Dement Neurocogn Disord. 2026 Apr;25(2):103-114
https://doi.org/10.12779/dnd.2026.25.2.103
pISSN 1738-1495·eISSN 2384-0757

DND Dementia and Neurocognitive Disorder

Original Article

Check for updates

Dysphagia Characteristics and Its Neuroanatomical Correlates in Korean Patients With Subcortical Vascular Cognitive Impairment

Mee Kyung Suh [1], **Yeong Ju Cho** [1], **Byung Hwa Lee** [1], **Yisuh Ahn** [2]

[1]Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea
[2]The BOM Brain Health Neuropsychology Center & Cognitive Rehabilitation Research Institute, Seoul, Korea

OPEN ACCESS

Received: Mar 9, 2026
Revised: Apr 6, 2026
Accepted: Apr 6, 2026
Published online: Apr 15, 2026

Correspondence to
Mee Kyung Suh
Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, 81 Irwon-ro, Gangnam-gu, Seoul 06351, Korea.
Email: mk.suh@samsung.com

ORCID iDs
Mee Kyung Suh
https://orcid.org/0000-0003-2558-8018
Yeong Ju Cho
https://orcid.org/0009-0005-0208-4716
Byung Hwa Lee
https://orcid.org/0000-0001-7415-8554
Yisuh Ahn
https://orcid.org/0009-0005-8576-1902

Conflict of interest
There is no conflict of interest to disclose.

ABSTRACT

Background and Purpose: Subcortical vascular cognitive impairment (SVCI) is frequently associated with dysphagia, but its specific characteristics and underlying neural mechanisms remain unclear. This study aims to investigate the swallowing characteristics in SVCI patients and examine their neuroanatomical correlates using diffusion tensor imaging-voxel-based morphometry (DTI-VBM).
Methods: Eighty-five SVCI patients and 20 normal controls underwent videofluoroscopic swallowing studies and 3.0-Tesla magnetic resonance imaging. Swallowing components were evaluated using Modified Barium Swallow Impairment Profile, Penetration-Aspiration Scale, and temporal measurements. Fractional anisotropy and mean diffusivity maps were analyzed to determine neuroanatomical correlates.
Results: SVCI patients exhibited significant differences in tongue control during bolus hold, bolus preparation/mastication, bolus transport/lingual motion, oral residue in the oral stage and anterior hyoid excursion, epiglottic movement, laryngeal vestibular closure, tongue base retraction, vallecular residue, pyriform sinus residue, pharyngeal esophageal segment opening in the pharyngeal stage. Temporal measurements revealed significant delay in pharyngeal transit times (PTT). Time gap was shorter between initiation and maximal displacement of hyoid, hyoid maximal displacement and aryepiglottic closure, and longer between initiating movement of hyoid and bolus head reaching vallecula. DTI-VBM demonstrated that prolonged oral transit time correlated with the right corona radiata and anterior cingulate cortex. Prolonged PTT significantly correlated with microstructural alterations in the anterior insula, anterior cingulate cortex, corona radiata, internal capsule, corpus callosum, thalamus, brainstem, and cerebellar circuits.
Conclusions: SVCI patients manifest dysphagia in the oral and pharyngeal stage. Temporal measurements reveal delay in PTT which is correlated with widespread disruptions in the corticobulbar tract, frontal subcortical circuits, and cerebellar control circuits.

Keywords: Dysphagia; Dementia, Vascular; Time Factors; Diffusion Tensor Imaging

DND Dementia and Neurocognitive Disorder

Author Contributions

Conceptualization: Suh MK; Data curation: Suh MK, Cho YJ; Formal analysis: Suh MK, Cho YJ, Lee BH, Ahn Y; Investigation: Suh MK; Methodology: Suh MK; Project administration: Suh MK; Writing - original draft: Suh MK; Writing - review & editing: Suh MK, Cho YJ, Lee BH.

INTRODUCTION

Subcortical vascular cognitive impairment (SVCI) is a group of disorders characterized by cognitive impairment associated with ischemic changes in the white matter or deep nuclei (basal ganglia or thalamus) caused by small vessel diseases.[1] It encompasses subcortical vascular dementia (SVaD) and its prodromal stage, subcortical vascular mild cognitive impairment (SVMCI).[2] The ischemic changes in SVCI result in both cognitive dysfunctions and motor impairments. Among the most common focal neurological signs of SVaD—dysarthria, gait disturbance, bradykinesia, and dysphagia[3]—dysphagia is particularly critical. It requires cautious monitoring as it directly leads to malnutrition,[4] dehydration, weight loss, and aspiration pneumonia, significantly affecting the morbidity and mortality of patients.[5] Although numerous studies report that dysphagia frequently manifests during the course of SVaD,[6-8] no studies have specifically addressed the dysphagia characteristics and the underlying neurological causes in this patient population. Currently, understanding of their swallowing characteristics relies largely on insights obtained from general vascular dementia, focal stroke lesions, or Alzheimer's disease.[7,8]

Swallowing is a complex phenomenon requiring a highly coordinated sequence of movements, demanding sophisticated activation among the cortical, subcortical, and brainstem regions. The supratentorial network of swallowing includes the primary motor cortex, supplementary motor area, insula, thalamus, basal ganglia, and the anterior cingulate, which are critical for modulation and initiation of the volitional oral stage.[9-17] The infratentorial network, consisting of the medulla's central pattern generator and cranial nerves, primarily orchestrates the automatic and reflexive pharyngeal stage.[18-20] One of the diagnostic hallmarks of SVCI is the disruption of the subcortical white matter. Based on Wallerian degeneration, this disruption in SVCI results in diffuse dysfunction across the cortex, basal ganglia, cerebellum, and brainstem.[21-24]

Crucially, there is a significant anatomical overlap between the neuroanatomical correlates of SVCI and the networks associated with swallowing functions. The disrupted networks in SVCI include the corticobulbar tract, frontal subcortical circuits (such as the cortico-basal ganglia-thalamic motor loop and the anterior cingulate circuit), and the corticopontocerebellar circuits.[25]

