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Case Report

Spaced Retrieval Training for Wernicke Korsakoff Syndrome with Long-Term Retrograde Amnesia

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Abstract

Objective: Wernicke-Korsakoff syndrome (WK) is characterized by peculiar memory deficits and peripheral symptoms, often including anterograde amnesia, retrograde amnesia, disorientation, confabulation, decreased spontaneity, and lack of insight of disease. We report herein a severe WK with severe short-term memory and long-term retrograde amnesia of 20- year durations, for which we performed cognitive rehabilitation centred on spaced retrieval training (SR) and achieved significant improvement in short-term memory impairment.

Methods: The patient was a 60-year-old man, presented with consistent with disorientation, impaired attention, short-term memory impairment, retrograde amnesia, confabulation, and lack of insight into his illness. On the neuropsychological examination, the FAB score was 15 points (out of 30), and RBMT score was 0 out of 24 points. After 3 months of usual neurocognitive rehabilitation, there was no significant improvement. Therefore, 2 episodes of SR training were performed.

Results: In the first episode, the patient was able to retain memory for 40 minutes or more in the 11th trial, and in the second episode, for 40 minutes or more in the 9th trial. Neuropsychological examination showed improvement of RBMT to 4 points.

Conclusions: This study is the first report of performing SR for WK. Memory impairment in WK is thought to involve faulty retrieval processes of “the encoded memory fragments”. SR is an effective cognitive rehabilitation tool for WK because it enhances the presentation of clues for correctly eliciting “the encoded memory-fragments”. The SR training method is promising and warrant further systematic studies in patients with WK syndrome.

Keywords: Spaced Retrieval Training; Wernicke Korsakoff Syndrome; Retrograde Amnesia; Cognition

Introduction

Korsakoff syndrome is a psychiatric disorder associated with polyneuritis, first reported by Korsakoff in the late 19th century [1]. It is more likely to be associated with chronic alcohol use, often after the acute syndrome of Wernicke encephalopathy, and is sometimes referred to as Wernicke-Korsakoff syndrome (WK) [2]. Encephalopathy is most often associated with severe and chronic alcohol abuse, but thiamine deficiency can be caused by other medical conditions producing malnourishment, including unbalanced nutrition, recurrent vomiting, chronic diarrhea, hyperthyroidism, systemic illness, or magnesium depletion [3,4]. Such cases are characterized by peripheral symptoms involving abducent nerve palsy, and peculiar cognitive deficits often with anterograde amnesia, retrograde amnesia, disorientation, confabulation, apathy, and lack of insight of disease. In terms of imaging findings, bilateral atrophy in the mammillary bodies and anterior thalamus have most often been implicated in the pathogenesis of WK, as well as in other sites in the periaqueductal gray, walls of the third ventricle, floor of the fourth ventricle, cerebellum, and frontal lobes [1,5,6]. In combination with other cognitive and behavioral deficits, in more severe forms of WK, this cause significant negative impact on daily life [7]. Usually, explicit episodic and remote memory is impaired, while short-term memory, semantic memory, and implicit memory are relatively preserved [8]. On the other hand, WK can cause retrograde amnesia of up to 20-30-year duration [9,10]. The Syndrome is also said to be associated with frontal lobe dysfunction likely caused by alcohol effects. Apart of thiamine replacement in the acute phase, permanent cognitive deficits especially memory impairment are usually addressed via cognitive rehabilitation [11]. Cognitive rehabilitation focuses on compensatory strategies to improve an individual's functioning and facilitate learning [12]. According to past reports, compensatory strategies such as error-less learning and the utilization of electronic devices are effective [7]. However, there are no reports of a direct approach to the underlying pathology of WK. In the present study, we report a severe short-term memory and frontal lobe dysfunction in addition to long-term retrograde amnesia spanning over a 20-year period. Cognitive rehabilitation centred on spaced retrieval training (SR) was performed for this patient.

