maps and/or were hypoperfused on PWI TTP maps (using a threshold of delay that has been previously demonstrated to correspond to

dysfunction). A trained technician, blinded to case-control status, examined 9 distinct, non-overlapping regions of interest (ROIs) listed in Table 4 for dysfunctional tissue.

Selection of ROI's

The ROI's were defined after a literature search to identify areas that have been implicated in swallowing function either through original research, case reports, meta-analysis or review articles. We chose to include areas whose function is unclear or whose involvement in swallowing has been questioned in addition to areas that have been clearly implicated in swallowing function. Details about the selected ROI's and their hypothesized roles in swallowing function are detailed in Table 1.

Data analysis

Sample characteristics were compared using 2-sample test of proportions or t-tests as applicable. Fisher's exact test was used when counts were fewer than 5 subjects per cell. Chi-square statistics were used to determine statistical significance of the crude odds ratios. Logistic regression models were used to adjust for stroke severity (NIHSS) and stroke volume (cc) in those cases where statistically significant crude odds ratios were identified. The critical value for statistical significance was set at p=0.05. Data analysis was performed using Intercooled Stata 8.2 (Statacorp, College station, Texas).

RESULTS

We identified 29 stroke cases that met inclusion and exclusion criteria, of which 14 were classified as cases and 15 as controls. The characteristics of the sample are described in table 2. All the cases had a documented clinical bedside evaluation performed by a speech language pathologist (SLP). Three controls (20%) had a clinical bedside evaluation performed by an SLP. Approximately 29% of cases also had a documented dysphagia screening evaluation by a nurse or physician (but all had an evaluation by an SLP), while 67% of controls had a documented dysphagia screening by a nurse or physician. This difference was statistically significant. Three controls (20%) had no documentation of a formal evaluation, but remained on regular diets throughout their inpatient stay and were discharged home on a regular diet. Of the total sample, 93% of stroke subjects were evaluated either by screening or formal SLP assessment.

There were no significant differences in the age or gender distribution between the groups. Whites and African-Americans each represented approximately half of the population in both groups, and no cases from other minority groups were identified, reflecting the racial distribution of our stroke unit. We determined the side of stroke for cases and controls but no statistically significant differences were found (Table 2). No bilateral stroke cases were identified.

Stroke infarct volume and stroke severity varied between the groups. Mean stroke volume was 51.7 cc for cases and 23.0 cc for controls (p=0.05). Mean NIHSS was 11.4 for cases and 6.2 for controls ($p < 0.02$).

We had data on the timing of the screening or SLP evaluation on 26 out of the 27 subjects who had a documented evaluation (Table 3). We had no date of evaluation on the bedside screening of one control. 65% of all subjects were evaluated by either screening or SLP within 1 day of admission; more than 75% were evaluated within 3 days; and 92% had a formal evaluation within 7 days. Of the 13 patients who had a documented time of screening, 12 (92%) had the evaluation within 1 day of admission. One subject was evaluated at day 6 because she had been intubated prior to that time. SLP bedside evaluation times were more variable: 59% had SLP evaluation within one day of admission, 29% had

the evaluation after 1 day but within 1 week, and 12% had the evaluation after 1 week. The NIHSS score for subjects who had only a bedside screening was 6.6, which was somewhat lower (less impaired) than the NIHSS score of 10.4 for subjects who had an SLP bedside evaluation (p=0.07).

Results of our ROI analysis are detailed in table 4. The unadjusted odds ratio of dysphagia in cases of stroke affecting the internal capsule was 26 (p=.005). Other areas with statistically significant unadjusted odds ratios for dysphagia included the primary somatosensory, motor and motor supplementary cortices (OR=10, p=.009), the orbitofrontal cortex (OR=6.5, p=0.044), and the basal ganglia (caudate and/or putamen) (OR=5.33, p=0.04). No statistically significant differences were found for the temporopolar cortex, insula, thalamus, or parietoccipital cortex.

Attempts to adjust for stroke volume and NIHSS were limited by our small sample size. Logistic regression models were used to adjust for both volume and NIHSS for the 4 ROIs that had statistically significant unadjusted odds ratios in our initial analysis (Table 5). The adjusted odds ratio for dysphagia was 17.8 for the internal capsule (p=0.027, adjusted for stroke volume and NIHSS). The adjusted odds ratios for dysphagia were 5.26 for the primary somatosensory, motor and motor supplementary cortices; 2.19 for the orbitofrontal cortex and 4.59 for the putamen, caudate and basal ganglia; these were not statistically significant.