Based on these shared neuroanatomical correlates, a unique pattern of swallowing characteristics in patients with SVCI is anticipated. The information would help in predicting and understanding the pattern of dysphagia in this patient population. Therefore, the purpose of this study is to investigate the characteristics of dysphagia in patients with SVCI, by comparing the swallowing characteristics between the patient group and normal controls. For this purpose, videofluoroscopic swallowing study (VFSS) was completed in both groups and the VFSS was analyzed using Modified Barium Swallow Impairment Profile (MBSImP) and the Penetration-Aspiration Scale (PAS). Additionally a discrete temporal measurements of swallowing events were measured to detect the subtle characteristics of swallowing performances and to examine the neuroanatomical correlates using diffusion tensor imaging-voxel-based morphometry (DTI-VBM).

https://doi.org/10.12779/dnd.2026.25.2.103

DND Dementia and Neurocognitive Disorder

METHODS

Participants

The study included 85 SVCI patients (53 with SVMCI and 32 with SVaD) and 20 age-matched normal controls (**Table 1**). Participants underwent a VFSS and a high-resolution T1-weighted volumetric magnetic resonance imaging (MRI) scan at a tertiary hospital in Seoul, Korea, between August 2008 and December 2009. This study was approved by the Institutional Review Board of Samsung Medical Center (IRB No. 2011-06-017-001). The diagnosis of SVMCI was based on modified criteria proposed by Petersen et al.,[3] encompassing subjective cognitive complaints, normal general cognitive function and activities of daily living, objective cognitive decline, presence of focal neurological signs, and significant small vessel ischemic changes without territorial infarction on MRI. SVaD patients met the diagnostic criteria for vascular dementia according to the DSM-IV and fulfilled the imaging criteria proposed by Erkinjuntti et al.[26]

Swallowing evaluation

To comprehensively evaluate swallowing functions, VFSS protocol was completed for all the patients and normal controls. The participants were given 3, 5, 9 cc, and cup drinking of thin liquid (milk), 5 and 9 cc of thick liquid (apple sauce thickness), 5 and 9 cc of puree (grinded rice porridge with pudding thickness), and 1/2 spoon and full spoon of semi-solid food (rice porridge). VFSS was performed by 2 speech-language pathologist and a radiologist and the images were independently analyzed by the speech-language pathologists.

To evaluate the clinical characteristics of dysphagia we analyzed the VFSS data using the component and scoring methods of a validated VFS tool, MBSImP[27] to evaluate the distinct physiologic components of swallow. MBSImP includes rating scales for 17 distinct physiologic components of swallowing, categorized into 3 functional domains: oral (components 1–6), pharyngeal (components 7–16) and esophageal (component 17). The tool uses an ordinal scale with higher scores indicating greater impairment. For the current study the authors analyzed the all 6 oral components, but partially altered the pharyngeal scale due to clinical purposes and limitation of data images. Out of the 10 pharyngeal components, component 11 (laryngeal vestibular closure) was excluded due to limitations in clinical settings. Component 13 (pharyngeal contraction) and component 17 (esophageal clearance upright positions) were not analyzed as well because the anterior-posterior view of the VFSS study were not available in some of the patient data. Component 16 (pharyngeal residue) was divided into 2 components, the vallecular residue and pyriform sinus residue, because clinically there are discrepancies between the amount of residue pooling in these 2 areas. Thus, our study includes 9 components in the pharyngeal stage of swallow. Further, to evaluate the airway entrance of the bolus, the PAS, an 8-point scale that describes the

Table 1. Demographic and clinical characteristics of the study participants

Variable	SVCI (n=85)	Normal (n=20)
Sex, female	56 (65.88)	12 (60.00)
Age (yr)	73.65±6.89	71.45±5.65
Education (yr)	9.28±5.13	11.84±4.33
MMSE	23.03±5.29	28.15±1.30
CDR	0.74±0.45	0.00±0.00

Values are presented as mean ± standard deviation or number (%). Within the SVCI group, 53 (62.35%) were classified as SVMCI and 32 (37.65%) as SVaD.
SVCI: subcortical vascular cognitive impairment, MMSE: Mini-Mental State Examination, CDR: Clinical Dementia Rating, SVMCI: subcortical vascular mild cognitive impairment, SVaD: subcortical vascular dementia.

https://doi.org/10.12779/dnd.2026.25.2.103

degree to which the bolus enters the airway and whether it is subsequently ejected,[28] was used. Mean value of each of the components of MBSImP and PAS was used for this study.

For the subclinical swallowing measurements, we utilized a temporal measurement method to detect subtle changes in the swallowing events. The VFSS recordings were digitized and analyzed frame-by-frame at 30 frames per second. The measurement criteria proposed by Kendall et al.[29] was used. Posterior nasal spine (B1) was designated as the anatomical baseline to divide the termination of the oral stage and the initiation of the pharyngeal stage (**Fig. 1**). The 'bolus transit' was divided into oral transit time (OTT), which is time measured from the initiation of tongue movement to begin the voluntary oral stage to B1 and the pharyngeal transit time (PTT), which is time measured from B1 to bolus exiting the upper esophageal sphincter (UES). The PTT was further divided into oropharyngeal transit (OPT), time measured from B1 to vallecula and laryngo-pharyngeal transit (LPT), which is time measure from bolus reaching vallecula (reachVALLE) to bolus exiting the UES (UESexit). For 'swallowing gestures' of the pharyngeal stage, B1 was set as baseline and lapse time to each of the pharyngeal structure movement was measured (**Fig. 1**). Temporal measurement points of swallowing gestures are as follows: soft palate starts to elevate (SPstart), soft palate reaches maximum closure (SPclose), initiation of aryepiglottic elevation (AEstart), aryepiglottic fold elevation to the point of supraglottic closure (AEclose), initial movement of hyoid elevation (hyoidST), hyoid reaching maximal displacement (hyoidMAX) and onset of upper esophageal sphincter opening (UES open). Additionally, for better understanding of the swallowing physiology in SVCI patients, time gaps between the initiating and finishing movement of a swallowing gestures and between bolus flow and starting or finishing point of swallowing gestures considered important for airway protection swallowing was calculated. Temporal measurement points for the time gap data are as follows: SPclose-SPstart, AEclose-AEstart, hyoidMax-hyoidST, hyoidST-AEstart, hyoidMAX-AEclose, hyoidST-valleRE, UESexit-UESopen, pyriformRE-UESopen, and UESopen-hyoidMAX.

