

Dysgraphia due to anterior choroidal artery territory infarction: a case report

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Introduction

The anterior choroidal artery (AChA), which normally originates from the internal carotid artery and rarely from the middle cerebral artery, supplies a wide variety of structures, including the lateral thalamus and posterior internal capsule [1]. The most common neurological deficits associated with infarcts in the AChA region are hemiplegia, hemisensory loss, and homonymous hemianopsia, with neuropsychological and perceptual deficits occurring occasionally [2, 3]. Right-sided lesions could result in spatial neglect, whereas left-sided lesions could be the source of aphasia, which most often involves transcortical aphasia, constructive apraxia, and amnesic syndrome. However, dysgraphia after recovery from aphasia has rarely been reported. Here, we report a case of a patient with dysgraphia due to an AChA-region infarction.

Case report

The patient was a 64-year-old right-handed man, an office employee with 12 years of education, who presented with mild aphasia, right hemiplegia, impaired thermal nociception, and impaired deep sensation on his right side. On admission, he was alert but disoriented with regards to time and place.

His visual acuity and visual field were normal, and there were no other abnormalities in the central nervous system. He had received treatment for diabetes mellitus but had not received any psychotropic drugs. Initially, he experienced weakness in the right arm and leg and had difficulty speaking after lunch. At our hospital, he received conservative treatment after being diagnosed with cerebral infarction on the basis of magnetic resonance imaging (MRI) findings. The next day, he visited our department to receive rehabilitation.

Neuropsychological findings

Using the standard language test for aphasia, a formal language assessment was undertaken 3 days after stroke onset (Fig. 1a) and the following findings were noted: fluent spontaneous speech without paraphasia; intact repetition of spoken words; slight difficulty in visual recognition of words and aural comprehension at the sentence level, although he could read words aloud, whether written in Kanji (Chinese-based characters) or Kana (syllable-based characters); and difficulty in writing letters, characterized by absence of response to dictation. No other apraxia or agnosia was observed. Based on the Wechsler Adult Intelligence Scale Third Edition, his IQ assessment score was as follows: 83, verbal; 71, performance; 75, full IQ; 88, verbal comprehension; 85, perceptual organisation; 72, working memory; and 57, processing speed.

