

[CASE REPORT]

Aphasia with No Apparent Paralysis in Progressive Stroke of the Anterior Choroidal Artery

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Abstract:

Some anterior choroidal artery (AChA) infarctions in the posterior limbs of the internal capsule (pLIC) have been reported to cause aphasia, typically with apparent paralysis. We herein report an 84-year-old woman with AChA infarction. Although her dysarthria remained mild with no apparent paralysis, we overlooked progression to branch atheromatous disease-related infarct with exacerbation of her anomia, which delayed the initiation of more intense therapy. Even in AChA infarction, especially when the lesion is located mainly in the anterior part of the pLIC, as in our case, it is possible to encounter progressive stroke predominantly with aphasia.

Key words: aphasia, anterior choroidal artery, internal capsule, progressive stroke

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Introduction

Anterior choroidal artery (AChA) infarction can cause cognitive disorders by disconnecting the cortico-subcortical networks in the posterior limbs of the internal capsule (pLIC) (1). Most cases with aphasia have been reported to present with other severe symptoms, including paralysis from the onset (2-6).

However, in our case, the anomia worsened day by day, whereas other symptoms remained unremarkable in the acute phase. Our case showed that occlusion of the proximal branches of the superior branches in the cisternal segment of the AChA could cause progressive stroke with aphasia accompanied by no apparent paralysis (7).

Case Report

An 84-year-old, right-handed woman with a history of hypertension and no dementia presented with mild speech difficulty 2 days before admission. Her National Institutes of Health Stroke Scale (NIHSS) score was 2 [i.e. 1 in item 10 (Dysarthria) and 1 in item 9 (Best language)], where she was unable to name 1 out of 6 items in the picture. She had no apparent visual field deficit, paralysis, or sensory disturbance.

Ultrasonic echocardiography and 24-h Holter electrocardiogram (ECG) showed no significant abnormalities. Mag-

netic resonance angiography (MRA) showed no significant stenosis in the major arteries (Fig. 1). In the axial view of diffusion-weighted magnetic resonance imaging (DWI), the inferior part of the infarction was located mainly in the genu of the internal capsule (gIC) and the anterior part of the pLIC on the left side, and the superior part extended obliquely backward along the pLIC (Fig. 2a).

Treatment with dual antiplatelet therapy and edaravone was started. The dysarthria remained mild with no new symptoms, but the anomia worsened day by day. On day six of admission, she could not name 4 out of 6 items in the picture of the NIHSS, and the infarction expanded to a diameter of 20 mm and 3 consecutive slices on the transverse plane on DWI (Fig. 2b). Thus, argatroban therapy was added.

Her naming in the Japanese Standard Language Test of Aphasia was impaired more significantly for nouns than for verbs: the score for object naming was 9 out of 20, and that for verbal explanation of behaviors and movements was 8 out of 10. In the verbal explanation of the picture story, anomia and paraphasia were observed. Auditory comprehension was moderately impaired, whereas the word and sentence repetition were normal.

The Mini-Mental State Examination score was 17 and the Frontal Assessment Battery (FAB) score six on day 14. The standard profile score of The Rivermead Behavioral Memory Test (RBMT) score was 6 out of 24, but the Digit and Tapping spans were normal on day 17. The low scores in these

