Anomia with Amnesia Caused by Hemorrhage in the Left Anterior and Medial Thalamus: Thalamic Anomia Particularly for Artificial Objects

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Thalamic anomia with amnesia · Mediodorsal nucleus · Ventral anterior nucleus · Category specificity · Episodic memory scale

Abstract
We herein report the case of a patient who showed pure anomia and amnesia caused by hemorrhage in the left thalamus, involving the anterior, ventral anterior, and mediodorsal nuclei. It was revealed that the anomia was characterized by impaired retrieval of object names, which was more pronounced in artificial objects, and abundant perseveration, whereas the amnesia was mild and limited to daily routine events, which was made clear from the results of an episodic memory scale. Detailed lesion localization and literature review revealed that a combination of pure anomia and amnesia can occur in a lesion involving the anterior, ventral anterior, or mediodorsal nucleus of the thalamus. The relative specificity to artificial objects can be explained by the locally damaged fiber connection to the putative category-specific lexical area in the temporal lobe.

Introduction
Anomia is observed in thalamic lesions, and it can exist without aphasia, i.e., with fully normal language function except for word-finding pauses [1]. Stereotaxic brain surgery and electrical stimulation studies of the thalamus revealed that anomia results from the stimulation to the ventral lateral and pulvinar nuclei [2] and perseveration from the mid-ventrolateral...
nucleus [2]. An isolated anomia without aphasia occurs in lesions in the ventral anterior (VA) nucleus [3, 4]. The VA nucleus receives fibers from the globus pallidus, substantia nigra, and frontal premotor cortex and projects to widespread regions of the frontal and parietal cortices [5]. Why anomia occurs following damage to the VA nucleus is unknown. It is suggested that fibers from Broca's area to the VA nucleus is interrupted [6], which might affect the motor aspect of word-finding.

The role of the thalamus in naming is yet to be elucidated. An interaction between the presupplementary motor area and thalamus was proposed to govern semantic retrieval [7] or lexical selection [6]. Another hypothesis is that the parvocellular portion (described below) of the mediodorsal (MD) nucleus is reciprocally connected with the dorsolateral prefrontal cortex, which is crucial for semantic memory [8].

On the other hand, the thalamic nuclei associated with memory are the anterior nuclei with the mammillothalamic tract and MD nucleus. The anterior nuclei receive projection fibers from the mammillary body via the mammillothalamic tract, which are incorporated into the Papez circuit and send efferent fibers to the anterior limbic area, cingulate gyrus, and parahippocampal gyrus [5]. Damage to the anterior nuclei and mammillothalamic tract causes amnesia, perseveration, apathy, intrusions of previous topics, transcortical motor aphasia [9], or a deficit of semantic memory [10]. On the other hand, the MD nucleus, which is known to be affected in Korsakoff's syndrome, consists of an anteromedial magnocellular division and a posterolateral parvocellular division. The magnocellular division receives input from the amygdala and has reciprocal connections to the medial prefrontal cortex, ventromedial cingulate cortex, inferior parietal cortex, and anterior insula [5]. The parvocellular division is connected reciprocally with the dorsolateral and dorsomedial prefrontal cortex, anterior cingulate gyrus, and supplementary motor area [5]. Damage to the MD nucleus causes amnesia, transcortical motor aphasia, apathy [9], or semantic retrieval deficit [8]. Amnesia due to a MD nucleus lesion is less severe than amnesia due to lesions in the anterior thalamic region or mammillothalamic tract [11, 12].

Although the coincidence of naming and memory impairment after thalamic stroke has already been reported in the anterior nuclei and VA nucleus [3, 4, 13], few studies have shown anomia with amnesia caused by damage to the MD nucleus. This study reports the case of a patient with left thalamic hemorrhage, involving the anterior, VA, and MD nuclei, who exhibited mild anomia and amnesia. The characteristics of anomia were described and discussed based on the localization of the lesion.

Case Report

A right-handed, 67-year-old office worker with 16 years of education was found to behave abnormally in the office and was referred to our hospital in January 2008. He had suffered hypertension and dyslipidemia for 2 years and had been treated with medicine. He was prescribed to undergo magnetic resonance imaging (MRI), but he forgot to undertake it twice. He was admitted to our hospital with a diagnosis of cerebral hemorrhage based on his MRI findings. Neurological examination showed elevated right deep tendon reflexes, tonic plantar response, and hyperalgesia of the right extremity.