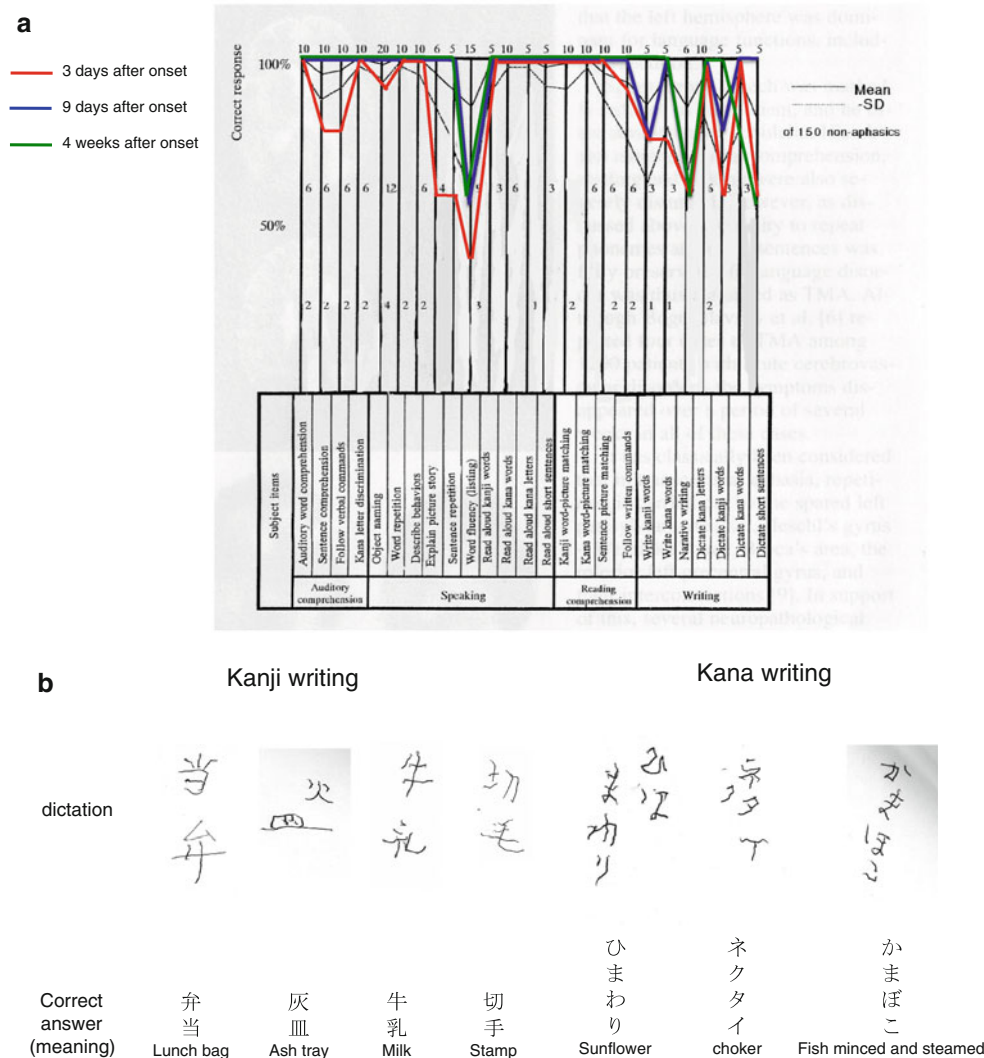
Neuroradiological findings

MRI on admission revealed cerebral infarction involving the left posterior limb of the internal capsule, corona radiata, and lateral thalamus (Fig. 2a). MR angiography of the head and neck revealed no stenosis or obstruction in the carotid artery and vertebral artery system. Six days after

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Fig. 1 **a** A standard language test for aphasia (SLTA) was performed 2 days, 9 days, and 4 weeks following the stroke onset. Aphasia was gradually resolved, with the patient's SLTA scores becoming normal in the areas of comprehension and oral language by 9 days after stroke onset. **b** Samples of dictation. This patient had difficulty in writing letters, characterized by absence of response to detection or paraphasia with misspellings



stroke onset, technetium-99 m L,L-ethyl cysteinate dimer (^{99m}Tc -ECD) single photon emission CT (SPECT) revealed a wider area of low perfusion in the left frontal and parietal lobes (Fig. 2b).

Clinical course

Aphasia was gradually resolved; by day 9, the patient's SLTA score for comprehension and oral language was normal (Fig. 1a). However, his writing ability remained clearly disturbed; errors included absence of response to dictation and paraphasia with misspellings. Although he could transcribe characters, generally in the correct stroke order, conversion of one character to another with a similar shape or sound, and omission of line(s) were observed (Fig. 1b). The selection and array of characters were normal.

Single photon emission CT performed 4 weeks after the stroke revealed improvement in the cerebral blood flow (CBF)

of the left frontal lobe; however, low perfusion persisted in parts of the frontal lobe and whole parietal lobes (Fig. 2c).

Discussion

In this case, the patient had intact oral language and reading comprehension, but continued to have severe writing difficulties 2 months after the stroke; he could transcribe characters, write them in the correct stroke order, and select an array of characters. He did not have apraxia or agnosia, and other cognitive functions remained relatively intact. In the absence of any dementia, aphasia, and disturbance of consciousness, his condition was classified as dysgraphia.

