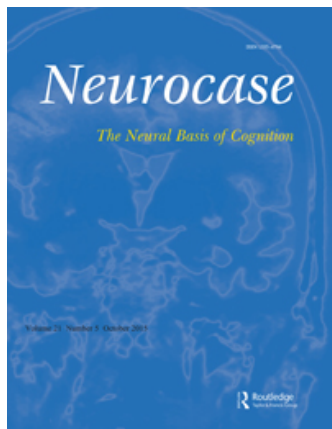


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Publication details, including instructions for authors and subscription information:
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Published online: 28 Oct 2014.

To cite this article: Fumiko Kusunoki Nakamoto, Michiko Tsutsumiuchi, Meiko Hashimoto Maeda, Yoshikazu Uesaka & Katsuhiko Takeda (2015) Memory impairment following right cerebellar infarction: a case study, *Neurocase: The Neural Basis of Cognition*, 21:5, 660-664, DOI: [10.1080/13554794.2014.969277](https://doi.org/10.1080/13554794.2014.969277)

To link to this article: <http://dx.doi.org/10.1080/13554794.2014.969277>

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Memory impairment following right cerebellar infarction: a case study

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(Received 25 April 2014; accepted 16 September 2014)

We reported a patient with a right cerebellar infarction who showed anterograde amnesia. Cognitive dysfunction caused by cerebellar lesions was called cerebellar cognitive affective syndrome, and deactivation of the contralateral prefrontal cortex function due to disconnections of cerebello-cerebral fiber tracts have been hypothesized as mechanism underlying the syndrome. The episodic memory impairment, however, could not be supported by the same mechanism because the prefrontal lesions cannot cause amnesia syndrome. The feature of the impairment of our patient was similar to that of diencephalic amnesia, and a single photon emission computed tomography study showed a relative hypoperfusion in the right cerebellar hemisphere and left anterior thalamus. We considered that the memory deficit was caused by the dysfunction of the thalamus, which is a relay center of the cerebello-cerebral connectivity network.

Keywords: cerebellar cognitive affective syndrome; cerebellar stroke; cerebellum; episodic memory; amnesia

Over the last few decades, neuroimaging studies and neuropsychological investigations have clarified the role of the cerebellum in higher-order cognitive functions. Schmahmann and Sherman evaluated patients with different cerebellar lesions, observing cognitive dysfunction and named this clinical entity “cerebellar cognitive affective syndrome (CCAS)” (Schmahmann & Sherman, 1998). This syndrome consists of executive, visuospatial, linguistic, and emotional deficits.

Disconnections of cerebello-cerebral fiber tracts have been hypothesized as the mechanism underlying the syndrome (O’Halloran, Kinsella, & Storey, 2012). Some functional magnetic resonance imaging (MRI) studies revealed the cerebello-cortical pathway with multiple fiber tracts that terminate within the regions of the contralateral prefrontal cortex (O’Halloran et al., 2012). According to previous reports, cognitive impairments caused by a cerebellar lesion have been often explained by the functional suppression of the contralateral frontal lobe (Baillieux et al., 2006; Cooper et al., 2012; Lagarde, Hantkie, Hajjioui, & Yelnik, 2009; Mariën et al., 2008, 1996; Neau, Arroyo-Anllo, Bonnaud, Ingrand, & Gil, 2000; Peterburs, Bellebaum, Koch, Schwarz, & Daum, 2010; Suenaga et al., 2008).

We herein report of a patient with CCAS whose deteriorations of executive function and language function were supported by a mechanism described in previous reports (O’Halloran et al., 2012); however, the episodic memory impairment could not be supported by the same

mechanism. Thus, we consider other possible causes of memory dysfunction.

Case report

Following 12 days of continuous headaches, a 42-year-old right-handed man with 16th grade education presented at our emergency room with acute onset of disturbance of consciousness. The patient was a government worker and did not have any history of serious illness. His neurologic examination revealed mild drowsiness, dysarthria, and minor motor dysfunctions such as mild cerebellar ataxia and dysmetria. Following 24 hr of admission, disturbance of consciousness, dysarthria, and motor features almost disappeared.