On neuropsychological examination, the patient exhibited time disorientation and word-finding difficulties. No obvious retrograde amnesia was noted. Anterograde amnesia was evident for daily events in a day (described later). The patient explained his career almost completely and was aware that he forgot visiting a clinic.

The following tests were conducted within a month after disease onset (Table 1). Written informed consent was obtained from the patient.
| Table 1. Neuropsychological test scores of the patient |
|-----------------------------------------------|
| **Onset** | **Follow-up** | **Controls, mean (SD)** |
| MMSE (22 days after disease onset) | 20.8*/30 | 29.0 (1.2) |
| WAIS-R (30 days after disease onset) | 106 | 100 (15) |
| Digit span forward 4, backward 3 | 50* | 54* |
| WAB (27 days after disease onset, follow-up 4 months later) | 9 | 9.7 (0.6) |
| Spontaneous speech | 10 | 10.0 (0.0) |
| Information content (/10) | 8* | 9.5 (0.6) |
| Fluency (/10) | 50* | 59.2 (2.4) |
| Naming total (/10) | 10 | 9.87 (0.3) |
| Object naming (/60) | 9.75 | 9.8 (0.1) |
| Performance IQb | 10 | 9.4 (0.8) |
| Recognition of orally spelled kanji (/6) | 4 | 5.1 (1.6) |
| Repetition (/10) | 60* | 9.7 (1.0) |
| Comprehension total (/10) | 28.5 | 24.6 (3.9) |
| Reading total (/10) | 24 | 23.1 (2.3) |
| Recognition of orally spelled kanji characters (/6) | 29 | 29.3 (6.7) |
| Writing total (/10) | 1t 60, rt 60 | 59.8 (0.7) |
| Calculation (/24) | 66* | 81 |
| Raven’s progressive matrices (/37) | 60* | 100 (15) |
| Verbal memory | 60* | 102 |
| Attention/concentration | 60* | 94 |
| Delayed recall | 105 | 100 (15) |
| Visual memory | 107 | 100 (15) |
| TLPA (23 days after disease onset, follow-up 4 months later) | 60* | 84 |
| Naming total (/200) | 139* | 149* |
| Living things total (/120) | 193.35 (5.43) |
| Nonliving things total (/120) | 90* | 98.93 (2.58) |
| High-familiarity objects (/100) | 49* | 94.43 (6.39) |
| Low-familiarity objects (/100) | 14* | 19.83 (0.54) |
| Indoor structures (/20) | 10* | 19.37 (1.20) |
| Plants (/20) | 13* | 19.02 (1.30) |
| Processed food (/20) | 16* | 19.26 (0.91) |
| Outdoor structures (/20) | 16* | 19.41 (1.01) |
| Animals (/20) | 17* | 18.70 (1.50) |
| Colors (/20) | 16* | 19.52 (0.86) |
| Vehicles (/20) | 17* | 19.31 (0.92) |
A mini-mental state examination revealed time disorientation, delayed memory impairment, and mental arithmetic deficit. He did not remember that he undertook the three-word immediate recall test 5 min before. His verbal IQ and performance IQ in the Wechsler Adult Intelligence Scale-Revised were within a normal range, but his digit span score (forward, 4) was slightly reduced. In the Western Aphasia Battery, naming was mildly impaired (correct response: 15 out of 20 items). Semantic and perseverative errors were frequently observed, and phonemic cues were helpful. Other than the naming impairment, there were no signs of aphasia, alexia, or agraphia. The Wechsler Memory Scale-Revised (WMS-R) revealed that verbal memory, visual memory, and delayed recall were all impaired, with preservation of attention/concentration. Naming was further evaluated with the Test of Lexical Processing in Aphasia (TLPA; Japan Logopedics and Phoniatrics Association Committee on Speech and Language). The naming tasks comprised 10 categories with high and low heard-word familiarity (above and under 6 on a 7-rating scale by a Japanese database [14]) in equal number: indoor structures (bath, ceiling, etc.), outdoor structures (school, traffic light, etc.), animals, colors, processed food, vegetables/ruits, plants, vehicles, body parts, and tools. No category specificity was found between living and nonliving things (p = 0.87 by Fisher’s exact method). The lowest scores were plants, processed food, and outdoor structures. A familiarity effect was found (p < 0.0001 by Fisher’s exact method), wherein low-familiarity objects were named more poorly than high-familiarity objects. The naming of famous people in the Visual Performance Test for Agnosia (Japan Society for
Higher Brain Dysfunction) was within the normal range; however, the patient had repeated perseverative errors.

