

Single Case – General Neurology

The Rehabilitation for Visual Cognitive Impairment due to Hippocampal Infarction: A Case Report

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Keywords

Visual cognitive impairment · Hippocampal infarction · Rehabilitation

Abstract

Introduction: Disturbances in the visual pathway cause visual cognitive impairment. There is a lack of information regarding the effect of rehabilitation on individuals affected by this condition. Therefore, it is crucial to understand the effectiveness of rehabilitation interventions in this condition. **Case Presentation:** We present the case of an 87-year-old woman with hippocampal infarction. While the patient's ability to perform daily activities and engage in conversations was normal, she faced challenges at the execution stage, such as naming, constructing sentences, and copying. We diagnosed cerebral embolism because of atrial fibrillation and initiated direct oral anticoagulant therapy. Subsequently, we initiated a rehabilitation treatment comprising visual agnosia training (attribute learning training), verbalization learning training (writing training), and semantic memory training (copying training) to address visual agnosia. **Conclusion:** Associative visual perception challenges arising from hippocampal infarcts are rare and can be improved with early intervention through a rehabilitation program for visual agnosia.

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Introduction

The posterior cerebral artery controls the hippocampus and embolisms, often leading to cerebral infarctions. The primary symptoms include memory loss and amnesia. Many cases of cerebral infarction are overlooked because they do not exhibit motor symptoms. Left-sided lesions

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commonly cause verbal memory impairment, whereas right-sided lesions lead to nonverbal memory impairment [1].

Visual agnosia is a disorder that disrupts the process of constructing a representation based on visual input and linking it with its meaning. Cavina et al. [2] classified visual agnosia symptoms into three categories: perceptual, integrated, and associative. Lesion analysis indicates that the bilateral medial occipital cortex is responsible for perceptual visual agnosia, whereas the left medial temporo-occipital cortex is associated with integrative and associative types. Rehabilitation for visual cognitive dysfunction is crucial, and here we present a case of visual cognitive impairment because of hippocampal infarction.

Case Presentation

We present a case of an 87-year-old woman with atrial fibrillation, hypertension, and dyslipidemia as comorbidities who suddenly developed confusion and light-headedness. When her condition did not improve after half a day, she was rushed to the hospital. The patient could independently perform activities of daily living, and no cognitive impairment was specified.

A neurological examination at admission revealed that the patient was alert and well-oriented. No abnormalities in the cranial nerves, gross muscle weakness, or sensory disturbances were observed. However, the patient exhibited ataxia in her right upper and lower extremities and dysmetria on the knee heel test. Her visual field was normal. Nevertheless, the patient complained of difficulty in seeing things clearly. Although she could perform daily activities and engage in conversations, she experienced challenges at the execution stage, such as naming, constructing sentences, and copying. While she could visually identify objects, she faced challenges recognizing them by associating, matching, and verbalizing them into words from her lexical memory.

Regarding the linguistic aspects, she had very mild hearing loss but could engage in daily conversations without difficulty. Furthermore, the patient acknowledged that her cognitive functions had deteriorated; however, she was mostly able to provide correct answers regarding her orientation.

The word-level understanding in the Standard Language Test of Aphasia (SLTA) was rated good (4/10) at the short sentence level. However, during item handling, she admitted to making mistakes, such as selecting a comb while saying, “I wonder if this is what you mean by a toothbrush.” In addition, the correct answer rate for names was 50% (10/20), with many errors being semantically or visually unrelated.

Numerous morphological errors were observed in the handwriting of the kanji words presented in the illustrations, such as “inu (dog) → kanu (canoe)” and “shibunshi (newspaper) → Shinseki (relatives).” Furthermore, compared to kanji, both the handwriting and dictation of kana were better. The language designation score was 17/30. The patient expressed difficulties such as “I understand, but I cannot remember the name” and “I know what it means, but I cannot speak it.”

The score for higher brain function on the Japanese version of the Mini-Mental State Examination (MMSE-J) was 19/30 points. During naming tasks, the patient referred to a thermometer as a “mechanical pencil.” When this was pointed out to the patient, she understood its intended use. She gestured toward measuring body temperature while saying “something to measure,” indicating comprehension but exhibiting symptoms of anomia. A total of ten items were presented for naming, and only 4 were named correctly.