Cranial MRI showed no structural damage at the supratentorial level including the thalamus, but revealed acute cerebellar infarction involving the vermis and superior part of the right cerebellar hemisphere (Figure 1(a–c)). Cerebral angiography showed a dissection of bilateral vertebral arteries and the basilar artery. A quantified N-isopropyl-p [¹²³I] iodoamphetamine single photon emission computed tomography (IMP SPECT) of the brain, performed 3 weeks after the onset of consciousness disturbance revealed a hypoperfusion in the right hemis cerebellum and the left frontotemporal region including the left ventrolateral prefrontal cortex and a slightly decreased perfusion in the left anterior thalamus (Figure 1(d–f)). The results of electroencephalography, performed 3 weeks after the onset were

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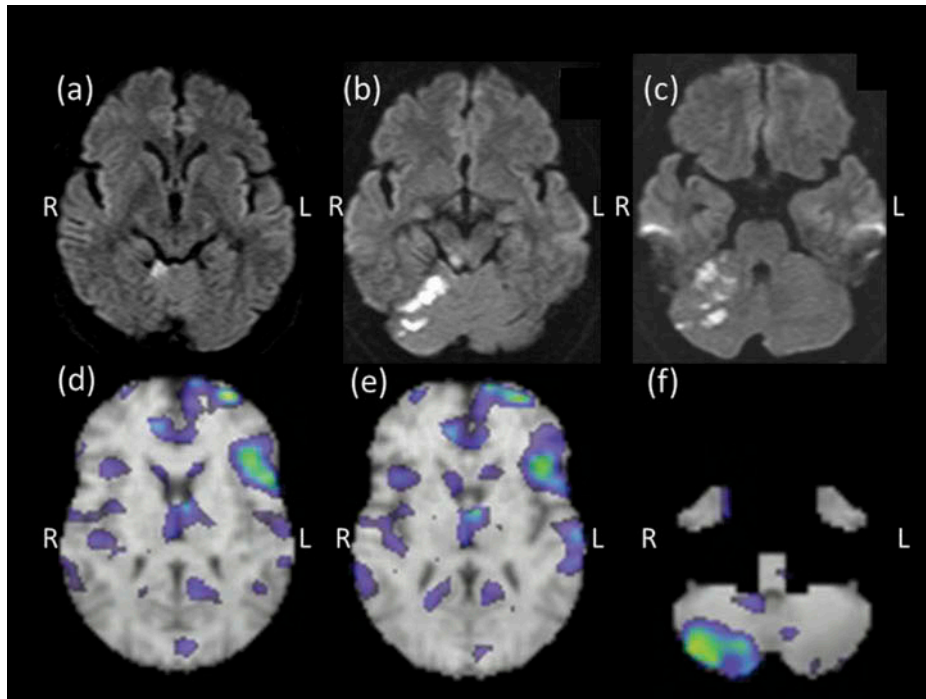


Figure 1. Brain MRI axial diffusion weighted imaging showing hyperintense signals in the anterior lobe of the superior right cerebellar hemisphere (right superior cerebellar artery). No structural damage was observed at the supratentorial level (a–c). Quantified IMP single photon emission CT (IMP SPECT) performed 22 days after the onset of consciousness disturbance showed hypoperfusion not only in the vermis and hemisphere where the infarction was found but also in the left inferior frontal area and anterior thalamus (d–f). [To view this figure in color, please see the online version of this journal.]

found to be within normal limits, suggesting that he had no disturbance of general brain function.

In his daily life, although he had a high educational level, the patient tended to ask the same questions several times and tended to forget the nursing care he had already received. To evaluate his cognitive dysfunction, neuropsychological evaluation was started 13 days after the onset.

His performance on Cognistat (The Neurobehavioral Cognitive Status Examination) was markedly deficient on the subtest of memory (Table 1). The Wechsler Memory Scale-Revise revealed a discrepancy of 51 index points between visual memory (index = 113) and verbal memory (index = 62). The index of delayed recall was 64, which

showed the episodic memory impairment. His scores revealed that his memory function was impaired for his age, and he had a deficit of verbal memory rather than visuospatial memory.

In the Rey auditory verbal learning test (revised Japanese version), his delayed recall score (3/15) was low, whereas his score in the recognition part was normal. These findings suggest that encoding of novel aspects of information into episodic memory was intact whereas memory retrieval was affected. While he was recalling the words, confabulation occurred.

The patient had a low score in the Rule Shift Card Test included in the Behavioral Assessment of the Dysexecutive Syndrome (BADs). In the modified Stroop

Table 1. Cognistat profile.

Subtest of Cognistat (test range)	Loc	Ori (0–12)	Atten (0–12)	Language					Reasoning		
				Comp (0–12)	Rep (0–12)	Nam (0–12)	Const (0–12)	Mem (0–12)	Calc (0–12)	Sim (0–12)	Judg (0–12)
1 week after stroke	Alrt	10	10	10	11	9	11	6	10	9	10
8 weeks after stroke	Alrt	10	10	10	11	10	11	9	10	10	10

Notes: Loc = level of consciousness; Ori = Orientation subtest; Atten = Attention subtest; Comp = Comprehension subtest; Rep = Repetition subtest; Nam = Naming subtest; Const = Construction subtest; Mem = Memory subtest; Calc = Calculations subtest; Sim = Similarities subtest; Judg = Judgment subtest; Alrt = Alert.

test, he was unable to disregard the verbal content. These results suggest that his executive function involving divided and switching attention was disturbed.

