



Research article

The pulvinar nucleus is associated with the presence of dysarthria in patients with basal ganglia hemorrhage



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HIGHLIGHTS

- Dysarthria is a frequent symptom in patients with basal ganglia hemorrhage (BGH).
- The left pulvinar nucleus is related to the dysarthria in patients with BGH.
- The right pulvinar nucleus is also associated with the presence of dysarthria.

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ABSTRACT

Dysarthria is a frequent symptom in patients with stroke. The anatomical structures responsible for dysarthria have been reported in patients with lacunar infarcts, but the related lesions in patients with basal ganglia hemorrhage (BGH) have not been investigated. The aim of this study was to identify associations between the lesion location and the presence/absence of dysarthria in patients with BGH using voxel-based lesion symptom mapping (VLSM) analyses. A retrospective analysis was conducted on 26 patients with acute BGH (mean age, 54.0 years; men:women, 14:12) who underwent conservative management. The patients were classified into groups based on the presence or absence of dysarthria at the time of admission, which was determined by reviewing the patients' medical records. Brain lesions were traced on magnetic resonance images that were acquired within the first 3 weeks after BGH onset, and then separate high-resolution region-of-interest images were generated. Associations between dysarthria and the lesion location were determined with the VLSM analyses. The average volume of the delimited lesions was $7.38 \pm 5.75 \text{ cm}^3$. The VLSM analyses identified several voxel clusters, mainly in the pulvinar nucleus of the left thalamus, that were significantly related to the presence of dysarthria at admission. These findings suggest that patients with BGH extending into the left pulvinar nucleus should be monitored for dysarthria.

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Abbreviations: BGH, basal ganglia hemorrhage; MRI, magnetic resonance imaging; VLSM, voxel-based lesion symptom mapping; Duration, duration from onset of basal ganglia hemorrhage to magnetic resonance image acquisition; AH, affected hemisphere; Volume, volume of the basal ganglia hemorrhage on magnetic resonance images; MRI year, year of magnetic resonance image acquisition; F, female; M, male; Rt., right; Lt., left; Y, yes; N, no; MNI, Montreal Neurological Institute.

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1. Introduction

Dysarthria is a motor disorder characterized by dysfunction of the structures implicated in the control, initiation, and coordination of speech output [4]. Dysarthria occurs in about 40% of stroke cases with lesions along the speech area of the motor system [8]. Structures related to the motor aspects of speech production are extensively involved, such as the upper and lower motor neurons, extrapyramidal system, peripheral nerves, and orofacial muscles. Most previous dysarthria studies examined the anatomical structures related to the vocal regions of the motor system, which are thought to be in the precentral gyrus or corticobulbar tract and cerebellum [12,15,19,21,23]. However, the regions within the

Table 1
General characteristics of the included subjects.

Patient	Sex	Age (years)	Duration (days)	AH	Volume (cm ³)	Dysarthria at admission	Dysarthria at discharge	MRI year
1	F	83	2	Rt.	1.68	Y	Y	2010
2	M	59	18	Lt.	1.54	N	N	2007
3	M	34	12	Lt.	11.36	Y	N	2008
4	M	70	1	Lt.	5.63	Y	Y	2008
5	F	67	4	Lt.	3.34	Y	N	2013
6	F	50	14	Lt.	2.59	Y	N	2007
7	M	62	6	Lt.	3.97	Y	N	2014
8	M	62	3	Lt.	1.09	N	N	2014
9	M	49	19	Rt.	23.12	N	N	2006
10	M	56	5	Lt.	7.73	N	N	2009
11	M	41	16	Rt.	10.95	Y	N	2007
12	M	61	15	Rt.	7.90	Y	N	2007
13	F	47	17	Rt.	12.50	Y	N	2007
14	M	51	5	Rt.	3.00	Y	N	2007
15	F	57	6	Lt.	6.43	N	N	2008
16	F	58	8	Lt.	12.95	Y	N	2012
17	F	53	1	Lt.	1.61	Y	N	2012
18	M	53	0	Lt.	3.58	Y	Y	2014
19	F	53	6	Lt.	4.10	N	N	2009
20	M	56	0	Rt.	0.97	N	N	2012
21	F	59	16	Lt.	12.62	N	N	2012
22	M	67	2	Rt.	0.96	Y	Y	2013
23	F	75	10	Lt.	11.34	N	N	2012
24	M	58	4	Rt.	16.37	N	N	2014
25	F	89	3	Rt.	12.40	N	N	2014
26	F	54	12	Rt.	12.26	N	N	2014

extrapyramidal system that are specifically related to dysarthria have not been well investigated.