In all, the naming impairment covered all categories, and phonemic cues were helpful, leading to the diagnosis of pure anomia [15]. Because the names of famous Japanese people and cities (described in the next session) were mostly recalled, naming specific to proper nouns (proper name anomia) can be excluded. In addition, he showed semantic and perseverative errors in confrontation naming. Some authors suggested that thalamic anomia results from a semantic retrieval deficit [8] or semantic memory impairment [10] (described in Introduction). Although the patient did not undergo a semantic association task (choose semantically related word pairs), the fact that he achieved normal scores for the WAIS-R Information and Vocabulary subtests (footnote of Table 1) suggested that he did not have a semantic memory impairment.

The following special neuropsychological tests were also conducted. The naming of 12 famous cities in Japan was perfect. The patient underwent the Episodic Memory Scale for Inpatients (Table 2), which we made to assess the features and severity of amnesia. The test, which aimed to evaluate the patient’s episodic memory qualitatively, consisted of 11 questions: everyday routine events (1–5), occasional routine events (6–8), and rare episodic events (9–11). For some patients, occasional routine events (e.g., taking a bath) can become everyday routine events. In this case, the event should be regarded as an everyday routine one. In each question, the time (when), person (who), place (where), or content (what) was asked. One point was given to each test item, amounting to a total of 30 points. If there were multiple events for one test item, the total subscores were divided by the number of events for each test item. If the event did not take place and the patient answered “No,” a full subscore (2 or 3) was given. The accuracy of the event was validated by a hospital staff. If no person testified the patient’s response, full points were given to each item. The test was administered at 8:00 p.m., when all daily events had ended. The results revealed that the scores were 4.3/14 for everyday events, 6.7/8 for occasional events, and 8/8 for rare events, suggesting that the patient’s amnesia was mostly derived from the memory impairment of everyday routine events. In fact, the patient did not remember the occurrence of some daily events (e.g., doctor’s visit and blood pressure monitoring).

T1- and T2-weighted MRI images, which were taken 13 days after disease onset, showed a high-intensity area in the left thalamus, involving the anterior, VA, and MD nuclei and mammillothalamic tract, based on the Morel atlas [16] (shown in Fig. 1a, upper panel). An old infarction in the left anterior periventricular white matter and a chronic ischemic change in the bilateral basal ganglia were noted.

The patient returned to work 2 weeks after being discharged. Memory impairments in the WMS-R resolved at 3 months after disease onset; however, verbal memory and delayed recall were in the lower limit of the normal range (Table 1). A follow-up of the TLPA battery at 4 months after disease onset showed an improvement in the naming of indoor structures ($p = 0.0253$ by the McNemar test) and outdoor structures ($p = 0.0455$ by the McNemar test) (Table 1). This fact implies that the patient exhibited pure anomia with relative specificity to artificial objects.

Three years later, a low-intensity area, which suggests hemosiderin deposition, was observed along the lateral wall of the third ventricle (shown in Fig. 1a, middle and lower panels). To identify damaged nuclei, we overlapped the axial slice images at 0.0 and 6.3 mm above the anterior commissure-posterior commissure line in the Morel atlas [16] with the actual T2-weighted MRI images at nearly the same axial level (0.0 and 6.4 mm above the anterior commissure-posterior commissure line, slice thickness = 6.0 mm) of the patient, using Photoshop CS6 Extended software (Adobe; Tokyo, Japan). We fitted the atlas image
with the MRI thalamus image without changing the length-to-width ratio. The overlapped image revealed damage to the anterior (anteromedial and anteroventral), VA, and MD nuclei and the mammillothalamic tract (shown in Fig. 1b).