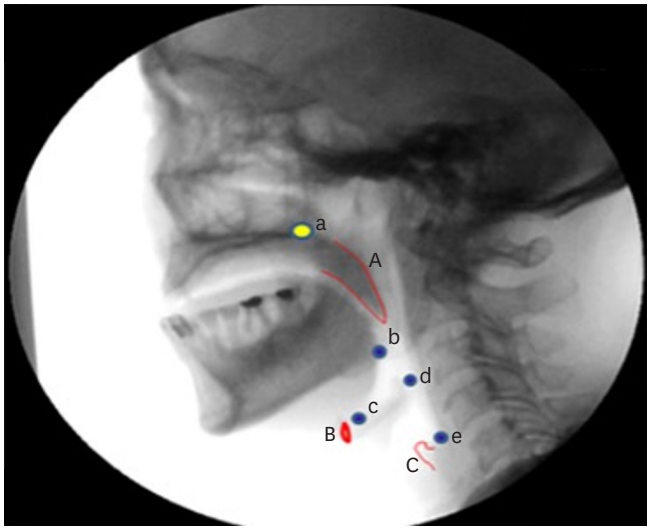

Fig. 1. Illustration of subclinical temporal measurement points in VFSS.
a: Baseline (B1), b: manRAM, c: valleRE, d: valleEX, e: pyriformRE, A: soft palate, B: hyoid bone, C: aryepiglottic fold.
VFSS: videofluoroscopic swallowing study, B1: posterior nasal spine, manRAM: bolus head reaching mandibular ramus, valleRE: bolus head reaching vallecula, valleEX: bolus tail exiting vallecula, pyriformRE: bolus head reaching pyriform sinus.

https://doi.org/10.12779/dnd.2026.25.2.103

DND Dementia and Neurocognitive Disorder

Image acquisition and processing

MRI was performed using a 3.0-Tesla scanner (GE Signa, Milwaukee, WI, USA). To measure white matter hyperintensities (WMH), T2-weighted fluid-attenuated inversion recovery (FLAIR) images were acquired (repetition time [TR]=11,000.0 ms, inversion time [TI]=2,800 ms, slice thickness=2 mm, flip angle=90°, matrix size=480×480). T1-weighted images were additionally acquired (TR=500.0 ms, TE=10.0 ms, slice thickness=5.0 mm, flip angle=90°, matrix size=512×512). For whole-brain diffusion tensor imaging (DTI), sets of axial diffusion-weighted single-shot echo-planar images were collected along 45 different directions, including a baseline image without diffusion weighting (b=0 s/mm^2), utilizing a b-factor of 600 s/mm^2.

The total volume of WMH in the brain was automatically quantified using the FLAIR and T1-weighted MRI data. Specifically, WMH areas displaying higher intensity than normal white matter on FLAIR images were segmented using the FMRIB Automatic Segmentation Tool within the FSL software suite (University of Oxford, Oxford, UK). To correct for considerable overlaps in signal intensities, false WMH regions were automatically eliminated using a white matter mask defined from the T1-weighted images.

For DTI data processing, the raw datasets were initially corrected for motion and eddy current distortions via affine registration using the FDT tool in FSL. Fractional anisotropy (FA) and mean diffusivity (MD) maps were subsequently generated. Customized template creation and spatial preprocessing were performed using voxel-based morphometry (VBM) implemented in Statistical Parametric Mapping (SPM5). The DTI volumes were spatially normalized to a customized template based on the MNI 152 standard brain. Finally, the registered FA and MD images were smoothed with an 8 mm full-width at half-maximum isotropic Gaussian kernel to prepare for statistical evaluation.

Statistical analysis

To compare the swallowing characteristics between SVCI and normal controls, *t*-test was performed.

Imaging analysis was conducted only with the SVCI data where voxel-based statistical analyses of the FA and MD maps were conducted using SPM5 to identify correlations between diffusion tensor indices and the quantified clinical swallowing scores. To account for potential confounding factors, age and education were entered as covariates for cognition-related analyses, while age alone was utilized as a covariate for motor-related analyses. The significances of the imaging analysis were set at corrected 0.05 or uncorrected $p<0.001$. To rigorously protect against type I errors, only clusters comprising more than 20 contiguous voxels were considered statistically significant.

RESULTS

Characteristics of the study participants

The study included a total of 105 participants, comprising 85 patients with SVCI and 20 normal controls. The SVCI group consisted 85 patients (mean age 73.65±6.89 years; 29 males, 56 females). Out of 85 patients, 51 were patients SVMCI (mean age 73.03±7.31 years; 18 males, 35 females) and 32 patients with SVaD (mean age 74.68±6.00 years; 11 males, 21 females). The normal control group had a mean age of 71.45±5.65 years (8 males, 12 females). The mean Mini-Mental State Examination scores were 23.03±5.29 for the SVCI group

https://doi.org/10.12779/dnd.2026.25.2.103

(25.26±5.29 for the SVMCI group, 19.03±5.00 for the SVaD group), and 28.15±1.30 for the normal controls. Correspondingly, the mean Clinical Dementia Rating (CDR) scores were 0.74±0.45 for the SVCI (0.53±0.19 for SVMCI, 1.09±0.53 for SVaD), and 0 for the normal controls (**Table 1**).

