



Research report

Semantic memory deficits are associated with pica in individuals with acquired brain injury



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ABSTRACT

Although pica is one of the most prominent signs in individuals with severe cognitive impairment, the mechanisms and neural basis for pica have not been well elucidated. To address this issue, patients with acquired brain injury who showed pica and hyperorality were investigated. Eleven patients with pica, i.e., individuals who eat non-food items, and eight patients with hyperorality but who never eat non-food items were recruited. The cognitive and behavioral assessments and neural substrates of the two groups were compared. For basic cognitive and behavioral functions, two kinds of mental state examination—the mini-mental state examination and the new clinical scale for rating of mental states of the elderly—were administered. For pica-related behavioral features, frontal release signs, semantic memory deficits, and changes in eating behaviors were compared. Compared with the hyperorality group, the pica group had more severe semantic memory deficits and fewer frontal release signs, whereas there was no significant difference in changes in eating behaviors. Individuals in the pica group always had a lesion in the posterior part of the middle temporal gyrus. These findings suggest that semantic memory deficits following temporal lobe damage are associated with pica.

1. Introduction

Pica is a persistent eating of non-nutritive, nonfood substances (DSM-5, American Psychiatric Association, 2013 [1]). It may lead to dangerous consequences, such as malnutrition, intoxication, suffocation, and ileus or intestinal perforation, which sometimes require emergency medical treatment [2]. Descriptions of pica as a syndrome are found in antiquity [3–6], but the mechanisms underlying pica have not been well elucidated except for a nutritional hypothesis for ice eating, which may result from iron deficiency anemia [6]. Definitions of pica vary among investigators. Most researchers apply the term pica to a pathological craving both for food and non-food items [2–7], whereas Walker et al. [8] defined it as the eating of non-food items. The mixing of both food and non-food items within the definition of pica might be responsible for difficulties in investigating the mechanisms underlying pica. In this article, we applied Walker's definition, in which pica is defined as the eating of non-food items, to better study the mechanisms and neural basis of this behavior.

Hyperorality, which was first reported in Klüver–Bucy syndrome [9,10], has symptoms that are similar to those of pica. The symptoms of

hyperorality have been described in terms of their neurological basis more thoroughly than those of pica and might provide a clue to the mechanisms underlying pica. Despite their similarities, there is a notable difference between pica and hyperorality as described in Klüver–Bucy syndrome [9,10]. Whereas monkeys with hyperorality never eat non-food items but instead discard them after examining them by mouth, patients with pica do eat non-food items. This clear distinction is not always maintained, as some individuals who were reported to have Klüver–Bucy syndrome did eat non-food items [11–14], whereas others had only a tendency to stuff or place food items in their mouths [15,16]. Our goal is to clarify the mechanisms that separate these two conditions.

In human studies, hyperorality has often been linked to frontotemporal dementia rather than Alzheimer's disease [14,17]. Although hyperorality in Klüver–Bucy syndrome is associated with temporal lobe deficits, human hyperorality has also been described in patients with focal frontal lobe lesions and in the context of frontal release signs [18,19]; it also has a remarkable dependency on external stimuli, e.g., utilization behavior [20]. Kertesz et al. [21] pointed out that hyperorality is one of the signs of frontal behavior abnormalities. These studies

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thus suggest that hyperorality might be related to frontal lobe damage and relevant frontal release signs and/or temporal lobe damage.

Although pica has been found in individuals with schizophrenia, intellectual disability, and pervasive development disorder, as well as in normal children and a pregnant woman [4], there have been several previous case reports of pica in individuals with degenerative disease and acquired brain injury. Cummings and Duchon [11] described a degenerative patient with pica who showed marked atrophy in the left anterior temporal region. Likewise, Lilly et al. [12] described a pica patient with anterior temporal atrophy. In the same report, they described a patient with pica that occurred after a traumatic brain disease in which neural damage was observed in the inferior portions of the bilateral temporal lobes. Hodges et al. [22] also reported a semantic dementia patient who ate non-food items such as cigarette ends during later stages of the disease. Mendez and Foti [23] reported a patient with focal left temporoparietal damage who underwent respiratory arrest after stuffing his mouth with surgical gauze. Funayama et al. [24] described three patients with pica-associated severe semantic memory deficits whose initial symptom was logopenic variant of primary progressive aphasia with focal left temporoparietal cortical atrophy. Funayama and Nakajima [25] also described a patient with temporoparietal cortical atrophy who had progressive transcortical sensory aphasia and progressive ideational apraxia at the onset and pica at later stages. From clinical observations, Morris et al. [26] suggested that a failure to recognize objects might account for the eating of inedible objects. Ikeda [27] also suggested that pica might be related to semantic memory deficits. These reports suggest that pica might be associated with temporal lobe damage and relevant semantic memory deficits.