Table 2. Episodic Memory Scale for Inpatients

| Item                                                                 | Yes, No | (In case of “Yes”)a | (In case of “Yes”)b | (In case of “Yes”)b | (In case of “Yes”)b |
|----------------------------------------------------------------------|---------|----------------------|----------------------|----------------------|----------------------|
| 1. Did you take temperature? Yes, No                                  |         | (1) When, how many?  | (2) Who asked?       | (1) When?            | (2) Who asked?       |
| 2. Did you take medicine (morning, noon, evening)? Yes, No           |         | (1) When?            | (2) Who reminded?    | (3) How many tablets?| (3) What did he/she do? |
| 3. Did you have (breakfast, lunch, supper)? Yes, No                  |         | (1) When?            | (2) What kind (2 items)?| (3) Which arm, how high? | (3) What kind, which arm? |
| 4. Did a doctor visit you? Yes, No                                  |         | (1) When, how many?  | (2) Who?            | (3) What did he/she do? | (3) Where is the room? |
| 5. Did you take blood pressure? Yes, No                              |         | (1) When?            | (2) Who did?         | (3) Which arm, how high? | (3) What? |
| 6. Did you take blood sampling or intravenous drip infusion? Yes, No |         | (1) When?            | (2) Who?            | (3) What kind, which arm? | (3) Where is the room? |
| 7. Did you take a bath? Yes, No                                      |         | (1) When?            | (2) Who told?        | (3) Where is the room? | (3) Where? |
| 8. Did you take blood sampling, intravenous infusion, or a bath yesterday or other routine work (e.g., sheet exchange, weight measurement) since yesterday? Yes, No |         | (1) When?            | (2) What?           | (3) Location?        | (3) Location? |
| 9. Did you go out or did someone visit you since yesterday? Yes, No |         | (1) When?            | (2) Where or who?    | (3) Location?        | (3) Location? |
| 10. Did you take some examination today? Yes, No                      |         | (1) When?            | (2) Where?           | (3) Location?        | (3) Location? |
| 11. Did you take some examination yesterday? Yes, No                 |         | (1) When?            | (2) Where?           | (3) Location?        | (3) Location? |

Daily events\(^c\)/11 Occasional events\(^c\)/6 Rare events\(^c\)/8 Total /30

\(^a\)Give a full subscore (2 or 3) when the event did not take place, and the patient answered “No.”
\(^b\)Ask about the latest event.
\(^c\)Daily event score is the sum of 1–5 test item scores, rare event score is the sum of 6–8 test item scores, and rare event score is the sum of 9–11 test item scores.
Fig. 1. a MRI T1- and T2-weighted axial images and T2-weighted coronal images of the patient. A high-intensity area suggesting a hemorrhage was observed in the left thalamus on the T1- (1.5 Tesla, TR = 480 ms, TE = 15 ms) and T2-weighted (TR = 3,500 ms, TE = 88 ms) images 13 days after disease onset (upper panel). The hemorrhage involved the MD and VA nuclei and the mammillothalamic tract. Three years later, a linear low-intensity area, which suggests a hemosiderin deposition (arrows), was observed along the lateral wall of the third ventricle on the T2-weighted axial and coronal images (middle and lower panels); the MD nucleus (lower, left, and middle images) appeared to be more involved than the VA nucleus and mammillothalamic tract (lower panel, right image). The fornix was not directly damaged; however, it is possible that its fiber connection to the anterior nuclei of the thalamus was affected. An old infarction in the left anterior periventricular white matter (middle panel, right image) and a chronic ischemic change in the bilateral basal ganglia were noted, which had already been observed at the time of the hemorrhage. b Overlapped images of the patient’s MRI and the Morel atlas slice at the same level through the left thalamus. Abnormal high-intensity (mesial side) and low-intensity areas (colored in red) included the AM, AV, MD, and VA nuclei and the mtt (slice thickness = 6.0 mm). The atlas slice images were modified from Morel [16] (DV0 and D6.3). Damaged thalamic structures were labeled on the atlas. AM, anteromedial; AV, anteroventral; CeM, central medial; CL, central lateral; MD (mc, pc, pl), mediodorsal (magnocellular, parvocellular, and paralamellar); mtt, mammillothalamic tract; MV, medioventral nucleus; PuM, medial pulvinar; Pvl, paraventricular; sm, stria medullaris; VA (mc, pc), ventral anterior (magnocellular and parvocellular); VLa, ventral lateral anterior; VLpl, ventral lateral posterior paralamellar.

(Figure continued on next page.)
Discussion

The patient presented as anomia with amnesia due to left thalamic hemorrhage, involving the anterior, VA, and MD nuclei. This case suggests that anomia with amnesia can result from a lesion in the anterior nuclei, VA nucleus, or MD nucleus. The anomia recovered in 4 months especially in the indoor and outdoor structures, suggesting relative category specificity to...
artificial objects. On the other hand, the amnesia was limited to memory impairment of daily events, whereas the recall of rare episodic or “big” events was preserved.