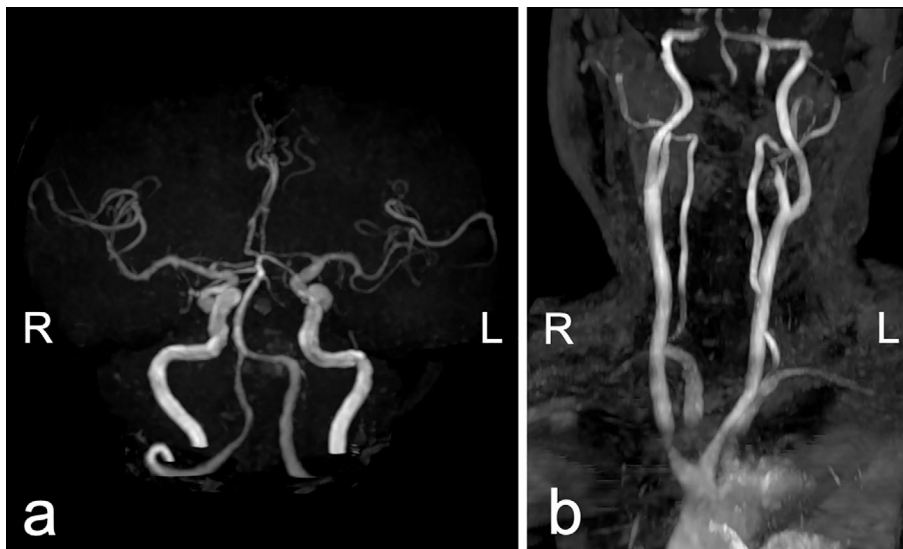


Figure 1. (a) Intracranial and (b) cervical magnetic resonance angiography (MRA) on day 1 (admission) showed no significant stenosis in the major arteries.

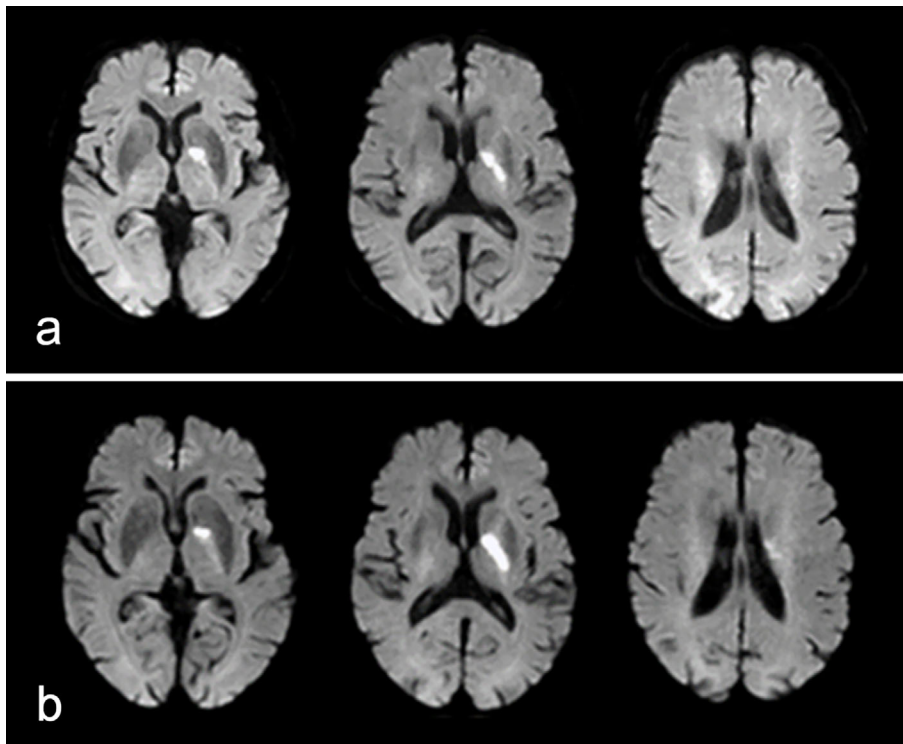


Figure 2. Axial views on diffusion-weighted imaging (slice thickness, 6 mm; interslice gap, 1 mm). (a) On day 1 (admission), high signal intensities were shown mainly in the genu of the internal capsule and the anterior part of the posterior limb of the internal capsule on the left side. (b) On day 6, the lesion expanded to the corona radiata.

neuropsychological examinations seemed to be partially due to her sentence comprehension deficit. ^{123}I -IMP single-photon emission computed tomography (SPECT) on day 19 revealed prominent hypoperfusion in the left prefrontal area (Fig. 3), which included Broca's area, as indicated by the stereotactic extraction estimation methods showing a high extent of Brodmann area 44 and 45 (Table). The Japanese test of lexical processing in aphasia on day 26 showed that

her naming was impaired invariably: the score for noun expression was 18 out of 40, and that for verb expression was 28 out of 40. The mean scores for noun and verb expression (-2 standard deviations) were 36.29 and 38.46, respectively, among normal individuals. One year later, her word-finding difficulty remained unchanged with no apparent dementia.