Furthermore, the patient struggled to name most objects, even after physical contact or semantic hints. When asked to write sentences freely, the patient wrote, “Do your best, cat,”

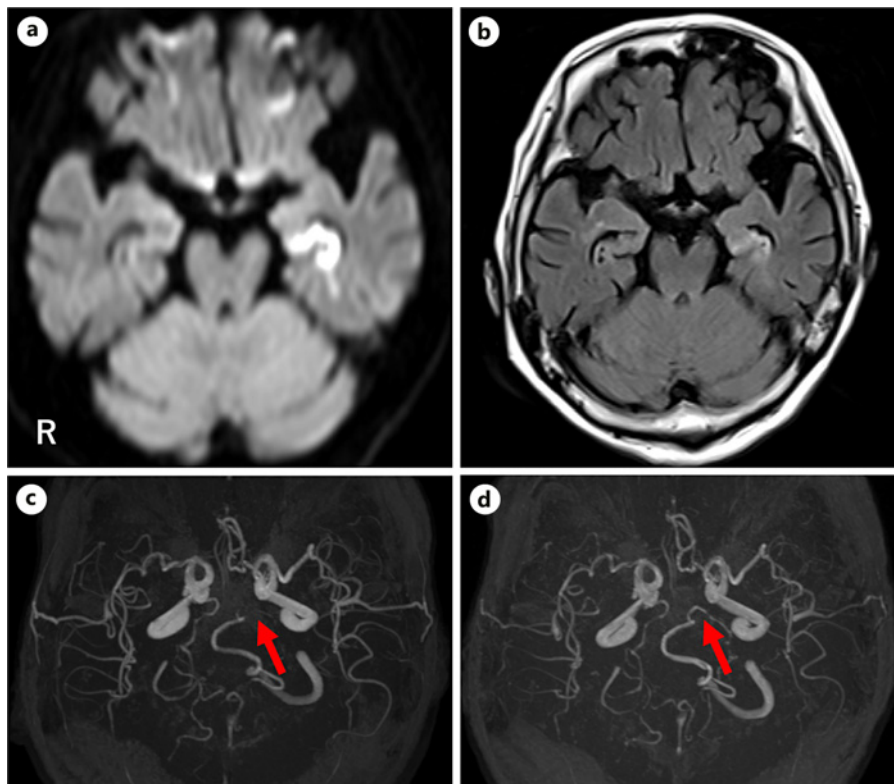


Fig. 1. Brain magnetic resonance imaging. **a** Axial view of fluid-attenuated inversion recovery (FLAIR) images. **b** Sagittal view of FLAIR. **c** Magnetic resonance angiography (MRA) in the initial stage. Red arrow: occlusion of the posterior cerebral artery. **d** MRA after 5 days. Red arrow: recanalization of posterior cerebral artery.

when asked about this, she responded, “I do not really understand.” In the behavioral neglect test, the patient scored 124/143 points. Many instances of no discernible left-right difference indicate an overall neglect.

In the copying test, the patient replicated the numbers and shapes relatively accurately. However, some mistakes were evident, such as the separation of hexagons (Fig. 1a). Additionally, while copying a cube, a mistake led to the distortion of the shape with depth (Fig. 1b).

During the extinction test, attention was drawn to previously checked targets, hindering searching for the next target [3]. However, performance improved when the previous target was erased, suggesting a potential role of disengagement where attention cannot shift from the target object, resulting in a score of 25/50.

The brain magnetic resonance imaging (MRI) showed a high signal intensity in the left hippocampus on diffusion-weighted images (Fig. 2a, b), a low apparent diffusion coefficient range, and a slightly high signal intensity on fluid-attenuated inversion recovery (FLAIR) images. Magnetic resonance angiography (MRA) showed fetal-type variations in the posterior communicating arteries on both sides and poor visualization of the left posterior cerebral artery after P2 (Fig. 2c). The patient was diagnosed with cerebral embolism because of atrial fibrillation and direct oral anticoagulant (DOAC) therapy was initiated. Five days later, a follow-up MRI revealed improvement in the left posterior cerebral artery (Fig. 2d).

Rehabilitation treatment for visual agnosia was initiated based on previously reported training for taste memory impairment [4, 5]. We conducted three types of training: visual

