Journal of
Alzheimer's Disease & Parkinsonism

Funayama, J Alzheimers Dis Parkinsonism 2017, 7:5
DOI: 10.4172/2161-0460.1000367

Mini Review Open Access

Pica after Acquired Brain Injury and in Degenerative Diseases is Associated with Temporal Lobe Dysfunction and its Related Semantic Memory Deficits

Michitaka Funayama*

Department of Neuropsychiatry, Ashikaga Red Cross Hospital, Japan

Abstract

Pica is one of the most enigmatic behaviors for human beings and its neural basis and underlying mechanisms have yet to be elucidated. We have addressed this issue by investigating patients with acquired brain injury who presented with pica and found that semantic memory deficits are associated with pica rather than frontal release signs or changes in eating behaviors. The brain region specific for pica in our study was the posterior part of the left middle and inferior temporal gyri. Our findings suggest that semantic memory deficits as a result of temporal lobe damage are associated with pica. For degenerative diseases, case reports and our data in this article suggest that Alzheimer's disease and semantic dementia are the two main etiologies that cause pica. Considering the temporal lobe involvement and semantic memory deficits for both etiologies, our findings on pica for acquired brain injury might also be applied to degenerative diseases. The most common items involved in pica are daily necessities that are easy to swallow or drink, such as lipstick or shampoo. These items should be avoided for patients with pica.

Keywords: Pica; Semantic memory deficits; Temporal lobe; Alzheimer's disease; Semantic dementia

Introduction

Among the variety of behavioral and psychological symptoms of dementia, pica, a persistent eating of non-nutritive substances [1], leads to particularly high burdens for family caregivers. Pica may cause digestive diseases, which sometimes require medical emergencies [2]. Although pica was first reported in pregnant women, it also occurs in children and adults, as a result of neurodevelopmental disorders or psychiatric disorders, and in individuals with acquired brain injuries or degenerative diseases [3-6]. In spite of detailed description for centuries [3-8], the mechanism of pica has been poorly understood.

Several hypotheses of pica have been proposed to explain this enigmatic behavior, including nutritional, psychological, cultural and pharmacologic theories. Among those hypotheses, nutritional theory is the most promising one, explaining ice cravings as a result of iron deficiency anemia [6]. However, except for ice cravings, the definite mechanisms underlying pica has not been well elucidated [6]. Regarding pica that occurs after acquired brain injury or in degenerative diseases little was known about the symptomatology or the neural substrates of this abnormal behavior.

DSM-5 defines pica as a persistent eating of non-nutritive, nonfood substances, which includes a pathological eating of both food and non-food items [2-7]. The mixing of both food and non-food items within the definition of pica might confuse scientific studies. To avoid confusion, we applied Walker's definition in this article, in which pica is defined as the eating of non-food items [8].

Previous case reports of pica in individuals with acquired brain injury and degenerative diseases had a lesion or degeneration in temporal lobe [9-14]. From clinical observations, Morris et al. [15] suggested that a failure to recognize objects might account for the eating of inedible objects. Ikeda [16] 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.

Hyperorality, which was first reported in Klüver-Bucy syndrome [17,18], has symptoms that are similar to those of pica, although there is a notable difference between pica and hyperorality as described in Klüver-Bucy syndrome [17,18]. 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. Human hyperorality has also been described in patients with focal frontal lobe lesions and in the context of frontal release signs [19,20]; it also has a remarkable dependency on external stimuli, e.g. utilization behavior [21].

To study pica, changes in eating such as appetite and food preference among dementia patients should be taken into account. Morris et al. [15] 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 and smell and in eating behaviors are common in dementia, especially in frontotemporal lobar degeneration [16].

These backgrounds prompted us to explore the mechanisms behind pica by using systematic cognitive and behavioral examinations that focused on frontal release signs, semantic memory deficits, and changes in eating behaviors.

Semantic Memory Deficits Associated with Pica after Acquired Brain Injury

There have been no systematic and comprehensive studies on pica. To

***Corresponding author:** Michitaka Funayama, Department of Neuropsychiatry, Ashikaga Red Cross Hospital 284-1 Yobe, Ashikaga-city, Tochigi, 326-0843, Japan, Tel: 081-284-21-0121; Fax: 081-284-22-0225; E-mail: mctkfnym@gmail.com

Received August 08, 2017; **Accepted** August 28, 2017; **Published** September 05, 2017

Citation: Funayama M (2017) Pica after Acquired Brain Injury and in Degenerative Diseases is Associated with Temporal Lobe Dysfunction and its Related Semantic Memory Deficits. J Alzheimers Dis Parkinsonism 7: 367. doi: 10.4172/2161-0460.1000367

address this issue, we recruited 11 patients with pica and 9 patients with hyperorality, who never ate non-food items despite eating excessively [22]. All of them were patients after acquired brain injury and those with neuropsychiatric, neurodevelopmental, or degenerative disorders were excluded. We compared the cognitive and behavioral functions and neural basis of the two groups. Regarding basic cognitive and behavioral functions, two mental state examinations the mini-mental state examination [23] and the new clinical scale for rating of mental states of the elderly [24] were administered. For behavioral features specific for pica, frontal release signs, semantic memory deficits and changes in eating behaviors were compared.