Swallowing characteristics: SVCI vs. normal controls

The results of the clinical swallowing characteristics are as follows. Out of the 6 components of the oral stage, there were significant differences between the SVCI patients and control groups in the following characteristics: tongue control during bolus hold (t=4.879, $p<0.001$), bolus preparation/mastication (t=3.213, $p=0.002$), bolus transport/lingual motion (t=4.513, $p<0.001$), and oral residue (t=2.705, $p=0.008$). Out of the nine components of the pharyngeal stage, there were significant differences between the two groups in the following: anterior hyoid excursion (t=4.102, $p<0.001$), epiglottic movement (t=4.396, $p<0.001$), laryngeal vestibular closure(t=2.023, $p=0.046$), tongue base retraction (t=−3.320, $p=0.002$), vallecular residue (t=2.365, $p=0.023$), pyriform sinus residue (t=4.424, $p<0.001$), pharyngeal esophageal segment opening (t=3.355, $p=0.001$). There were no significant differences between the two groups in the PAS scores (**Table 2**).

The results of the subclinical swallowing measurements via temporal measurement analysis are as follows. For the 'bolus transit' characteristics, there were no significant differences between the 2 groups for OTT but significant differences in PTT (t=3.331, $p=0.001$). In the subdivision of PTT, there was significant differences in the OPT (t=2.038, $p=0.044$). Results for the 'swallowing gestures' characteristics, there were significant differences between the patient and normal groups in all of the temporal measurement points, which are SPstart (t=2.496, $p=0.022$), SPclose (t=2.328, $p=0.031$), AEstart (t=2.497, $p=0.014$), AEclose (t=3.021, $p=0.003$), hyoidST (t=3.369, $p=0.001$), hyoidMAX (t=2.390, $p=0.019$), UESopen (t=2.117, $p=0.037$). Time gap between the initiating and finishing swallowing gestures or the bolus flow and a certain swallowing gesture revealed significant differences in hyoidMAX-hypoidST (t=−3.648, $p<0.001$), hyoidMAX-AEclose (t=−5.687, $p<0.001$), and hyoidST-reachVALLE (t=3.003, $p=0.003$) (**Table 3**).

Table 2. Comparison of clinical and subclinical swallowing characteristics between SVCI and normal controls

Swallowing stages	Swallowing components	SVCI	Normal control	t	*p*-value
Oral stage	Lip closure	0.02±0.11	0.00±0.00	0.952	0.343
	Tongue control during bolus hold	0.29±0.35	0.08±0.09	4.879	<0.001***
	Bolus preparation/mastication	0.01±0.04	0.00±0.00	3.214	0.002**
	Bolus transport/lingual motion	0.39±0.74	0.01±0.08	4.513	<0.001***
	Oral residue	0.75±0.31	0.55±0.20	2.705	0.008**
	Initiation of pharyngeal swallow	1.12±0.83	0.83±0.87	1.359	0.186
Pharyngeal stage	Soft palate elevation	0.01±0.72	0.00±0.00	0.631	0.530
	Anterior hyoid excursion	0.14±0.32	0.00±0.00	4.102	<0.001***
	Epiglottic movement	0.43±0.69	0.05±0.19	4.396	<0.001***
	Laryngeal vestibular closure	0.02±0.10	0.00±0.00	2.023	0.046*
	Pharyngeal stripping wave	0.03±0.14	0.05±0.22	−0.331	0.741
	Tongue base retraction	0.73±0.59	0.43±0.29	−3.320	0.002**
	Vallecular residue	0.94±0.54	0.70±0.36	2.365	0.023*
	Pyriform sinus residue	0.62±0.37	0.37±0.17	4.424	<0.001***
	Pharyngeal esophageal segment opening	0.09±0.27	0.00±0.00	3.355	0.001**
Laryngeal stage	Penetration/aspiration	1.20±0.50	1.10±0.20	0.940	0.349

Values are presented as mean ± standard deviation.
SVCI: subcortical vascular cognitive impairment.
*$p<0.05$, **$p<0.01$, ***$p<0.001$.

https://doi.org/10.12779/dnd.2026.25.2.103

Table 3. Comparison of temporal measurements of swallowing between SVCI and normal control groups

Measurement characteristics	Measurement points	SVCI	Normal control	t	*p*-value
Bolus transit	OTT	0.553±0.476	0.504±0.299	0.437	0.663
	PTT	1.484±0.721	1.122±0.336	3.331	0.001**
	OPT	0.551±0.416	0.353±0.246	2.038	0.044*
	LPT	0.932±0.623	0.769±0.312	1.683	0.098
Swallowing gestures	SPstart	0.586±0.594	−0.612±2.129	2.496	0.022*
	SPclose	0.768±0.676	−0.329±2.083	2.328	0.031*
	AEstart	0.808±0.776	0.097±2.107	2.497	0.014*
	AEclose	0.990±0.788	0.197±1.817	3.021	0.003**
	hyoidST	0.939±0.832	0.026±1.838	3.369	0.001**
	hyoidMAX	1.057±0.776	0.434±1.820	2.390	0.019*
	UESopen	1.036±0.800	0.649±0.327	2.117	0.037*
Time interval between measurement points	SPclose-SPstart	0.182±0.190	0.304±0263	−1.960	0.053
	AEclose-AEstart	0.183±0.091	0.116±0.816	0.395	0.697
	hyoidMAX-hyoidST	0.119±0.337	0.407±0.191	−3.648	<0.001***
	hyoidST-AEstart	0.131±0.421	−0.072±0.844	1.658	0.100
	hyoidMAX-AEclose	0.067±0.114	0.219±0.129	−5.687	<0.001***
	hyoidST-reachVALLE	0.447±0.738	−0.271±1.612	3.003	0.003**
	exitUES-openUES	0.413±0.304	0.455±0.100	−0.875	0.384
	reachPYR-openUES	−0.145±0.265	−0.122±0.236	−0.222	0.825
	openUES-hyoidMAX	−0.021±0.197	0.144±1.545	−0.598	0.557