His total Wechsler intelligence quotient (IQ) was 79. This result was caused by decreased verbal IQ and deterioration of executive function caused by diaschisis in the left frontal cortex. Although he did not show aphasia in the Western aphasia battery (WAB), he showed mild impairment of language function, such as impairment of comprehension of the meaning of written sentences, impairment of speech production, and reduced phonemic fluency. These findings suggest decreased verbal IQ that was not due to aphasia.

A repeat neuropsychological evaluation 4–6 weeks after his stroke showed slight improvement. Table 2 shows a summary of his cognitive assessments at 2–3 weeks, 4–6 weeks, and 4 months after the stroke. Follow-up investigations using Cognistat 8 weeks postonset revealed an improvement of the memory profile (Table 1).

Discussion

The patient developed episodic memory deficit and deteriorations of executive function and language function. The deteriorations of executive function and language function in CCAS were noted in previous reports, in which the mechanism underlying the syndrome was deactivation of the contralateral prefrontal cortex function due to the loss of excitatory impulses through cerebellar cortical pathways.

The episodic memory impairment, however, could not be supported by this mechanism because the prefrontal lesions cannot cause an amnesic syndrome. Although the lesions can affect the memory process and cause secondary long-term memory deficits, such as source memory impairment, false memory, and prospective memory impairment, the memory deficits of our patient differed from those findings. Therefore, we needed to evaluate the mechanism underlying the condition, and we considered some possible causes of memory dysfunction.

First, we considered that the decreased blood flow in the left anterior thalamus caused the amnesia in our patient, because his brain SPECT showed decreased perfusion in this region. The feedback or efferent loop originates from the deep nuclei of the cerebellum, which project to the nuclei of the thalamus. In turn, the nuclei of the thalamus project to the motor and premotor cortices and also to nonmotor areas including the prefrontal cortex (Marien, Engelborghs, Fabbro, & De Deyn, 2001; Smet, Paquier, Verhoeven, & Mariën, 2013). Several clinical reports suggest evidences for cerebello-thalamo-cortical pathways. Kim et al. reported amnesia syndrome following left anterior thalamic infarction, and the brain SPECT image revealed decreased uptake in the ipsilateral fronto-temporoparietal cortex and contralateral cerebellum that caused by disconnection of the cerebello-thalamo-cortical

pathways (Kim, Hong, & Roh, 1994). The phenomenon of cerebello-cerebral diaschisis was also shown in patients of posterior fossa syndrome, and the ^{99m}Tc -Ethyl cysteinate dimer SPECT results showed decreased perfusion in the thalamus as well as an area of hypoperfusion in the vermis (Baillieux et al., 2006; Mariën et al., 2013). It is considered that disconnection of cerebello-thalamic tracts causes functional suppression of the left anterior thalamus. Besides, several reports suggest that a thalamic lesion can cause episodic memory impairment (Kim et al., 1994). Diencephalic amnesia is characterized by the clinical features of normal intelligence and immediate memory, anterograde and retrograde amnesia, disorientation, anosodiaphoria, and confabulation. Our patient did not show significant retrograde amnesia and encoding and storage deficits; however, the feature of confabulation was similar to that of diencephalic amnesia. It is considered that the memory deficit of our patient was caused by the dysfunction of the thalamus, which is a relay center of the cerebello-cerebral connectivity network.

Second, we considered that the cerebellum itself has a role in episodic memory. Both neuroimaging and neuropsychological lesion studies suggest that the cerebellum contributes to verbal working memory processes. Some neuropsychological and neuroimaging studies have shown indirect supports for the assumption that the cerebellum contributes to episodic memory process because of involvement of verbal working memory processes (Davachi, Maril, & Wagner, 2001; Ranganath, Cohen, & Brozinsky, 2005). These concepts are supported by imaging studies that demonstrate an overlap between brain regions involved both in working memory processing and in long-term memory encoding (Fliessbach, Trautner, Quesada, Elger, & Weber, 2007). Impaired working memory of our patient was, however, not severe, and we speculate that decline in working memory may not be involved in his episodic memory disturbance. On the other hand, using functional MRI, some studies suggested that the cerebellum supports episodic memory formation and retrieval, which was not explained by involvement of verbal working memory (Fliessbach, Weis, Klaver, Elger, & Weber, 2006; Weis, Klaver, Reul, Elger, & Fernández, 2004). It is speculated that activations of the cerebellar hemispheres during episodic memory retrieval is due to the induction of imagined manipulations by concrete words such as tools because the cerebellum is known to be involved in motor imagery (Fliessbach et al., 2006). These studies suggest that the cerebellum has a role in episodic memory.