Frontal release signs were studied by assessing primitive grasp and sucking reflexes as well as by administering a questionnaire to caregivers about utilization behavior in the frontal behavior inventory [25]. Semantic memory deficits were assessed by three methods, including the patient-based manipulation of familiar tools to investigate semantic errors; three questions regarding semantic memory from the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) (http://cmhr.anu.edu.au/ageing/Iqcode/), which were answered by caregivers; and a semi-structured interview, also given to caregivers, to look for semantic memory errors in tool function and food recognition. For example, the question related to tool function was "Does he or she sometimes try to use tools for inappropriate purposes, such as, trying to use a toothbrush for a comb, shaving cream for a toothbrush, or a watering can for a dustpan?" Changes in eating behaviors were assessed using the Swallowing/Appetite/Eating Habits Questionnaire [16]. Neural substrates were studied with morphological imaging, and areas of lesion overlap were determined using MRIcro software.

The most common objects involved in pica were daily necessities, such as cotton, lipstick, toilet paper, and shampoo, most of which are easy to swallow or drink. Background demographic information, cognitive and behavioral assessments, and eating behaviors showed no significant differences between the pica and hyperorality groups. Semantic memory deficits were observed more often in the pica group, whereas the frontal release signs were noted more in the hyperorality group. The brain region specific for pica was the posterior part of the left middle and inferior temporal gyri. Our results suggest that semantic memory deficits after temporal lobe damage might lead to pica.

Our findings are compatible with the hub-and-spoke theory for semantic memory concerning function [26]. According to this theory, the reciprocal connection between the anterior temporal lobe and the posterior part of the middle temporal gyrus leads these two regions to act as a hub and spoke, respectively. That is, collaborations between a common transmodal region (the anterior temporal lobe) and a modality-specific region for function (the posterior part of the middle temporal gyrus) contribute to semantic memory for function. The authors also pointed out that selective damage to a spoke can lead to a category-specific deficit, suggesting that a lesion in the posterior part of the middle temporal gyrus superior temporal sulcus might cause semantic deficits for function. As the most common items for pica are items that are used for daily necessities with specific functions and patients with pica frequently show semantic memory errors for tool function, our finding that the posterior part of the left middle temporal gyrus is involved in pica is compatible with this theory.

Degenerative Diseases and Pica

In previous reports, pica that occurs in degenerative diseases is associated with semantic dementia [9-11] and early-onset Alzheimer's disease [13,14]. In our facility, the Cognitive Dysfunction Clinic associated with Ashikaga Red Cross Hospital, Tochigi, Japan, 19 patients with advanced-stage degenerative diseases presented with pica during the period from January 2007 to August 2014. Of the 19 patients, 11 had Alzheimer's disease, 6 had semantic dementia, and the remaining 2 had behavior variant frontotemporal dementia. Results of the mini-mental state examination for the 19 degenerative patients were extremely poor, with an average score of 3.1 ± 3.0 out of 30, suggesting severe cognitive decline. Whereas frontal release signs, i.e., grasp reflex or sucking reflex, were found in only 4 patients (21.1%) and changes in eating behaviors in 10 patients (52.6%), semantic memory errors in tool function, which were obtained from a semi-structured interview given to caregivers [22], were noted for 16 patients (84.2%). These findings most likely reflect a close relationship between pica and semantic memory deficits.

Of note, among the 11 patients with Alzheimer's disease, 7 patients were categorized as having early-onset Alzheimer's disease (i.e., they were diagnosed before the age of 65). Based on clinical observations, most of the individuals with early-onset Alzheimer's disease did not show episodic memory impairment at the onset of the disease, the epitome of typical Alzheimer's disease. Instead, they often showed progressive aphasia, such as logopenic variant primary progressive aphasia [13] and progressive transcortical sensory aphasia [14], as an initial symptom. These cases suggested that dysfunction at the left temporoparietal cortices for logopenic variant primary progressive aphasia [13] or the left posterior temporal cortex for progressive transcortical sensory aphasia [14] might contribute to the development of pica. Hypofunction within these areas is compatible with our findings on the neural basis for pica after acquired brain injury [15], even if slightly different areas are involved. Pica in semantic dementia is also compatible with the hub-and-spoke theory for function [26]. Mounting convergent evidence for the importance of the anterior temporal lobe in amodal semantic memory has come from studies of patients with anterior temporal lobe damage (e.g. semantic dementia), functional neuroimaging and studies of repetitive transcranial magnetic stimulation in patients with anterior temporal lobe lesions that cause semantic memory impairment [27].

Taken together, pica in degenerative diseases might also be caused by temporal lobe degeneration and its related deficits on semantic memory function.