Values are presented as mean ± standard deviation.
SVCI: subcortical vascular cognitive impairment, OTT: oral transit time (time from the initiation of tongue movement to begin the voluntary oral stage until the stage where the bolus head reaches the posterior nasal spine [B1]), PTT: pharyngeal transit time (time from B1 to the point where bolus tail exits the upper esophageal sphincter, OPT: oropharyngeal transit (time from B1 to the point where bolus head reaches vallecula), LPT: laryngo-pharyngeal transit (time from bolus head reahcing vallecuale to bolus tail exiting upper esophgeal sphincter), SPstart: soft palate starts to elevate, SPclose: soft palate reached maximum closure, AEstart: initation of aryepiglottic elevation, AEclose: aryepiglottic fold elevation to the point of supraglottic closure, hyoidST: initial movement of hyoid elevation, hyoidMAX: hyoid reaches maximum displacement, valleRE: bolus head reaching vallecula, pyriformRE: bolus head reaching pyriform sinus, UESopen: onset of upper esophgeal sphincter opening, UESexit: bolus tail exits upper esophgeal sphincter.
*p<0.05, **p<0.01, ***p<0.001.

Neuroanatomical correlates of swallowing (DTI-VBM)

The MD map corresponding to OTT revealed significant microstructural changes in the right corona radiata and the anterior cingulate cortex (uncorrected, p<0.001, k=100) (**Fig. 2**).

For the PTT, both FA and MD maps demonstrated significant diffusion alterations across widespread regions. These regions included the right anterior insula, anterior cingulate

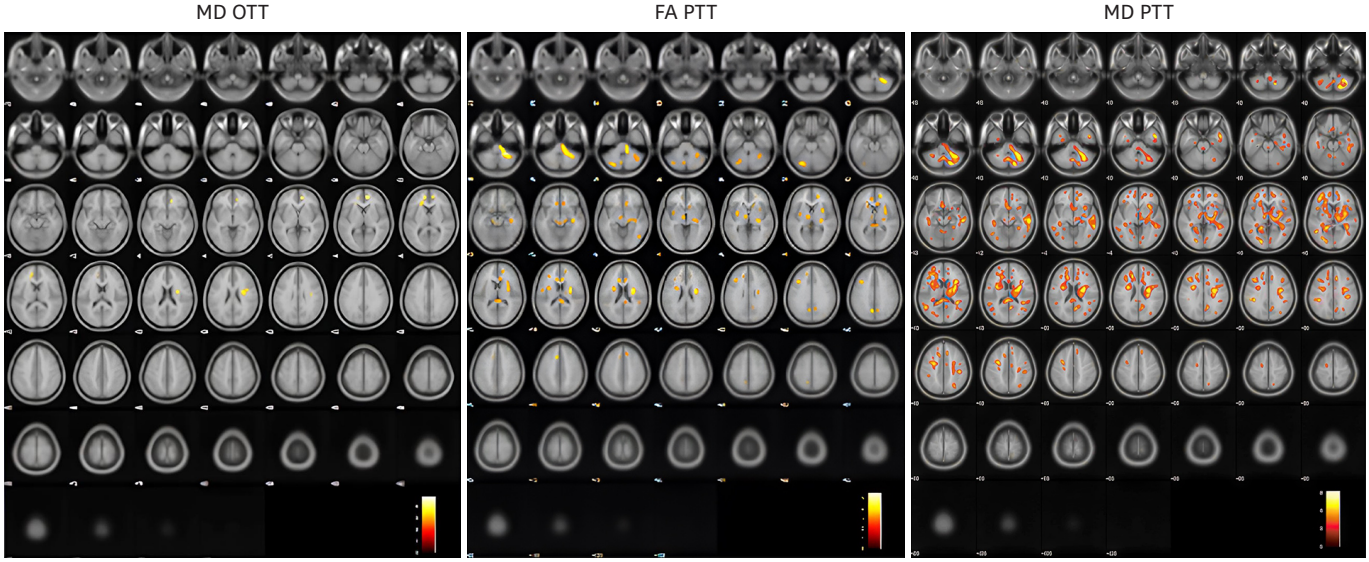

Fig. 2. DTI-VBM maps for OTT and overall PTTs.
DTI-VBM: diffusion tensor imaging-voxel-based morphometry, OTT: oral transit time, PTT: pharyngeal transit time, FA: fractional anisotropy, MD: mean diffusivity.

https://doi.org/10.12779/dnd.2026.25.2.103

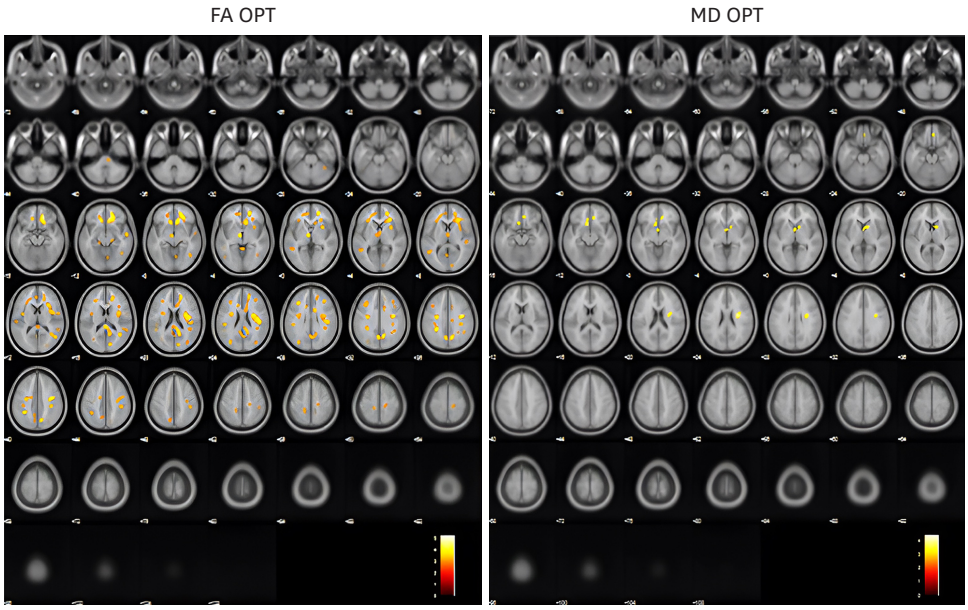

Fig. 3. DTI-VBM maps for OPT.
DTI-VBM: diffusion tensor imaging-voxel-based morphometry, OPT: oropharyngeal transit, FA: fractional anisotropy, MD: mean diffusivity.