DISCUSSION

We found a positive association between the development of dysphagia and acute stroke involving the internal capsule. This association was significant after adjusting for stroke severity (NIHSS) and stroke volume. Other areas that were associated with dysphagia were the primary somatosensory, motor and motor supplementary cortices, the orbitofrontal cortex, and the basal ganglia (caudate and/or putamen). After adjustment for stroke severity and volume, these differences were not statistically significant. The fact that these areas failed to reach statistical significance is most likely a reflection of the small sample size in this study and not the lack of a true biological association, but this speculation needs to be confirmed in a larger study. Our analyses also demonstrate that it is feasible to perform a prospective cohort study of stroke patients obtaining MRI neuroimaging and dysphagia ascertainment in a timely manner.

In our study we chose to define dysphagia using clinical criteria. Our focus was clinically significant dysphagia; thus, our definition includes dysphagia that required intervention by diet modification. This definition is likely to exclude individuals who might have physiologic abnormalities but whose dysphagia was not relevant for treatment purposes. Future studies using similar techniques can also include evaluation with videofluoroscopy so that stroke location can be correlated with specific physiologic deficits.

Another challenge in studying swallowing dysfunction in stroke cases is that dysphagia is time-dependent. Dysphagia improves for half of stroke patients within 7 days and less than 15% have persistent dysfunction after 6 months 10›11. The conclusions we can draw about the brain locations affected in association with the diagnosis of dysphagia, or the lack of thereof, are limited if the ascertainment of both stroke and dysphagia are delayed. Previous studies report varying dysphagia incidence related to different times to dysphagia assessment [12]. Early evaluation is critical to identify areas whose involvement might be associated with dysphagia in the acute setting, or with chronic swallowing dysfunction. In our study, the time to MRI was less than 24 hours. To our knowledge, no studies in this area have examined such an acute population. Dysphagia was consistently evaluated within 7

days after the stroke for the vast majority of subjects which makes it feasible to study this problem in a prospective cohort with minimal changes to clinical practice. The imaging employed in this study as well as the clinical swallowing evaluations available allow for prospective studies in which serial imaging and dysphagia evaluations are obtained to elucidate the areas of the brain associated with acute (resolving within 7 days) versus chronic swallowing dysfunction after stroke.

We adjusted for stroke severity and volume despite the small sample size. The fact that a positive association was found in the IC is striking. The IC is an important area in the relay of information from the brain stem to the cortex as previously reported[13,14]. It is reasonable to hypothesize that strokes in this location may result in an acute disconnection between cortical swallowing centers and the central pattern generator for swallowing in the rostral medulla. Even more interesting would be to determine if strokes affecting this area of the brain are associated with acute versus chronic dysfunction. We hypothesize that dysphagia resulting from acute disconnection between the cortex and the brain stem is likely to improve quickly as compared to dysphagia resulting from damage to important areas for swallowing control in the brain stem and cortex. This hypothesis will be the focus of future studies and can contribute to elucidating the functional importance of the internal capsule and other areas of the brain.

The absolute magnitude of the effect reported for the internal capsule should be interpreted with caution since the small sample size reduced the precision of our estimates. We are confident that, in the future, larger studies will demonstrate significant differences in other brain regions and generate more precise risk estimates.

Analysis of other areas that are important in swallowing function was limited by small number of strokes involving those regions. It is well known that the brain stem is critical in swallowing function as evidenced in by the high incidence of dysphagia in cases with lateral medullary stroke. Therefore, we focused on indentifying supratentorial structures where tissue dysfunction is associated with dysphagia in this study.

Previous studies have attempted to describe brain locations associated with dysphagia and aspiration after stroke using CT and MRI, but failed to find an association 15,[16]. Early MRI techniques did not allow evaluation of the total area of dysfunctional brain tissue (e.g. with PWI), and this may explain the negative studies. We believe that studying diffusion and perfusion abnormalities gives us a better picture of acutely dysfunctional brain tissue and allows for correlation with acute dysphagia.