Conclusion

In our study on pica after acquired brain injury and in degenerative diseases, semantic memory deficits were associated with pica rather than frontal release signs or changes in eating behaviors. The brain region specific for pica was the posterior part of the left middle and inferior temporal gyri. Our findings suggest that semantic memory deficits as a result of temporal lobe damage or degeneration might lead to pica. Items related to daily necessities that are easy to swallow or drink are the most common objects involved in pica. These items should be avoided for those with pica after acquired brain injury or in degenerative diseases.

Acknowledgement

We thank the patients and their family members for allowing us to perform these studies.

References

1. American Psychiatric Association (2013) Diagnostic and statistical manual of mental disorders: DSM5. Arlington: American Psychiatric Association Publishing.

2. Anderson JE, Akmal M, Kittur DS (1991) Surgical complications of pica: Report of a case of intestinal obstruction and a review of the literature. Am Surg 57: 663-667.
3. Danford DE (1982) Pica and nutrition. Annu Rev Nutr 2: 303-322.
4. Feldman MD (1986) Pica: Current perspectives. Psychosomatics 27: 519-523.
5. Lacey EP (1990) Broadening the perspective of pica: literature review. Public Health Rep 105: 29-35.
6. Moore DF Jr, Sears DA (1994) Pica, iron deficiency and the medical history. Am J Med 97: 390-393.
7. Horner RD, Lackey CJ, Kolasa K, Warren K (1991) Pica practices of pregnant women. J Am Diet Assoc 91: 34-38.
8. Walker AR, Walker BF, Sookaria FI, Cannan RJ (1997) Pica. J R Soc Health 117: 280-284.
9. Cummings JL, Duchen LW (1981) Klüver-Bucy syndrome in Pick disease: Clinical and pathologic correlations. Neurology 31: 1415-1422.
10. Lilly R, Cummings JL, Benson F, Frankel M (1983) The human Klüver-Bucy syndrome. Neurology 33: 1141-1145.
11. Hodges JR, Patterson K, Oxbury S, Funnell E (1992) Semantic dementia. Progressive fluent aphasia with temporal lobe atrophy. Brain 115: 1783-1806.
12. Mendez MF, Foti DJ (1997) Lethal hyperoral behaviour from the Klüver-Bucy syndrome. J Neurol Neurosurg Psychiatry 62: 293-294.
13. Funayama M, Nakagawa Y, Yamaya Y, Yoshino F, Mimura M, et al. (2013) Progression of logopenic variant primary progressive aphasia to apraxia and semantic memory deficits. BMC Neurol 13: 158.
14. Funayama M, Nakajima A (2015) Progressive transcortical sensory aphasia and progressive ideational apraxia owing to temporoparietal cortical atrophy. BMC Neurol 15: 231.
15. Morris CH, Hope RA, Fairburn CG (1989) Eating habits in dementia. A descriptive study. Br J Psychiatry 154: 801-806.
16. Ikeda M (2005) Eating behaviors in patients with dementia in their daily life. Japanese J Neuropsychol 21: 98-109.
17. Klüver H, Bucy PC (1937) Psychic blindness and other symptoms following bilateral temporal lobectomy in rhesus monkeys. Am J Physiology119: 352-353.
18. Klüver H, Bucy PC (1939) Preliminary anaylysis of functions of the temporal lobes in monkeys. Arch of Neurol Psychiatry 42: 979-1000.
19. Takahashi N, Kawamura M (2001) Oral tendency due to frontal lobe lesion. Neurology 57: 739-740.
20. Spiegel DR, Lamm K (2011) A case of utilization behavior and hyperorality following bilateral anterior cerebral artery infarct partially responsive to carbamazepine: Can both behaviors be attributed to lesions in different frontal lobe circuits? Psychosomatics 52: 563-567.
21. Lhermitte F (1983) 'Utilization behaviour' and its relation to lesions of the frontal lobes. Brain 106: 237-255.
22. Funayama M, Muramatsu T, Koreki A, Kato M, Mimura M, et al. (2017) Semantic memory deficits are associated with pica in individuals with acquired brain injury. Behav Brain Res 329: 172-179.
23. Sugishita M, Hemmi I (2010) Validity and reliability of Mini Mental State Examination-Japanese (MMSE-J): A preliminary report. Japanese J Cogn Neurosci 12: 186-190.
24. Kobayashi T, Hariguchi Y, Nishimura K (1988) The new clinical scale for rating of mental states of the elderly and the new clinical scale for rating of activities of daily living of the elderly. Clin Psychiatr 17: 1653-1668.
25. Matsui M, Mimura M, Tabuchi H, Kato K, Suzuki M, et al. (2008) Development of Japanese version of Friontal Behavioral Inventory (FBI). Higher Brain Function 28: 373-382.
26. Ralph MA, Jefferies E, Patterson K, Rogers TT (2017) The neural and computational bases of semantic cognition. Nat Rev Neurosci 18: 42-55.
27. Lambon Ralph MA, Ehsan S, Baker GA, Rogers TT (2012) Semantic memory is impaired in patients with unilateral anterior temporal lobe resection for temporal lobe epilepsy. Brain 135: 242-258.