The extrapyramidal system and its indirect activation pathways are complex and include the basal ganglia, thalamus, cerebellum, and even the cerebral cortex. For this reason, the roles individual structures play in the neural control of speech are unclear. Previous studies of dysarthria associated with extrapyramidal lesions have been described, especially in connection with parkinsonian syndromes [2,4,11]. However, it is difficult to clarify the lesioned areas in Parkinson's disease and parkinsonian syndromes because various regions of the brain may be involved. In contrast, the injured region is clearly defined in patients with basal ganglia hemorrhage (BGH). Furthermore, dysarthria has been frequently reported in cases with basal ganglia lesions [4,5]. Thus, the present study was conducted to evaluate the relationships between the BGH lesion location and the presence/absence of dysarthria using voxel-based lesion symptom mapping (VLSM).

2. Methods

2.1. Subjects

This study retrospectively reviewed the medical records of patients with BGH who were admitted to our hospital between January 2006 and December 2014. Twenty-six patients with BGH who met the below inclusion and exclusion criteria were finally enrolled from among the 256 potential subjects. The inclusion criteria were as follows: (1) confirmation of BGH with computed tomography or magnetic resonance imaging (MRI), (2) first-ever stroke, (3) MRI conducted within 3 weeks after BGH, and (4) the presence/absence of BGH was absolutely confirmed within the first 24 h after BGH onset according to the patient's medical records. All patients had been examined clinically by psychiatrists, who clinically diagnosed/confirmed the presence of dysarthria. This information was then retrieved from the medical records of each subject to determine whether the subject had dysarthria at the time of admission and discharge. Exclusion criteria were as follows: (1) any coexisting neurological disease that could influence speech function, and (2) prior operation or catheter insertion for

the treatment of BGH. The institutional review board at our hospital approved the procedures and protocols of this study. The institutional review board committee waived the requirement for informed consent given the retrospective nature of the study.

2.2. Magnetic resonance image acquisition

All images were acquired with a 3-T clinical whole-body magnetic resonance scanner (GE Signa, Milwaukee, WI). Before 2012, 13 patients had their MRI scans acquired with a Discovery MR 750 scanner; after 2012, the other 13 patients had their images acquired using a Discovery SIGNA EXCITE scanner (Table 1). A high-resolution three-dimensional T1-weighted image was obtained in all patients. The imaging parameters for the images acquired before 2012 were as follows: repetition time/echo time = 6.76/6.76 ms, thickness = 1 mm, field of view = 210 × 210 mm², matrix = 256 × 256 (reconstructed to 512 × 512), flip angle = 20°, and reconstructed voxel size = 0.412 × 0.412 × 4 mm. The imaging parameters for the images acquired during and after 2012 were as follows: repetition time/echo time = 8.29/3.28 ms, thickness = 1 mm, field of view = 220 × 220 mm², matrix = 256 × 256 (reconstructed to 512 × 512), flip angle = 12°, and reconstructed voxel size = 0.430 × 0.430 × 1 mm.

2.3. Voxel-based lesion symptom mapping and statistical analyses

For the VLSM analysis, we performed the following procedures. First, the hemorrhagic lesion of each patient was drawn on the high-resolution T1-weighted image in the native space using the MRicro software (<http://www.mricro.com>). Second, each individual's T1-weighted image and native-space lesion image were nonlinearly transformed to the standardized Montreal Neurological Institute space using SPM8 (<http://www.fil.ion.ucl.ac.uk/spm>). Third, the presence/absence of dysarthria was converted to a binary mask image for the VLSM analysis. Finally, we performed a Pearson's chi-squared test at each voxel to identify dysarthria-related brain regions. The level of significance was set to $p < 0.005$ at each voxel,

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