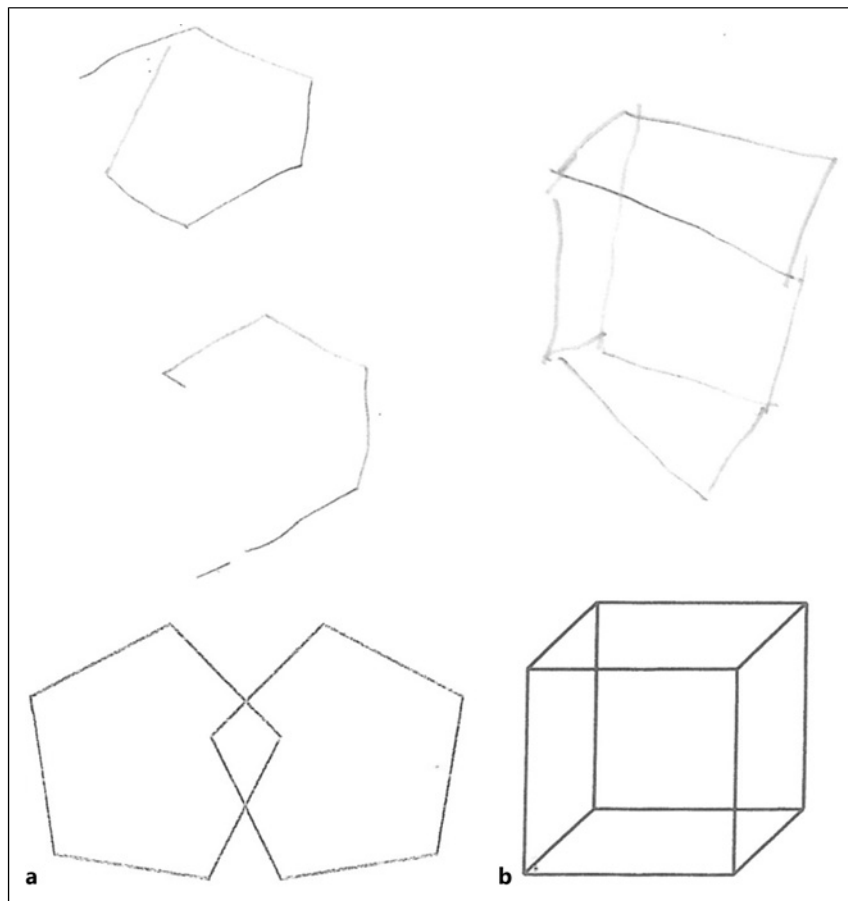


Fig. 2. Visual cognitive function test. **a** Separation of hexagons. **b** Copying a cube.

agnosia training (attribute learning training), verbalization learning training (writing training), and semantic memory training (copying training). In the copying training, the patient identified a photograph in the text (language) and copied it using colored pencils. The attribute-learning training involved learning about colors, shapes, tastes, eating methods, and seasons through audio and written text while observing photographs. Fourteen unidentified photographs were used for this purpose.

Following attribute-learning training, the number of images the patient could name was fewer than those recognized during copying training. However, there was an improvement compared to pre-training levels, enabling the patient to provide target semantic information, with seven out of 10 items named correctly (improvement rate; 75.0%). Problems related to sentence structure and copying improved during the implementation stage. Form errors in the handwriting of kanji characters in illustrations decreased, and the language specification score improved to 25/30 (improvement rate; 47.1%). The MMSE-J higher brain function score was 24/30 points (improvement rate; 26.3%). Hexagon separation and cube copying also demonstrated improvement. Even in the extinction test, performance improved to 45/50 (improvement rate; 40.0%). However, challenges persist in object recognition and verbalization disorders, where the patient struggles with associating, matching, and recognizing objects.

During physical therapy, the patient received rhythm training and basic movement training for ataxia, resulting in almost complete disappearance of symptoms after a week. The patient was discharged after a convalescent rehabilitation hospital stay 2 months after the

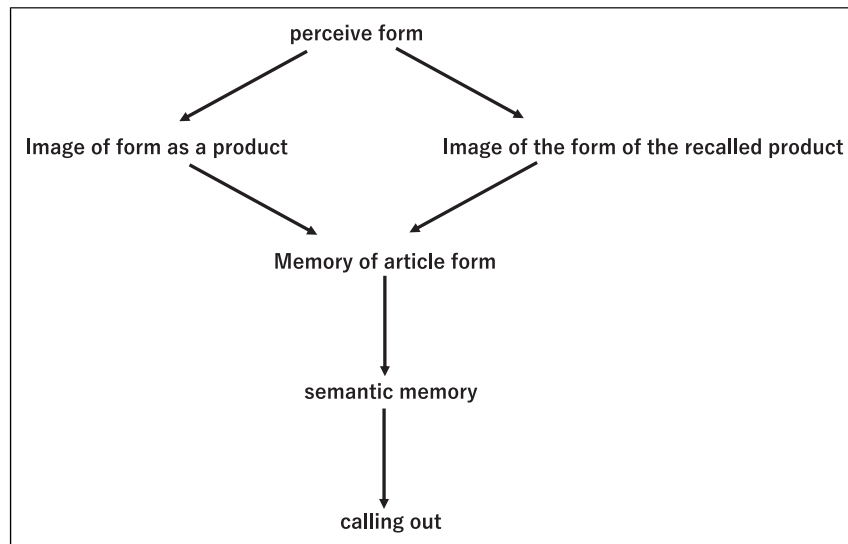


Fig. 3. Process showing visual recognition of objects.

onset of symptoms. We adhered to the CARE guidelines checklist and attached it as online supplementary material (for all online suppl. material, see <https://doi.org/10.1159/000538915>). [6].