Although we were unable to study differences between right and left strokes because of small sample size, the effect of stroke laterality on swallowing function is important. Robbins and Levine were among the first to describe disordered swallowing in unilateral cortical strokes [17]. Later they described differences in pharyngeal transit time for middle cerebral artery territory strokes when comparing location (anterior vs. posterior) and side (left vs right) [18]. Studies by Hamdy et al. 19–22 suggest that swallowing is represented bilaterally but asymmetrically with no clear right or left laterality and that the size of the cortical area associated with swallowing in the unaffected cortex determines the presence or absence of dysphagia. This suggests that there is the possibility of unilateral hemispheric dominance that varies between individuals. Further studies using serial imaging and transcranial magnetic stimulation to identify the swallow-dominant side can help determine whether there is a dominant hemisphere in each individual that, if affected, results in swallowing dysfunction or if the increased representation of swallowing in the unaffected hemisphere is a result of cortical reorganization and compensation.

Dysphagia ascertainment and diagnosis presents some challenges. Trained SLPs usually perform bedside evaluations and determine whether further testing is warranted. Their diagnostic assessment is typically based on subjective as well as objective findings. It is not feasible to use the gold standard, videofluoroscopy (VFSS), in all stroke patients or in the number that would be required in a prospective study. In the present study we defined dysphagia as swallowing dysfunction identified during a bedside swallowing evaluation that required diet modification. This definition was chosen since it was likely to identify cases that were truly dysphagic (reducing the possibility of false positives) at the expense of possibly missing milder cases of dysphagia.

A bedside evaluation protocol has been recently validated for stroke patients [23]. This assessment creates a total composite score base on items traditionally evaluated by SLP during a bedside swallowing evaluation. This scale can provide objective evidence to the presence of dysphagia and potentially allow for the inclusion of a larger spectrum of dysphagic cases in future studies.

It was also important to determine whether patients were consistently evaluated for dysphagia. The percentage of patients who had a documented bedside screening test was much larger for control subjects than for dysphagic stroke patients. It is likely that our cases had such obvious signs of dysphagia that the SLP was consulted and the screening not documented. It is also important to note that out of the 10 screening tests documented in controls only one led to further evaluation. This also leads us to believe that the screen was infrequently documented when there were obvious signs of dysphagia.

It is encouraging that 93% of our total sample had documented clinical evaluation of swallowing function. Only 3 controls lacked formal dysphagia evaluation. In those cases lacking documentation as to the absence of dysphagia, we carefully reviewed all records for evidence of swallowing dysfunction. These subjects were never evaluated by an SLP, did not have a VFSS, their diet was never modified, and they were discharged on a regular diet. We believe that the screening might not be documented in patients who appear to be clinically “normal” to the evaluators. In a larger prospective sample, special attention will be required to insure that there is documentation of the absence of dysphagia whether by screening or SLP evaluation. We and other stroke units have recently started to require documentation of a dysphagia bedside screen or SLP evaluation on all stroke patients, consistent with the American Heart Association “Get with the Guidelines” initiative (http://www.americanheart.org/presenter.jhtml?identifier=1165), which will facilitate future studies.

CONCLUSION

A positive association was found between infarct/hypoperfusion involving the internal capsule and development of clinically important dysphagia after stroke. This association was significant after adjusting for stroke severity and stroke volume. Statistically significant association of dysphagia with other stroke locations was precluded by sample size. We also determined that the data necessary to study this problem prospectively can be obtained accurately and in a timely manner with minimal alteration of current clinical practice. A prospective cohort study of acute stroke patients is likely to allow for more detailed comparisons and aid in the development of a comprehensive model of swallowing control.

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Table 1

Regions of Interest selected for analysis and their hypothesized role in swallowing function.