cortex, bilateral corona radiata, corpus callosum (anterior body and splenium), internal capsule (anterior and posterior limbs), right caudate nucleus, bilateral thalamus (right<left), midbrain, pons, and the upper medulla. The bilateral cerebellum and right pontocerebellar circuits were also significantly involved (corrected, $p<0.05$) (**Fig. 2**).

The FA and MD map of the OPT designated by bolus flow from B1 to valleRE revealed significant diffusion in the right anterior insula, anterior cingulate cortex, bilateral corona radiata (right>left), corpus callosum (anterior, splenium), anterior limb of the internal capsule, right caudate nucleus, left thalamus and the midbrain (corrected, $p<0.05$) (**Fig. 3**).

The FA and MD map of the LPT designated by bolus flow from valleRE to UESexit reveals areas of dysfunction in the bilateral corona radiata (right>left), right posterior limb of the internal capsule, bilateral anterior insula, corpus callosum (anterior and splenium), bilateral thalamus, midbrain, pons, cerebellum and right pontocerebellar circuit (corrected, $p<0.05$) (**Fig. 4**).

DISCUSSION

In this study, we evaluated the swallowing characteristics between patients with SVCI and normal controls and examined the underlying neuroanatomical correlates of the swallowing functions in patients with SVCI using DTI-VBM. Our major findings were as follows. First, SVCI patients exhibited clinical swallowing characteristics involving both the oral and most of the pharyngeal stage of swallow. Second, subclinical characteristics evaluated through temporal measurements, revealed swallowing impairments predominantly characterized by significant delayed bolus flow in the pharyngeal stage and delayed swallowing gestures of the overall pharyngeal stage of swallow. Time gap results showed shorter time was taken for the hyoid movement to start and finish, delayed aryepiglottic closure relative to the

https://doi.org/10.12779/dnd.2026.25.2.103

DND Dementia and Neurocognitive Disorder

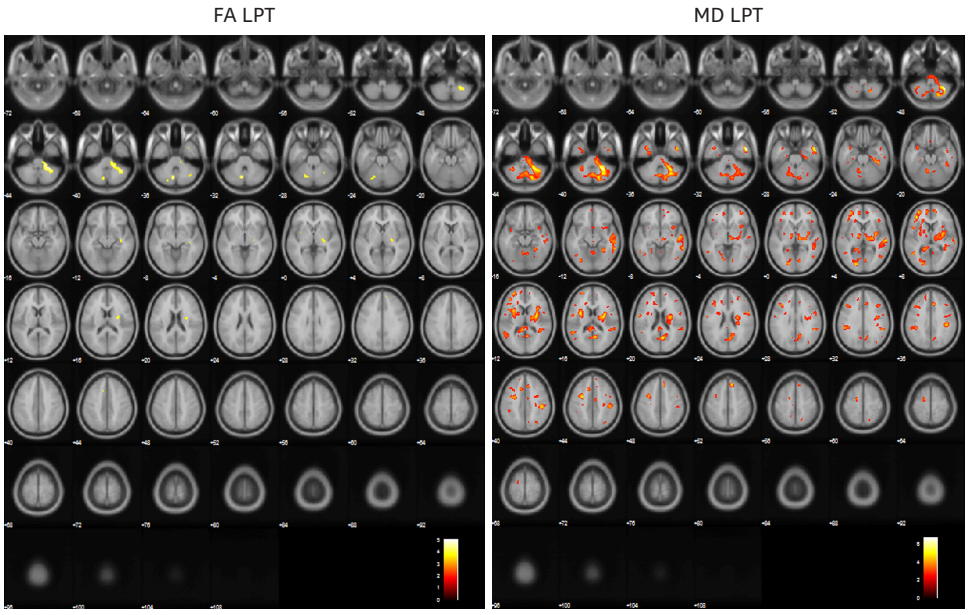

Fig. 4. DTI-VBM maps for LPT.
DTI-VBM: diffusion tensor imaging-voxel-based morphometry, LPT: laryngo-pharyngeal transit, FA: fractional anisotropy, MD: mean diffusivity.

hyoid maximal displacement, and delayed initiation of the starting movement of the hyoid relative to the bolus flow reaching the vallecula. Finally, these swallowing patterns of the patients significantly correlated with widespread microstructural disruptions across both supratentorial and infratentorial networks. Taken together, these findings confirm that dysphagia in SVCI is driven by extensive neural network disruptions, emphasizing the need for early and systematic clinical monitoring.

The clinical swallowing characteristics manifested in our study, show both reduction in the oral and the pharyngeal stage of swallow. Regarding the oral stage, significant differences were in 'tongue control during bolus hold,' 'bolus hold and mastication,' 'bolus preparation,' and 'mastication and bolus transport/lingual motions' all of which required lingual movements. This result is in line with the previous report of patients with lesions in the periventricular white matter manifesting swallowing deficits involving oral control and transfer.[30] Subclinical temporal data of our study show that there were no significant differences in OTT, which is in line with the previous result that the patients with vascular dementia manifested deficits in bolus formation and mastication and no oral transit delay over 5 seconds.[11] Combining the clinical and subclinical results together, it may indicate that SVCI patients that have ischemic changes manifest reduced movement range and/or strength of the lingual area but for the patients in our study it was not severe enough to affect the temporal aspect of the bolus transport.