On comparing the performance after cognitive rehabilitation with the performance immediately after stroke, an improvement of verbal IQ was shown while the scores of performance IQ, processing speed, verbal memory, and delayed recall were almost unchanged or showed partial recovery. The observation suggested that it is difficult to

Table 2. Neuropsychological test results in the present case.

	2–3 weeks after stroke	4–6 weeks after stroke	4 months after stroke
Measures			
General cognition ability and memory			
HDS-R (/30)	27	30	
MMSE (/30)	30		
Frontal Assessment Battery (/18)		15	
WAIS-III			
Total IQ	79		93
Verbal IQ	77		101
Performance IQ	85		83
Verbal comprehension	78		105
Sensory integration	99		95
Working memory	85		98
Processing speed	78		78
Wechsler Memory Scale-R			
Verbal memory	62		82
Visual memory	113		115
General memory	73		90
Attention/concentration	103		101
Delayed recall	64		82
RBMT (/24)			
Standard profile	16	19	
Scleening	8	8	
Learning and retrieval			
Rey Auditory Verbal Learning Test			
Immediate recall 1st (/15)	5	4	7
2nd (/15)	9	8	8
3rd (/15)	8	10	9
4th (/15)	6	10	12
5th (/15)	4	9	13
Recognition (/44)	42	41	44
Delayed recall (/15)	3	7	10
Rey–Osterrieth Figure (/36)			
Copy	35		
Delayed recall	18.5		
Attention			
Trail making Test A (execution time)	106 s	94 s	
Trail making Test B (execution time)	105 s	105 s	
BADS			
Rule Shift Card (/4)	1		
Action Program (/4)	3		
Key Search (/4)	4		
Temporal Judgement (/4)	2		
Zoo Map (/4)	4		
Modified Six Elements (/4)	3		
Overall profile (/24)	17		
Standardized score	95		
Age-corrected score	93		
Stroop			
Part I (execution time)	25 s		
Part III (execution time)	29 s		
Language function			
WAB (AQ%)			99.8

Note: HDS-R, Hasegawa's Dementia Scale Revised; MMSE, Mini-Mental State Examination; WAIS-III, Wechsler Adult Intelligence Scale-Third Edition; AQ, Aphasia quotient.

improve executive dysfunction and memory impairment, while language impairment tends to improve earlier than these functions. In this patient, the language impairment is caused by the deactivation of the contralateral cortex

function due to disconnections of cerebello-cerebral fiber tracts, and the language impairment showed improvement following functional recovery after cerebellar damage. On the other hand, memory impairment was caused by

disconnection of neural circuits through the anterior thalamus, and it could not be improved immediately after recovery of cerebellar function. Although the executive dysfunction is also caused by the deactivation of the left frontal cortex function due to diaschisis of the cerebellar lesion, improvement of executive dysfunction was delayed. We speculated that the cause of the delay of improvement of executive dysfunction is the delay of improvement of memory function because executive function was associated strongly with memory function rather than with language function which showed significant improvement.

In summary, with a lesion in the right cerebellum, the patient showed episodic memory impairment, and we observed diaschisis in the anterior thalamus on brain SPECT. This finding suggests that memory disturbance in the right cerebellum caused by a lesion is a type of disconnection syndrome interrupting the cerebellar modulation of neural circuits linked to the anterior thalamus.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