To study pica and hyperorality, changes in eating such as appetite and food preference among dementia patients should be taken into account. Morris et al. [26] suggested that changes in eating, including pica, could result from a change in the sense of taste and of smell. Changes in the sense of taste [29] and smell [29,30] and in eating behaviors [27] are common in dementia, especially in frontotemporal lobar degeneration.

These earlier findings prompted us to explore the mechanisms behind pica and hyperorality by recruiting patients with acquired brain injury who developed pica and hyperorality and using systematic cognitive and behavioral examinations that focused on frontal release signs, semantic memory deficits, and changes in eating behaviors. We focused on patients with acquired brain injury rather than degenerative diseases, as they have relatively focal brain damage in contrast to patients with degenerative diseases, and analyzed their lesions to determine common and disease-specific regions.

2. Materials and methods

2.1. Participants

Ethical aspects of this study were reviewed and approved by the Ashikaga Red Cross Hospital Human Research Ethics Committee. Because the subjects were incapable of giving consent because of their severe cognitive impairment resulting from acquired brain injury, this study was performed after obtaining informed consent from all caregivers who had legal custody of the subjects. As we defined pica as the eating of non-food items, the compulsive eating of food items, e.g., ice eating because of iron deficiency anemia, was not counted as pica. As it is difficult to clearly assess hyperorality, we defined hyperorality as having to remove substances from the mouth because of an excessive eating of nutritive substances [14] or because of a strong tendency to examine non-food items by mouth [9,10]. Thus, patients who do eat non-food items in the context of hyperorality were classified into the pica group, not the hyperorality group.

The study participants were recruited from the Cognitive Dysfunction Clinic associated with Ashikaga Red Cross Hospital,

Tochigi, Japan, during the period from January 2007 to December 2016 and were limited to those with acquired brain injury. Patients who had neuropsychiatric, developmental, or degenerative diseases before the onset of acquired brain injury were excluded. No children or pregnant women were included in this study. Also excluded were those with acute or subacute confusional state. All the participants were screened to rule out iron deficiency anemia.

2.2. Methods

Background demographic information about the patients included their etiologies, the age of pica or hyperorality onset, gender, and level of education. The following assessments were carried out at the time of onset of pica or hyperorality.

2.2.1. Basic cognitive and behavioral assessments

We used the Japanese version of the Mini Mental State Examination (MMSE-J) [31] and the new clinical scale for the rating of mental states of the elderly (NM scale) [32], which assesses cognitive functions for everyday life, i.e., the ability to do housework and to communicate, along with measures of speech, memory, and orientation, with a maximum of 50 points. Both tests have high validity and reliability [31,32]. For episodic/autobiographic memory performance, we used the subscale for episodic memory performance in the MMSE-J, which includes an orientation task and a delayed recall task.

We also assessed symptoms of Klüver–Bucy syndrome. The five symptoms of Klüver–Bucy syndrome [9,10] are psychic blindness (i.e., multi-modal agnosia or semantic memory deficits), hyperorality, hypermetamorphosis, changes in emotional behavior, and changes in sexual behavior. Assessments for the first two symptoms are described in detail below. Hypermetamorphosis, a strong tendency to attend and react to every visual stimulus, can be considered as a utilization behavior in humans, the assessment of which is described below. The remaining two symptoms, changes in emotional behavior (i.e., the complete absence of all emotional reactions) and changes in sexual behavior (i.e., an increase in sexual activity), were assessed from clinical observations.

2.2.2. Frontal release signs

Typical frontal release signs/symptoms include primitive reflexes [33,34], utilization behavior [20], imitation behavior [35], and environmental dependency syndrome [36]. Among those, primitive reflexes are the most basic signs and are usually involved in the other symptoms. In this context, we first examined the grasp reflex and sucking reflex as frontal release signs for which their neural substrates lie in the frontal lobe, in particular, in the medial frontal lobe [37,38]. For the grasp reflex, we followed Seyffarth's method [33], in which the grasp reflex is assessed as the finger flexion with thumb adduction that occurs in response to a distally moving pressure applied to the palm before traction of the finger flexors occurs, while the shoulder, arm, and forearm are held in a fixed position. In this study, the examiner used the pressure of his/her finger on each of the patients' palms to trigger the reflex. For the sucking reflex, we followed the definition of Schott and Rossor [39], which defines the sucking reflex as instinctive sucking in response to tactile stimulation in the oral region. The examiner assessed this reflex by tapping each subject's upper lip lightly with his finger.

Second, we used the scale of utilization behavior from the Japanese version of the Frontal Behavioral Inventory [21,40–42] to further assess frontal release signs. In this scale, utilization behavior is defined as follows: “Does he/she seem to need to touch, feel, examine, or pick-up objects within reach and sight?” The question was answered by a caregiver familiar with the patient's everyday life. In the inventory, the frequency of utilization behavior is scored as follows: 0, “never”; 1, “occasionally”; 2, “moderately often”; and 3, “most of the time”, and the severity is scored as follows: 0, “none”; 1, “mild”; 2, “moderate”; 3, “severe”. A total score is expressed as the product of the frequency score

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