There were also significant differences indicating reduced movement range in major pharyngeal stages of swallow which include the anterior hyoid, epiglottis, laryngeal vestibular closing movement, tongue base retraction, pharyngeal esophageal segment opening movements resulting in residue pooling in the vallecula and pyriform sinus. These results are in line with the previous report that patients with vascular dementia manifested reduced hyo-laryngeal excursion, epiglottic inversion.[11] Temporal analysis revealed delayed

https://doi.org/10.12779/dnd.2026.25.2.103

bolus transit in the PTT and when PTT was subdivided into the OPT and LPT, OPT was significantly delayed. Retraction of the tongue base provides thrust to propel the bolus into the pharynx, reduced lingual movements and reduced tongue base retraction in SVCI patients could have delayed the bolus flow in the OPT area. There was also delay in the initiation and finishing movements of all the swallowing gestures in the pharyngeal stage including SPstart, SPclose, AEstart, AEclose, hyoidST, hyoidMAX, and the UES open. As soft palate elevation is the initiating movement of the pharyngeal stage of swallow, it is only natural that all of the following swallowing gestures of the pharyngeal stage is delayed when compared to the normal controls. However, time gap between the initiating and finishing swallowing gestures as well as the bolus flow and a certain swallowing gesture show results that could be characteristic of the SVCI patients. The first result is that it took shorter time from the starting movement the hyoid to the point where it reaches the maximal displacement (hyoidMAX-hyoidST) in the SVCI group, which could be interpreted as reduced range of hyoid movement in the patient group, as was also indicated in the clinical results. The second result is that the time gap between the maximal hyoid displacement and complete aryepiglottic closure (hyoidMAX-AEclose) is shorter for the SVCI patients compared to the controls. This could indicate that although the hyoid displacement has reached its' peak stage, the completion of aryeiglottic closure is delayed in the SVCI group. Thirdly, the time gap between the initiating movement of the hyoid and the time the bolus reaches vallecula show that in the normal controls the hyoid movement is initiated before the bolus reaches the vallecula whereas in the patient group, the swallowing gesture is initiated after the bolus reaches the vallecula.

There were no significant differences between the SVCI and the normal controls in the PAS scores. Our study included SVMCI patient who are on the earlier stage of the disease, and the mean CDR of the SVCI patients in our study were 0.78 (±0.55), therefore, although our patients started showing swallowing changes, they may have not yet reached the disease severity to the point of provoking aspiration.

Neuroimaging results revealed that the unique dysphagia patterns in SVCI significantly correlated with widespread microstructural disruptions across both supratentorial and infratentorial networks. Both oral and pharyngeal transits were associated with the corona radiata and the anterior cingulate cortex, confirming the reliance on corticobulbar fibers[31] and the necessity of spontaneity for the oral stage.[32] Subdividing the pharyngeal stage revealed that the OPT was associated with the caudate nucleus, whereas the LPT correlated with the putamen, globus pallidus, and cerebellar control circuits.[33] This underscores that the laryngo-pharyngeal stage, paramount for airway protection, demands rigorous smooth and timely control from frontal subcortical and cerebellar circuits. Lastly, the upper medulla was exclusively involved in the LPT, indicating that disruptions here, alongside compromised corticobulbar tracts, delay sensory processing and induce the subclinical slowing of swallowing gestures.

The present study demonstrated the detailed clinical swallowing characteristics and precise neuroanatomical correlates of swallowing in SVCI. Given that these subclinical delays manifest early due to progressive white matter ischemic changes, recognizing this vulnerability provides a crucial window for proactive monitoring. These findings provide essential evidence supporting the necessity of early swallowing assessments in SVCI patients to prevent severe complications such as aspiration pneumonia.

https://doi.org/10.12779/dnd.2026.25.2.103

Strengths of this study include the systematic investigation of dysphagia in a well-characterized SVCI cohort; the dual approach utilizing both clinical scales (MBSImP and PAS) and subclinical temporal measurements; and the stage-by-stage neuroanatomical matching using advanced DTI-VBM techniques. Limitations merit acknowledgment. First, our cross-sectional design limits causal inferences regarding the progression of dysphagia over time. Second, further longitudinal studies and comparisons with other dementia subtypes (e.g., Alzheimer's disease) are required to clarify the specificity of these swallowing profiles. Nonetheless, these results provide robust evidence mapping the clinical phenomenology of SVCI dysphagia to its underlying neural damage.

In conclusion, dysphagia in SVCI involves reduced oral stage functions involving the lingual movement. In the pharyngeal stage, movement range of most of the major pharyngeal structures were reduced but was primarily characterized by reduced hyoid displacement range, delayed initiating movement of the hyoid as well as delay in aryepiglottic closure at the point of maximal hyoid displacement resulting from widespread corticobulbar, frontal subcortical, and cerebellar disruptions. The identification of clinical and subclinical swallowing impairments in the early stages SVCI patients may help clinicians evaluate and predict the pattern of dysphagia as the disease progresses.