Case

The patient was a 60-year old man, married, but living alone after retirement. He had no existing psychiatric or neurological history, drank beer daily, and smoked 12 cigarettes daily for 40 years. He was found at home collapsed and unconscious, and was taken to our hospital's emergency department. Neurological examination during transport revealed bilateral abduces nerve paralysis and vertical nystagmus in upward gaze. Fluid-Attenuated Inversion Recovery (FLAIR) Magnetic Resonance Imaging (MRI) of the head showed hyper intensity in the bilateral cerebral aqueduct, third ventricle, and mammillary body, which led to a diagnosis of WK and the patient being hospitalized (Figure 1). The patient's height was 170 cm and weight was 58 kg. He had no significant medical history. His blood chemistry test showed a vitamin B1 level of 17 ng/mlL, which was below the reference value (Normal value: 24-66 ng / mL). Therefore, thiamine replacement therapy was started on the same day. Neuropsychological evaluation at admission revealed Glasgow Coma Scale (GCS): Eye opening 2, Verbal response 2, Motor response 4 (date unknown), bilateral abduces nerve paralysis, and vertical nystagmus in upward gaze. He had no other abnormal findings in the cranial nerves. In addition, he did not have any obvious paralysis, sensory impairment, or cerebellar ataxia. He showed a slight decrease in his Achilles tendon reflexes. On the day 4 after symptom onset, we confirmed that his vital signs were normal and that he had no deep vein thrombosis, and then started rehabilitation. At his beginning, he was generally within in walking, climbing stairs, and activities of daily living. However, he had significant disorientation and short-term memory impairment, as well as retrograde amnesia. He also had a lack of insight of disease, was unaware of memory loss, with significant confabulation, answering out of context to the conversation. He replied 1998 for the data and "Heisei" for the Japanese era. When we suggested that the correct answer was "Reiwa" for the Japanese era, he replied, "I don't completely know that the Japanese era changed to Reiwa in 2019". The patient was unable to correctly identify the date or season. Neuropsychological testing at the beginning of rehabilitation showed the following scores Raven's Coloured Progressive Matrices (RCPM), 26 points (max, 36 points); Frontal Assessment Battery , 15 points (max 18 points); and Rivermead Behavioural Memory Test (RBMT), 0 points (max 24 points), indicating severe attention and memory impairment (Table 1) The Trail Making Test parts A and B could not be performed. One month after the intervention, training was conducted to compensate for the memory disorder by checking the date on the calendar and making memory notes. However, neither was helpful. In addition, the confabulation continued; e.g., "My daughter is also in the hospital with the same illness", and "The other day, I met and talked with 2 of my bosses at work. I am hospitalized and go to work. Since they go to the hospital too, my workplace is very hard work." The chronological order of questions regarding her family's age and social status was irregular. At 1 month of intervention, he was able to perform the Trail Making Test (TMT), but the Rivermead Behavioural Memory Test (RBMT) score remained at 0 (Table 1). Two months after onset, he still lacked insight into his cognitive deficits. He knew he was in the hospital but did not know why and gave confabulatory responses such as: "I have been in and out of the

hospital since last year," "I was in another hospital for 3 days until yesterday," and "I currently live with my younger brother." All of these statements were contrary to the facts. Regarding the degree of memory impairment at this time, short-term memory impairment and retrograde amnesia became prominent. As for his autobiographic (personal) memory, while he was able to talk about his family life more than 10 years ago, roughly 20 years of memories about past external world events were lost. When we probed him around past events by direct questioning or using keywords as clues, he had partial knowledge of events like the 9/11 in the US, while he reported knowing more about "the Nagano Olympics." He had no recollection of the major earthquakes in Japan, the Tokyo Olympics, or the COVID-19 pandemic. In his training, he was able to perform tasks centred on attention and memory; he would forget the intent of the question in about 10-20 minutes. His performance on neuropsychological tests, including the RBMT, did not improve. He was unable to establish the current date using memory notes or calendars. He was also presented with a total of 8 photographs of successive prime ministers of Japan who served over the last 2 decades, without any clues about them. He was able to correctly identify the names of the people in all photos but when asked who the current prime minister was, he replied, "I know Koizumi has already quit, but I don't know after that, I don't think it's Abe. Others aren't prime ministers." He was able to correctly identify only the prime minister who had served 20 years ago. At three months after his intervention, he was able to retain immediate memory compared to his onset (retention of information for a few seconds). However, he continued to have the defects in recognition and delayed reproduction tasks, and his memory retention time was short. In addition, he continued to have short-term memory impairment and retrograde amnesia. Therefore, we decided to use SR as rehabilitation for him.