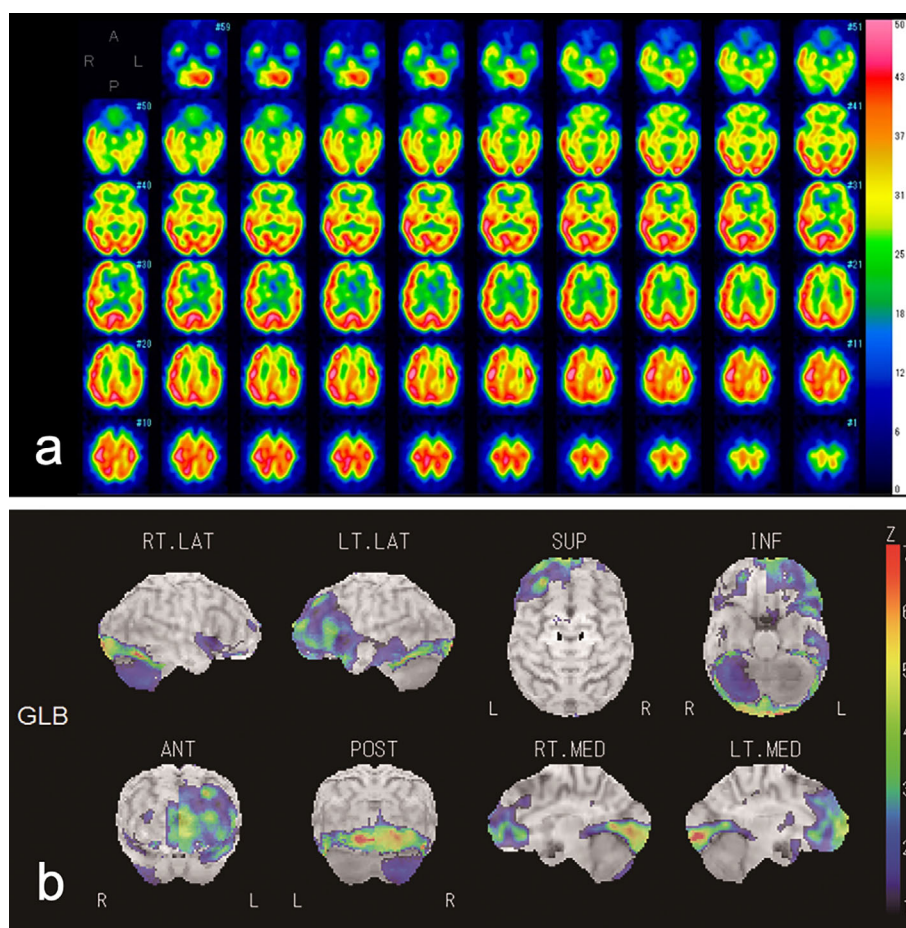


Figure 3. ^{123}I -IMP single photon emission computed tomography (SPECT) on day 19. (a) Original image and (b) three-dimensional stereotactic surface projection (3D-SSP) revealed prominent hypoperfusion in the left prefrontal and bilateral occipital areas.

Table. The Stereotactic Extraction Estimation Methods Analyzing the 3D-SSP Data (Z-score Decrease>2.00).