ROI	Hypothesized role	References
Primary Somatosensory, motor and Motor Supplementary cortices (BA 1, 2, 3, 4, and 6)	Cortical processing of swallowing, including motor regulation and execution and sensorimotor control.	Hamdy, et al. [24,25] Mosier and Bereznaya[3] Martin, et al.[26]
Anterior cingulate (BA 24 and 32)	Higher order motor processing: swallowing movement planning and execution.	Hamdy et al. [24,25]
	Cognitive perceptual processes such as attention and response selection.	Martin, et al.[26] Martin, et al. [27]
Orbitofrontal cortex (BA 10, 11, 12, 44, 45, and 47)	Unclear	Mosier, et al. [14]
Parieto-occipital cortex (BA 7, 17, 18, 40)	Sensory processing of swallowing.	Hamdy, et al. [24] Kern, et al. [28]
	Task-cue processing not swallowing per se.	Toogood, et al. [29]
	Movement planning and execution.	Mosier and Bereznaya[3]
Temporopolar cortex (BA 22 and 38)	Unclear	Mosier, et al. [14]
Insular cortex	Processing of gustatory input.	Daniels, et al. [30] Daniels and Foundas [31]
	Intraoral sensory modulation.	Mosier, et al. [13,14]
Internal capsule	Functional connection of the cortical and brain stem nuclei via the corticobulbar tracts.	Mosier, et al. [13,14]
Thalamus	Sensory and motor input processing via thalamocortical and thalamostriatal pathways.	Daniels, et al.[1] Mosier, et al.[14] Mosier, et al.[3]
Basal Ganglia (caudate and/or putamen)	Gating of Sensory Input.	Mosier and Bereznaya[3] Daniels, et al.[1] Suzuki, et al.[32]
Cerebral Peduncle	Descending pathways from the cortex.	Miller[33]
Brain Stem	Central pattern generator, swallowing regulation.	Jean [34–36]
Cerebellum	Regulation of adaptive coordination, sequencing, timing, learning and memory of motion.	Zald and Pardo [37] Mosier and Bereznaya[3] Suzuki, et al.[32]

Table 2

Demographic Characteristics of Stroke Subjects, n=29

Characteristic	Cases N=14	Controls N=15	p-value
Age, mean (SD)	62.6 (14.3)	57.2 (19.5)	0.40
Male, count (%)	7 (50.0)	7 (46.7)	0.85
African-American, count (%)	6 (42.9)	8 (53.3)	0.55
NIH Stroke Scale, mean (SD)	11.4 (5.3)	6.2 (5.9)	0.02
Stroke volume, cc (SD)	51.7 (44.9)	23.0 (30.6)	0.05
Left hemisphere stroke, count (%)	4 (26.7)	7 (50)	0.26
Water swallow screen, count (%)	4 (28.6)	10 (66.7)	0.06
Bedside evaluation, count (%)	14 (100)	3 (20)	--
Both screening and bedside, count (%)	4 (28.6)	1 (6.7)	--
No documented evaluation, count (%)	0	3 (20)	--
Had Videofluoroscopy, count (%)	10 (71.4)	0	--

Table 3

Time from onset to swallowing evaluation, n=26

Time to swallow evaluation	Screening evaluation n=13	SLP Bedside evaluation n=17	Had either or both n=26	Cases n=14	Controls n=12	p- value
<=1 day, n (%)	12 (92)	10 (59)	17 (65)	9 (64)	8 (67)	0.89
<=3 days, n (%)	12 (92)	12 (71)	20 (77)	10 (71)	10 (83)	0.65
<= 7days, n (%)	13 (100)	15 (88)	24 (92)	13 (93)	11 (92)	0.91
>7days, n	0	2	2	1	1	

*All are cumulative percentages

Table 4

Region of Interest (ROI) analysis, Stroke subjects, n=29

Location	Cases (14)	Controls (15)	Crude OR	p-value
Primary Somatosensory, motor and motor Supplementary, n (%)	10(71)	3(20)	10	0.009
Orbitofrontal, n (%)	7(50)	2(13)	6.5	0.033
Basal ganglia, n (%)	8(57)	3(20)	5.3	0.039
Temporopolarcortex, n (%)	8(57)	5(33)	2.7	0.198
Insula, n (%)	8(57)	4(27)	3.7	0.096
Internal Capsule, n (%)	13(92.9)	5(33)	26	0.001
Thalamus, n (%)	2(14.3)	4(27)	--	0.410
Parietooccipital, n (%)	6(42.9)	7(47)	--	0.837
Anterior cingulate, n	0	1	--	--

Table 5

Multivariable Logistic regression models, odds ratio of dysphagia for each ROI, n=29

Model	aOR	95% CI	
1. Internal Capsule	17.76	1.39	>100
NIHSS	1.01	0.98	1.05
Stroke Volume	1.15	0.97	1.38
2. Primary SS/Motor	5.26	0.74	37.42
NIHSS	1.01	0.98	1.04
Stroke Volume	1.15	0.97	1.37
3. Orbitofrontal	2.19	0.22	21.47
NIHSS	1.01	0.98	1.046
Stroke Volume	1.17	0.98	1.40
4. Putamen/Caud/BG	4.59	0.68	30.79
NIHSS	1.01	0.99	1.05
Stroke Volume	1.18	0.98	1.42

aOR – adjusted odds ratio