Methods

SR is a memory training method that ultimately leads to information retention and recall over a long period of time while extending the time interval for items and actions that patients want to remember [13]. The first task was to remember the name of the therapist in charge ("What is my name?"). When there was an erroneous reaction, the therapist immediately corrected it, provided new information, immediately returned to the correct time interval, and then asked the question again. The time interval was set to 5 s, 10, s, 20 s, 30 s, 1 min, 2 min, 5 min, 10 min, 20 min, or 40 min. Since the maximum rehabilitation time in 1 session was 40 min, the memory retention time of 40 min was set as the maximum memory retention time in a single day. We decided to ask the same question again at the start of rehabilitation the next day. If there was an erroneous reaction, the question was repeated from the retention time 1 step before the maximum retention time on the previous day. For example, if the memory was retained for 40 minutes and there was an erroneous reaction the next day, the retention time until the next question would be set to 20 min. If there was an erroneous reaction at this time, the retention time was set to 1 step before that. If it was possible to hold the task for 1 day, the next task was to be executed. The second question was, "What is today's date?" The therapist who asked this question was a different therapist than the therapist who asked the first question. The time interval was set to be the same as for the first task. When performing the 2 tasks at the same time, the rehabilitation time was 40 minutes per session, with 2 sessions per day.

Results

Table 1 shows the results of neuropsychological examinations during the entire course from baseline to 4 months after the start of the intervention. At the pre-intervention, the Rivermead Behavioural Memory Test (RBMT) scored 0 to 1 and the non-Relationship of Standard verbal paired-associate learning test (s-PA) scored 0, indicating severe memory impairment. The Kohs' Block-Design Test was below the cut-off, suggesting a decline in cognitive function. However, the Frontal Assessment Battery (FAB) was 15 points, and frontal lobe function was relatively maintained. At the time of intervention, WAIS-Ⅲ scores were: verbal IQ, 104; performance IQ, 87; verbal comprehension, 99; perceptual organization, 89; working memory, 100; processing speed, 69; and total IQ, 96. Performance IQ was lower than verbal IQ. Performance IQ and perceptual organization were slightly lower than total IQ. The processing speed was below the lower limit. Figure 2 shows the change in memory retention time with SR. In the first task, the patient was able to achieve up to 20 minutes of memory retention on the first day. After that, the memory retention time remained between 20-40 minutes until the 10th trial. For the first time in the 11th trial, it became possible to retain memory for more than 1 day. After that, the training was continued until the 16th trial, and the patient was able to sustain memory retention for 1 day or more. The second task was started at the 12th trial, when the first task of retaining memory for 1 or more days in a row for 2 or more days was completed. The second task reached 20 minutes of memory retention in a systematic the First trial. After that, the memory retention times ranging from 10 -40 minutes continued. Finally, the 9th trial made it possible to retain memory for more than 1 day. A neuropsychological examination 1 month after the SR intervention showed significant improvement in TMT, s-PA, and Paced Auditory Serial Addition Task (PASAT) compared to before the intervention (Table 1). In addition, RBMT showed a significant improvement of 4 points (Table 1). Although severe memory loss remained, he was discharged with the support of his caregiver.

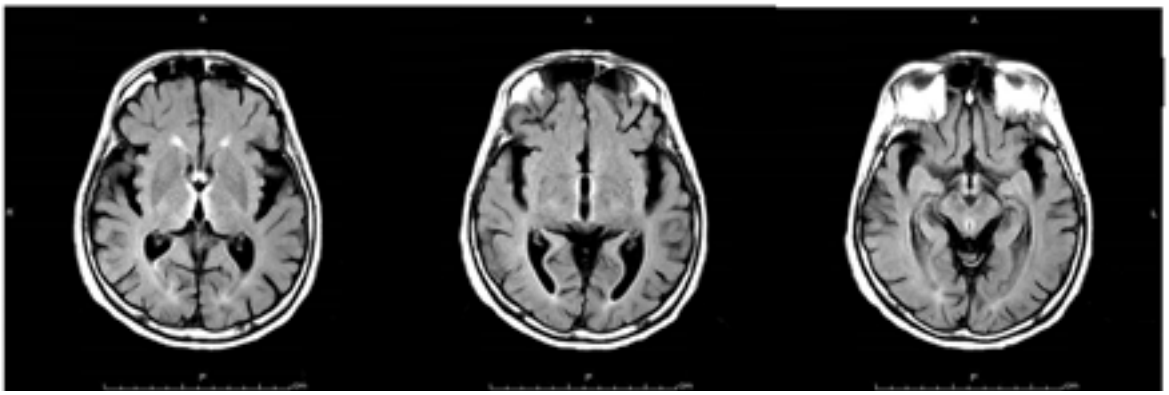

Figure 1: Fluid-attenuated inversion recovery-magnetic resonance imaging of the head: Hyper intensity was observed in the bilateral cerebral aqueduct, third ventricle, and mammillary body.