First, we discuss category specificity in thalamic anomia. The patient's anomia involved both living objects and nonliving objects, with no significant difference between them. However, a follow-up study revealed that the scores for indoor and outdoor structures increased in 4 months, so it is possible that the hemorrhage predominantly affected the naming of certain nonliving objects. A case of category-specific anomia with an anterior thalamic lesion was reported [3]. In this case, animate categories (vegetables, animals, and body parts) had more errors than inanimate categories. However, it is difficult to argue the category specificity in this patient because an inanimate category (toys) was also subject to errors. In our patient, the score for naming animate (nearly equal to living) categories and that for naming inanimate categories did not significantly differ. On the other hand, the fact that the scores for indoor and outdoor objects improved suggests that these artificial objects are subject to lexical retrieval disturbances in thalamic anomia. Whether this minor category specificity was characteristic of thalamic anomia or generally observed in mild anomia, i.e., naming of artificial objects is preferentially disturbed in pure anomia [17], remains to be elucidated. In this regard, category-specific naming deficits in a thalamus lesion might result from destruction of the presupplementary motor area (pre-SMA)-VA nucleus circuit that selectively passes lexical-semantic information [6].

The second problem is concerning episodic memory impairment of daily events. The patient did not exhibit obvious retrograde amnesia, but anterograde amnesia was evident. He remembered the episodic events before the disease onset. However, an episodic memory questionnaire revealed that he forgot experiencing some daily events. This is similar to patients with amnestic mild cognitive impairment or early-phase Alzheimer’s disease, who do not remember where they placed their eyeglasses, key, or mobile phone in their houses. These examples seem to be derived from attentional impairment [18]. However, daily events involve little attention; thus, the episode is less consolidated in memory. This is probably why patients with mild memory impairment are more likely to fail in recalling the event. It should be noted that minimal or mild amnesia presents with forgetting the occurrence of everyday events. Our episodic memory scale is different from some autobiographical episodic memory tests (e.g., [19]) in that the tested period is limited to 2 days, and it covers all episodic events including daily routine events.

Finally, we discuss the mechanism underlying thalamic anomia with amnesia. The combination of isolated (pure) anomia and amnesia can occur in patients with lesions in the anterior nuclei with mammillothalamic tract, VA nucleus, or MD nucleus. However, why anomia accompanies amnesia is unknown. Pure anomia is attributable to lesions involving the left posterior fusiform/inferior temporal gyri [17, 20]. It is suggested that a disconnection of associated fibers between the temporal gyri is critical for the production of anomia [20]. Considering the anterior nuclei are directly connected to the parahippocampal gyrus [5], it is plausible that damage to the anterior nuclei may affect intratemporal lexical processing via the parahippocampal gyrus, resulting in anomia.

The pathophysiology of the VA nucleus in category-specific anomia was discussed earlier. However, it is unknown how the VA nucleus is associated with the temporal lobe where the lexical-semantic knowledge is stored.

On the other hand, a diffusion tensor imaging study revealed that the anterior and medial area of the thalamus including the anterior nuclei and MD nucleus is connected to the prefrontal cortex and the temporal lobe [21]; therefore, damage to the anterior nuclei or MD nucleus may interfere with the retrieval of some lexical knowledge separately stored in the temporal lobe [22]. The relative specificity to artificial objects can be explained by the locally damaged fiber connection to the putative category-specific lexical area (i.e., site
of lexicon for tools) in the temporal lobe. Further studies are needed to evaluate the category specificity of thalamic anomia.

**Statement of Ethics**

Written informed consent was obtained from the patient for publication of the details of his medical case and any accompanying images. The patient is able to provide confirmation for the publication of this case report as not being classified as vulnerable based on the 2000 Hague Protection of Adults Convention (https://www hcch net/en/instruments/conventions/full-text/?cid=71). The authors confirm that the paper is exempt from Ethical Committee approval because this work is a case report, and all information is fully anonymized, in accordance with the institutional (Research Ethics Committee of the Mitsui Memorial Hospital) and national (Japanese Ministry of Education, Culture, Sports, Science and Technology, Ministry of Health, Labour and Welfare, and Ministry of Economy, Trade and Industry) guidelines.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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**Author Contributions**

Yasuhisa Sakurai collected neuropsychological data and prepared the manuscript. Toru Mannen examined the patient during his admission and supervised preparing the manuscript.

**Data Availability Statement**

The original contributions presented in the study are included in the article; further inquiries can be directed to the corresponding author.

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