- Baillieux, H., De Smet, H. J., Lesage, G., Paquier, P., de Deyn, P. P., & Mariën, P. (2006). Neurobehavioral alterations in an adolescent following posterior fossa tumor resection. *The Cerebellum*, 5, 289–295. doi:10.1080/14734220601009606
- Cooper, F. E., Grube, M., Von Kriegstein, K., Kumar, S., English, P., Kelly, T. P., ... Griffiths, T. D. (2012). Distinct critical cerebellar subregions for components of verbal working memory. *Neuropsychologia*, 50, 189–197. doi:10.1016/j.neuropsychologia.2011.11.017
- Davachi, L., Maril, A., & Wagner, A. D. (2001). When keeping in mind supports later bringing to mind: Neural markers of phonological rehearsal predict subsequent remembering. *Journal of Cognitive Neuroscience*, 13, 1059–1070. doi:10.1162/089892901753294356
- Fliessbach, K., Trautner, P., Quesada, C. M., Elger, C. E., & Weber, B. (2007). Cerebellar contributions to episodic memory encoding as revealed by fMRI. *NeuroImage*, 35, 1330–1337. doi:10.1016/j.neuroimage.2007.02.004
- Fliessbach, K., Weis, S., Klaver, P., Elger, C. E., & Weber, B. (2006). The effect of word concreteness on recognition memory. *Neuropsychologia*, 32, 1413–1421.
- Kim, M. H., Hong, S. B., & Roh, J. K. (1994). Amnesia syndrome following left anterior thalamic infarction; with intrahemispheric and crossed cerebro-cerebellar diaschisis on brain SPECT. *Journal of Korean Medical Science*, 9, 427–431. doi:10.3346/jkms.1994.9.5.427
- Lagarde, J., Hantkic, O., Hajjioui, A., & Yelnik, A. (2009). Neuropsychological disorders induced by cerebellar damage. *Annals of Physical and Rehabilitation Medicine*, 52, 360–370. doi:10.1016/j.rehab.2009.02.002
- Mariën, P., Brouns, R., Engelborghs, S., Wackenier, P., Verhoeven, J. O., Ceulemans, B., & De Deyn, P. P. (2008). Cerebellar cognitive affective syndrome without global mental retardation in two relatives with Gillespie syndrome. *Cortex: A Journal Devoted to the Study of the Nervous System and Behavior*, 44, 54–67. doi:10.1016/j.cortex.2005.12.001
- Mariën, P., De Smet, E., de Smet, H. J., Wackenier, P., Dobbeleir, A., & Verhoeven, J. O. (2013). “Apraxic dysgraphia” in a 15-year-old left-handed patient: Disruption of the cerebello-cerebral network involved in the planning and execution of graphomotor movements. *Cerebellum (London, England)*, 12, 131–139. doi:10.1007/s12311-012-0395-1
- Mariën, P., Engelborghs, S., Fabbro, F., & De Deyn, P. P. (2001). The lateralized linguistic cerebellum: A review and a new hypothesis. *Brain and Language*, 79, 580–600. doi:10.1006/brln.2001.2569
- Mariën, P., Scaerens, J., Nanhoe, R., Moens, E., Nagels, G., Pickut, B. A., ... De Deyn, P. P. (1996). Cerebellar induced aphasia: Case report of cerebellar induced prefrontal aphasic language phenomena supported by SPECT findings. *Journal of the Neurological Sciences*, 144, 34–43. doi:10.1016/S0022-510X(96)00059-7
- Neau, J.-P., Arroyo-Anllo, E., Bonnaud, V., Ingrand, P., & Gil, R. (2000). Neuropsychological disturbances in cerebellar infarcts. *Acta Neurologica Scandinavica*, 102, 363–370. doi:10.1034/j.1600-0404.2000.102006363.x
- O’Halloran, C. J., Kinsella, G. J., & Storey, E. (2012). The cerebellum and neuropsychological functioning: A critical review. *Journal of Clinical and Experimental Neuropsychology*, 34, 35–56. doi:10.1080/13803395.2011.614599
- Peterburs, J., Bellebaum, C., Koch, B., Schwarz, M., & Daum, I. (2010). Working memory and verbal fluency deficits following cerebellar lesions: Relation to interindividual differences in patient variables. *Cerebellum (London, England)*, 9, 375–383. doi:10.1007/s12311-010-0171-z
- Ranganath, C., Cohen, M. X., & Brozinsky, C. J. (2005). Working memory maintenance contributes to long-term memory formation: Neural and behavioral evidence. *Journal of Cognitive Neuroscience*, 17, 994–1010. doi:10.1162/0898929054475118
- Schmahmann, J. D., & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain*, 121, 561–579. doi:10.1093/brain/121.4.561
- Smet, H. J. D., Paquier, P., Verhoeven, J., & Mariën, P. (2013). The cerebellum: Its role in language and related cognitive and affective functions. *Brain and Language*, 127, 334–342. doi:10.1016/j.bandl.2012.11.001
- Suenaga, M., Kawai, Y., Watanabe, H., Atsuta, N., Ito, M., Tanaka, F., ... Sobue, G. (2008). Cognitive impairment in spinocerebellar ataxia type 6. *Journal of Neurology, Neurosurgery & Psychiatry*, 79, 496–499. doi:10.1136/jnnp.2007.119883
- Weis, S., Klaver, P., Reul, J., Elger, C. E., & Fernández, G. (2004). Temporal and cerebellar brain regions that support both declarative memory formation and retrieval. *Cerebral Cortex*, 14, 256–267. doi:10.1093/cercor/bhg125