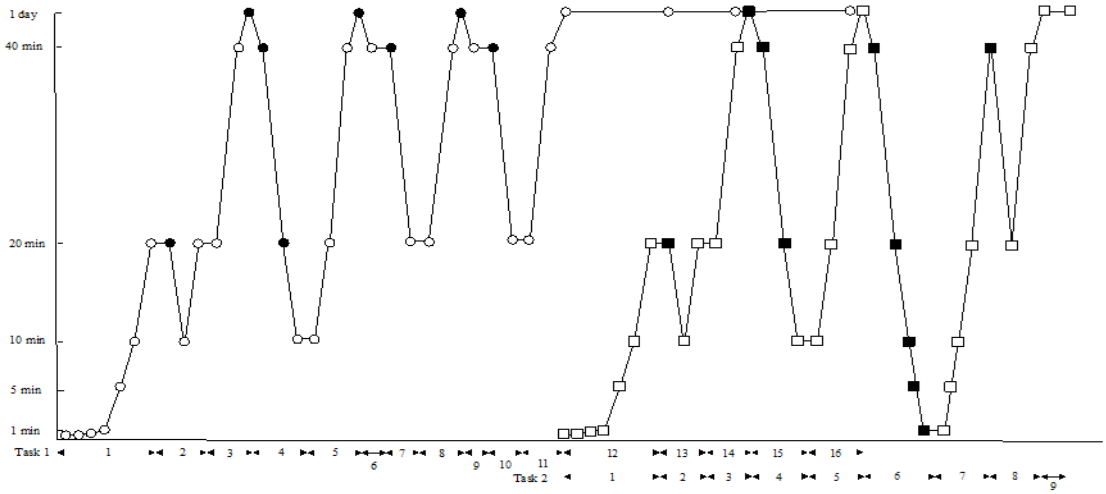

Figure 2.

Figure 2: Transition of memory retention time using the spaced retrieval training (SR) method: ○ indicates a correct answer in Task 1, and ● indicates an incorrect answer in Task 1. □ indicates the correct answer in Task 2, and ■ indicates the incorrect answer in Task 2. The horizontal axis shows the number of days calculated from the training implementation start date for Task 1 and Task 2. The vertical axis shows the memory retention time of each task.

		At the start of rehabilitation	1 months after onset	2 months after onset	3 months after onset At the start of SR training	4 months after onset 1 months after SR training
RBMT [0–24]		0	0	1	1	4
TMT(s)	A	× *	335			158
	B	× *	259			93
FAB [0–18]		15		15	15	16
Kohs' Block-Design Test		72.3	82.8			
S-PA	Relationship /		2-4-3/		4-4-5/	6-8-9/

	non-Relationship		0-0-0		0-0-1	0-0-1
PASAT(%)	2' test				50	67
	1' test				38	52
RCPM [0-36]		26			35	35

TMT-A: Trail Making Test part A, TMT-B: Trail Making Test part B, RBMT: Rivermead Behavioural Memory Test, PASAT: Paced Auditory Serial Addition Task, FAB: Frontal Assessment Battery, S-PA: Standard verbal paired-associate learning test, RCPM : Raven's Coloured Progressive Matrices. *: The patient could not understand the rules of the test.

Table 1: Results of neuropsychological examination.

Discussion

We report herein a severe short-term memory and frontal lobe dysfunction in addition to long-term retrograde amnesia of 20-year duration. Cognitive rehabilitation centred on SR as performed for this patient, and improvement of memory impairment was observed. This report is a valuable contribution to the literature given the scarcity of reports on long-term retrograde amnesia (20 years) and on cognitive rehabilitation in this population. The general clinical features and imaging findings of WK are anterograde amnesia, retrograde amnesia, disorientation, confabulation, apathy, and lack of insight of disease, with peculiar memory impairment and peripheral symptoms [2,4]. In this case, anterograde amnesia, retrograde amnesia, disorientation, confabulation, and lack of insight of disease were observed. Hyper-intensity areas were observed in the ventricle and mammillary body, similar to previous reports. Mammillary body shrinkage is observed in upwards of 60%-80% of post-mortem neuropathology studies and in the MRI study, has been observed in 78% of WE patients [14-16]. It has also been suggested that disruption of neural network circuits of memory involving interactions between the thalamus, hippocampus, frontal lobe, and cerebellum, including the mammillary body, is involved in memory impairment in WK [17-19]. Various cognitive dysfunctions in this case are suggested to be disorders of these neural network circuits. From a neuropsychological examination point of view, WK is thought to involve disproportionate memory impairment; namely, it is a memory disorder that is disproportionately recognized even though general intelligence measured by WAIS and the elemental attention function and language function shown by the repetition [1]. In this case, as well, cognitive function was relatively maintained at about 2-3 months after the onset of the acute phase. In the aspect of WAIS-Ⅲ from the measured total IQ and his past work history, we recognized that the expected IQ was within the normal range from before the onset. Performance IQ and perceptual organization were slightly lower than total IQ. However, it was within the lower limit when converted from total IQ. The processing speed was below the lower limit. We have recognized that the decrease in performance IQ is an effect of processing speed. The reduced processing speed was an expected result from the course of onset and treatment. In terms of memory, there were obvious clinical findings of memory impairment, with an RBMT score of 0-1. The results of these neuropsychological tests likely reflects the above-mentioned disproportionate memory impairment.