The mechanism underlying dysgraphia may involve secondary cortical impairments due to thalamic lesions. The thalamus and the cerebral cortex are connected by projection fibres, of which the posterior lateral nucleus and

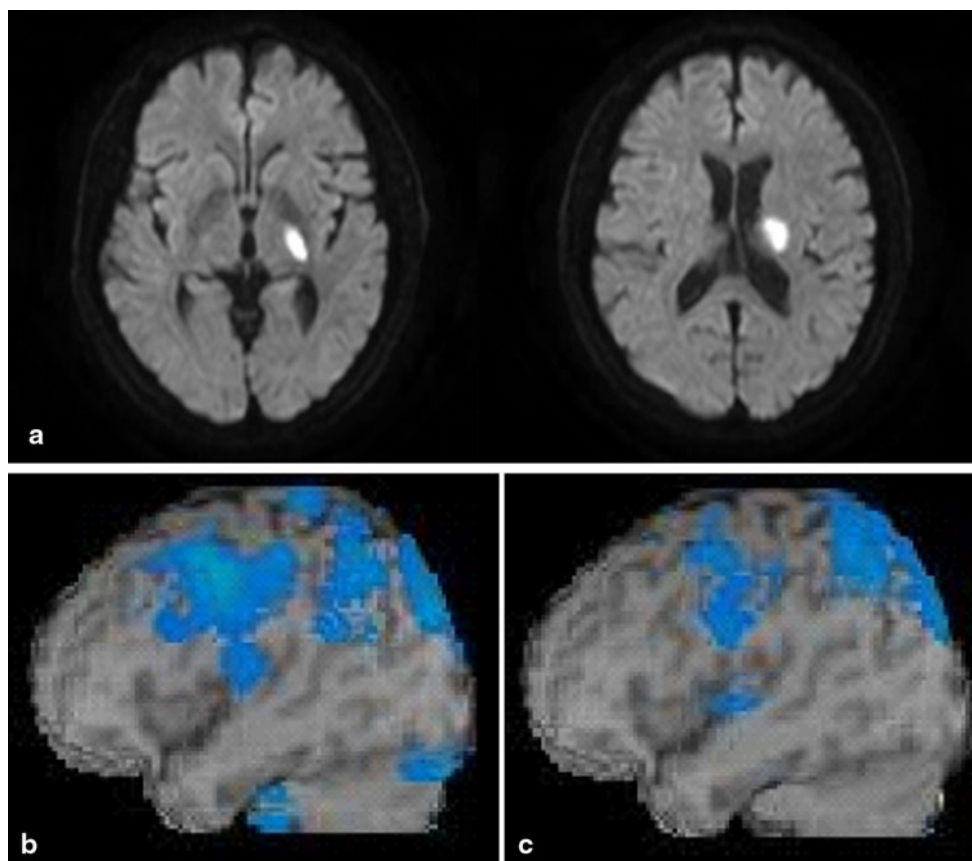


Fig. 2 Magnetic resonance imaging of the head revealed infarct lesions in the left posterior limb of the internal capsule, corona radiata, and lateral thalamus (**a**). A single photon emission computed tomography (SPECT) performed 6 days after the onset showed a

wider area of low perfusion in the frontal and parietal lobe (**b**). SPECT performed 4 weeks after the onset revealed an improvement in the cerebral blood flow of the frontal lobe; however, low perfusion persisted in parts of the frontal lobe and in whole parietal lobes (**c**)

the pulvinar nucleus of the lateral nuclear group connect the thalamus to the parietal lobe [4].

The dysgraphia observed in this patient is considered associated with the damage to these projection fibres, resulting from a thalamic lesion and causing a secondary decrease in left cerebral cortex function [5]. Although MRI revealed no abnormalities except for the finding in the left subcortical region, SPECT revealed a decrease in regional cerebral blood flow in the frontal and parietal cortex. This syndrome likely resulted from disconnection, since AChA infarct lesions typically involve the associative tracts joining subcortical structures to the cortex. AChA infarcts are responsible for reduction in metabolic activity and cerebral blood flow to the thalamus, lentiform nucleus, and dorsolateral hemispheric cortex [6], especially the frontal, rolandic, and parietotemporal cortex. This downstream consequence of AChA infarcts can contribute to cognitive disorders.

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