REFERENCES

1. Román GC, Tatemichi TK, Erkinjuntti T, Cummings JL, Masdeu JC, Garcia JH, et al. Vascular dementia: diagnostic criteria for research studies. Report of the NINDS-AIREN International Workshop. Neurology 1993;43:250-260. PUBMED | CROSSREF
2. Román GC, Erkinjuntti T, Wallin A, Pantoni L, Chui HC. Subcortical ischaemic vascular dementia. Lancet Neurol 2002;1:426-436. PUBMED | CROSSREF
3. Petersen RC, Smith GE, Waring SC, Ivnik RJ, Tangalos EG, Kokmen E. Mild cognitive impairment: clinical characterization and outcome. Arch Neurol 1999;56:303-308. PUBMED | CROSSREF
4. Ku B, Lee J, Na D. The demographic and clinical characteristics of subcortical vascular dementia: an analysis of 125 patients. J Neurol Sci 2009;283:306. CROSSREF
5. Staekenborg SS, van der Flier WM, van Straaten EC, Lane R, Barkhof F, Scheltens P. Neurological signs in relation to type of cerebrovascular disease in vascular dementia. Stroke 2008;39:317-322. PUBMED | CROSSREF
6. Wallin A, Milos V, Sjögren M, Pantoni L, Erkinjuntti T. Classification and subtypes of vascular dementia. Int Psychogeriatr 2003;15 Suppl 1:27-37. PUBMED | CROSSREF
7. Logemann JA. *Evaluation and Treatment of Swallowing Disorders*. 2nd ed. Austin: Pro-Ed, 1998.
8. Logemann JA. Swallowing physiology and pathophysiology. Otolaryngol Clin North Am 1988;21:613-623. PUBMED | CROSSREF
9. Stach CB. Vascular dementia and dysphagia. Top Stroke Rehabil 2000;7:1-10. CROSSREF
10. Feinberg MJ, Ekberg O, Segall L, Tully J. Deglutition in elderly patients with dementia: findings of videofluorographic evaluation and impact on staging and management. Radiology 1992;183:811-814. PUBMED | CROSSREF
11. Suh MK, Kim H, Na DL. Dysphagia in patients with dementia: Alzheimer versus vascular. Alzheimer Dis Assoc Disord 2009;23:178-184. PUBMED | CROSSREF
12. Alexander GE, Crutcher MD. Functional architecture of basal ganglia circuits: neural substrates of parallel processing. Trends Neurosci 1990;13:266-271. PUBMED | CROSSREF
13. Cummings JL. Frontal-subcortical circuits and human behavior. Arch Neurol 1993;50:873-880. PUBMED | CROSSREF
14. Kessler JP, Jean A. Identification of the medullary swallowing regions in the rat. Exp Brain Res 1985;57:256-263. PUBMED | CROSSREF
15. Jean A, Car A. Inputs to the swallowing medullary neurons from the peripheral afferent fibers and the swallowing cortical area. Brain Res 1979;178:567-572. PUBMED | CROSSREF

16. Carpenter MB. *Core Text of Neuroanatomy*. 1st ed. Baltimore: Williams & Wilkins, 1978.
17. Amri M, Car A, Roman C. Axonal branching of medullary swallowing neurons projecting on the trigeminal and hypoglossal motor nuclei: demonstration by electrophysiological and fluorescent double labeling techniques. Exp Brain Res 1990;81:384-390. PUBMED | CROSSREF
18. Jean A, Amri M, Calas A. Connections between the ventral medullary swallowing area and the trigeminal motor nucleus of the sheep studied by tracing techniques. J Auton Nerv Syst 1983;7:87-96. PUBMED | CROSSREF
19. Roman C. Neural control of deglutition and esophageal motility in mammals. J Physiol (Paris) 1986;81:118-131. PUBMED
20. Robbins J, Levine RL, Maser A, Rosenbek JC, Kempster GB. Swallowing after unilateral stroke of the cerebral cortex. Arch Phys Med Rehabil 1993;74:1295-1300. PUBMED | CROSSREF
21. Daniels SK, Foundas AL, Iglesia GC, Sullivan MA. Lesion site in unilateral stroke patients with dysphagia. J Stroke Cerebrovasc Dis 1996;6:30-34. PUBMED | CROSSREF
22. Daniels SK, Foundas AL. The role of the insular cortex in dysphagia. Dysphagia 1997;12:146-156. PUBMED | CROSSREF
23. Logemann JA, Shanahan T, Rademaker AW, Kahrilas PJ, Lazar R, Halper A. Oropharyngeal swallowing after stroke in the left basal ganglion/internal capsule. Dysphagia 1993;8:230-234. PUBMED | CROSSREF
24. Mosier KM, Liu WC, Maldjian JA, Shah R, Modi B. Lateralization of cortical function in swallowing: a functional MR imaging study. AJNR Am J Neuroradiol 1999;20:1520-1526. PUBMED
25. Zald DH, Pardo JV. The functional neuroanatomy of voluntary swallowing. Ann Neurol 1999;46:281-286. PUBMED | CROSSREF
26. Erkinjuntti T, Inzitari D, Pantoni L, Wallin A, Scheltens P, Rockwood K, et al. Research criteria for subcortical vascular dementia in clinical trials. J Neural Transm Suppl 2000;59:23-30. PUBMED | CROSSREF
27. Martin-Harris B, Brodsky MB, Michel Y, Castell DO, Schleicher M, Sandidge J, et al. MBS measurement tool for swallow impairment--MBSImP: establishing a standard. Dysphagia 2008;23:392-405. PUBMED | CROSSREF
28. Rosenbek JC, Robbins JA, Roecker EB, Coyle JL, Wood JL. A penetration-aspiration scale. Dysphagia 1996;11:93-98. PUBMED | CROSSREF
29. Kendall KA, McKenzie S, Leonard RJ, Gonçalves MI, Walker A. Timing of events in normal swallowing: a videofluoroscopic study. Dysphagia 2000;15:74-83. PUBMED | CROSSREF
30. Cola MG, Daniels SK, Corey DM, Lemen LC, Romero M, Foundas AL. Relevance of subcortical stroke in dysphagia. Stroke 2010;41:482-486. PUBMED | CROSSREF
31. Kim H. *Neuroanatomy for Speech-Language Pathology*. Seoul: Sigma Press, 2009.
32. Martin RE, Sessle BJ. The role of the cerebral cortex in swallowing. Dysphagia 1993;8:195-202. PUBMED | CROSSREF
33. Benarroch EE. *Basic Neuroscience with Clinical Applications*. Philadelphia: Butterworth-Heinemann, 2006.