Disorders of long-term memory and episodic memory that are characteristic of WK include extensive retrograde amnesia, remote memory impairment, and temporal gradient. Patients with WK usually have impaired remote memory, such as memory of their past events (autobiographical memory), personal historical semantic memory, past social events, and memory of famous people. However, very old memories of childhood and adolescence, referred to as temporary gradients, tend to be preserved [1,8,20]. These three signs were noted with respect to some episodic memory findings in this case, especially in the case of public events and recognition of famous faces. As described above, the process of memory impairment in WK suggests impairment of the neural network of memory including the mammillary body, and it is thought that these impairments involve 2 elements: faulty retrieval processes and encoding deficits [8]. Faulty retrieval processes are thought to result from an inability to access information in long-term memory rather than an inability to store information in long-term memory [21]. Encoding is one of the basic processes of memory. It refers to the process of capturing information, retaining it as memory, and fixing it [22]. Support for this evidence of encoding deficits initially came from reports of verbal and non-verbal learning deficits in long-term alcoholics as well as reports that the magnitude of anterograde memory impairment in KS positively correlates with the magnitude of remote memory impairment [22,23]. The face photographs test before SR in this case were able to evoke all the memory-retained person names based on the facial clues provided by the photographs. Conversely, it appeared that the characteristic episodes of distinct individuals (i.e. they are the all past prime ministers.), which are fragmentary, could not be correctly extracted due to the influence of faulty retrieval processes. Therefore, this case supports the hypothesis of faulty retrieval processes and reaffirms that it is important to present clues in order to correctly retrieve "the encoded memory-

fragments". Moreover, as will be described below, SR can serve as a training method for these disorders. With respect to confabulation, true memory and false memory tend to be confused, which is one of the clinical features of WK [24].Confabulation has also been linked to deficits in the controlled aspects of memory retrieval, such as post-retrieval monitoring and verification [25,26]. Previous reports have shown that WK patients demonstrate improvements in memory performance when tested by recognition or cued recall [9,27,28]. Therefore, search support and errorless learning using such cued recall are important in rehabilitation, and these may be considered compensatory strategies by giving clues for improvement and reacquisition of activities of daily living.

Therefore, for the current case, we performed SR to address the patient's memory impairment due to WK. SR has been used mainly for dementia, which causes episodic memory impairment, and its effectiveness has been reported in past studies [29-32]. On the other hand, there is a report of SR being used to improve activities of daily living for memory impairment caused by traumatic brain injury [33]. However, in studies of cognitive rehabilitation and training conducted for WK, recognition testing or cued retrieval was used, but none reported using SR [9,27,28]. SR training was performed for our case and improvement in neuropsychological examination results was observed. It is suggested that SR training enhances the ability to correctly extract "the encoded memory-fragments" for faulty retrieval processes, which is the concept of memory impairment in WK, and contributes to the improvement of memory function. These are some limitations to our case study. Initially, we only carried out evaluations during a short period after SR. It is however important to perform long-term follow-up in the future. Second, our patient showed RBMT improvement from 1 point to 4 points, but still presented with severe memory impairment. Since this case had severe memory impairment, we used the names and dates as a training task. However, in order for SR to be used more progressively, it may be necessary to verify its effectiveness in other tasks including activities of daily living. Third, in WK, the medial aspect of the cerebrum is damaged, and these changes in cerebral blood flow and functional brain imaging are academically interesting fields. Comprehensive examination of the neuropsychological examinations findings and the changes in functional brain images may help verify whether SR is a direct therapeutic strategy for memory impairment [11].

Conclusion

We performed cognitive rehabilitation using SR for a WK case with severe short-term memory and frontal lobe dysfunction in addition to long-term retrograde amnesia of 20-year duration. As a result, a neuropsychological examination showed improvement of memory impairment. WK is thought to involve faulty retrieval processes of "the encoded memory-fragments" as a process of memory impairment. SR is an effective cognitive rehabilitation tool for WK because it enhances the presentation of clues for correctly eliciting "encoded memory-fragments".

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