Level 5 Classification		Total projection	Extent		Severity	
			Ratio	Projection	Mean	SD
Brodmann area 44	Right	38	0.00%	0	Null	Null
	Left	38	57.90%	22	2.33	0.16
Brodmann area 45	Right	68	1.00%	0	Null	Null
	Left	68	77.90%	53	3.02	0.58

Discussion

The pLIC is supplied by the superior branches of the cisternal segment of the AChA, which are divided into distal and proximal branches (7). The posterior two-thirds of the pLIC and the retro-lenticular part of the internal capsule (rLIC) are supplied by the distal branches, and the anterior one-third of the pLIC and the gIC is supplied by the proximal branches (7). Although the anterior one-third can also be supplied by other arteries, such as the Heubner's or lenticulostriate artery, the infarction in our case was attributed mainly to occlusion of the proximal branches of the superior

branches in the cisternal segment of the AChA because of the extension pattern in the oblique direction among the pLIC (7).

The human language function can be regulated by several cortico-subcortical networks (8). The anterior complex loop (frontal association cortex - caudate - globus pallidus - ventral anterior nucleus thalami - frontal association cortex) may play a role in oral expression, and the anterior motor loop (sensor-motor cortex - putamen - globus pallidus - ventral lateral nucleus thalami - sensor-motor cortex) may be involved in articulation (8). These anterior loops pass through the superior thalamic radiation (STR) in the pLIC (9). The posterior loop (temporo-parietal cortex - pulvi-

nar nucleus thalami - temporo-parietal cortex) may be involved in auditory comprehension (8) and passes through the posterior thalamic radiation (PTR) in the rIIC (9). In previously reported cases with aphasia due to AChA infarction, the most common lesions, which were located in the posterior two-thirds of the pIIC, including the STR, probably disrupted the anterior loops and caused non-fluent speech and naming deficits (2-6). The lesions extending to the rIIC, including the PTR, tended to cause relatively severe deficits in auditory comprehension, probably due to additional disconnection of the posterior loop (4-6). These lesions supplied by the distal branches of the superior branches in the cisternal segment of the AChA also disrupted the corticospinal tract and the optic radiation, leading to paralysis and visual field deficit from the onset (2-6). Thus, the regimen could be easily established depending on the degree of these symptoms, not aphasia.

In our case, however, the lesion including the anterior part of the pIIC supplied by the proximal branches of the superior branches in the cisternal segment of the AChA disrupted the STR including the anterior complex loop, not the corticospinal tract, as suggested by the prominent hypoperfusion in the left prefrontal area on SPECT (8, 9). Overlooking progression to branch atheromatous disease-related infarct with aphasia accompanied by no apparent palsy may have led to a delay in the administration of argatroban (10, 11), probably resulting in her verbal sequela.

The present case report has several limitations. First, our patient was older than the previously reported cases (2-6). There is no denying that diffuse intracranial atherosclerosis related to aging may have exacerbated her cognitive impairment due to AChA infarction. Second, her language disturbance may have been affected by other cognitive dysfunctions. Her low FAB and RBMT scores may have been due to not only her sentence comprehension deficit but also executive and memory dysfunction arising from the gIC infarction (12). Such dysfunction might have affected her aphasia, especially anomia. Third, auditory comprehension in our patient was moderately impaired despite sparing of the PTR in the rIIC comprising the posterior loop, which may be involved in auditory comprehension (8, 9). Impairment of auditory sentence comprehension has been reported to be possibly caused by damage to Broca's area (13). In our case, disruption of the anterior complex loop in the STR due to the infarction may have led to hypoactivity in Broca's area included in the frontal association cortex as demonstrated by SPECT, resulting in a moderate deficit in her sentence comprehension. Finally, there is a possibility that non-convulsive status epilepticus (NCSE) may have influenced the aphasia in our case. Aphasia can be the sole symptom of NCSE, and this type of NCSE has been reported as aphasic status epilepticus (ASE) in various neuro-

logical disorders (14). We cannot completely deny that secondary cortical dysfunction due to AChA infarction may have given rise to ASE influencing exacerbation of her anomia as an acute symptomatic seizure.

In conclusion, selective occlusion of the proximal branches of the superior branches in the cisternal segment of the AChA can cause progressive stroke characterized by aphasia with no apparent palsy. Clinicians need to keep this in mind and initiate appropriate early treatment.

The authors state that they have no Conflict of Interest (COI).

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