Discussion

This case involves visual cognitive impairment following hippocampal infarction. The patient demonstrated the ability to copy shapes and differentiate between similar shapes. Therefore, we concluded that the patient's "simple form perception" ability was maintained. Furthermore, in a piecemeal approach, no symptoms indicating poor overall visibility were observed, suggesting no impairment in the integration of form perception. The patient's performance in copying tasks and form discrimination accuracy further indicated nearly normal form perception. However, semantic association tasks, such as categorizing line drawings, remain challenging, indicating visual cognition has not yet been achieved. Notably, tactile and auditory naming abilities and language remain unaffected. The patient's ability to name objects based on their gender definition indicated preserved "semantic memory" of objects. Consequently, we diagnosed the patient with associative visual agnosia, with the level of impairment ranging from the stage of matching form perception with the "memory of the shape of objects" to "semantic memory."

In this case, the ability to visually perceive an object and determine its nature was retained. However, there were challenges at the object recognition stage, involving associating objects with their meanings, matching them, and recognizing their identities. Visual processing involves the following stages: (1) observation of the object (pencil) and visually confirming it; (2) visual association cortex analyzes and integrates information such as shape and color; (3) comparison of the integrated information with information stored in the semantic memory; (4) determination of the object's identity; (5) initiation of movement commands from the frontal lobe and cingulate gyrus. In this case, we observed disorder at step (4) and above. It can be assumed that the symptoms of visual cognitive impairment because of hippocampal infarction may vary depending on the stage of impairment (Fig. 3).

Regarding the differences in memory impairment because of left and right hippocampal lesions, various reports on lobectomy for temporal lobe epilepsy have been documented [7–9]. Verbal memory disorder [7] has been associated with left temporal lobe damage, whereas visual memory disorder [8] has been associated with right temporal lobe damage. However, visual memory may exhibit bilateral dominance [9]. More case studies focusing on the dominant inferior parietal lobe are required to improve the precision of this regional neurology.

Several studies have investigated the relationship between hippocampal infarction and memory impairment [10, 11]. Milner et al. [12] reported verbal memory impairment in patients with left-sided lesions. However, verbal and nonverbal memory impairments were observed in this case, alongside a decline in visual memory. The significance of the lateral hippocampus and its association with short-term memory impairment has been highlighted in a previous report [13]. Similarly, in this case, memory tests revealed that long-term and immediate memories were relatively preserved, whereas short-term memory was significantly impaired. A previous report showed that memory impairment often persists, even with lesions confined to the left side [14].

Maintaining cognitive, physical, and visual functions is important to live independently in an aging society. Visual impairments contribute to other functional impairments and increase the risk of mortality [15]. Therefore, improving visual perception is essential for improving long-term prognosis. Despite numerous studies on rehabilitation for visuospatial cognitive dysfunction, such as spatial neglect [16–18], there is a scarcity of research on rehabilitation for visual cognitive impairment. In visual agnosia training, recommendations are included to preserve modality, language use, environmental adjustment, and more [19]. A previous study highlighted symptom amelioration through teaching and using training to enhance visual cognition [4]. Subsequently, it has been reported that combining visual discrimination exercises with a method that incorporates learning semantic information, including perceptual attributes (such as color and shape) and functional attributes (such as usage), leads to improvements without error [4].

In this case, MRA revealed disruption of the posterior cerebral artery, with a cardiogenic cerebral embolism being considered as the underlying mechanism. Although the infarct image on MRI was localized to the hippocampus, the physical symptoms, such as ataxia and higher brain function tests, suggested a wider area of cerebral ischemia. Although these neurological symptoms improved within a few days, they are believed to have been caused by impaired blood flow because of posterior cerebral artery stenosis. One week after onset, the associative visual cognitive dysfunction persisted, attributed to the infarct observed on MRI.

Acknowledgments

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Statement of Ethics

This study protocol was reviewed and approved by the Ethics Committee of Nara Prefectural General Medical Center (approval number 888). Written informed consent was obtained from the patient to publish this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Conceptualization: T.Mano. and S.K.; validation: T.Mano., S.K., and Y.U.; formal analysis: T.Masuda.; investigation and resources: Y.U.; data curation and visualization: S.K.; writing – original draft preparation, project administration, and writing – review and editing: T.Mano.; and supervision: H.T. and T.Masuda. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

The data that support the findings of this study are not publicly available due to their containing information that could compromise the privacy of research participant's but are available from the corresponding author T.M.

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