

Reversible Verbal and Visual Memory Deficits after Left Retrosplenial Infarction

Jong Hun Kim, M.D.* , Kwang-Yeol Park, M.D.† , Sang Won Seo, M.D.* , Duk L. Na, M.D.* ,
Chin-Sang Chung, M.D.* , Kwang Ho Lee, M.D.* , Gyeong-Moon Kim, M.D.*

*Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

†Department of Neurology, Chung-Ang University Medical Center, Chung-Ang University School of Medicine, Seoul, Korea

The retrosplenial cortex is a cytoarchitecturally distinct brain structure located in the posterior cingulate gyrus and bordering the splenium, precuneus, and calcarine fissure. Functional imaging suggests that the retrosplenium is involved in memory, visuospatial processing, proprioception, and emotion.

We report on a patient who developed reversible verbal and visual memory deficits following a stroke. Neuropsychological testing revealed both anterograde and retrograde memory deficits in verbal and visual modalities. Brain diffusion-weighted and T2-weighted magnetic resonance imaging (MRI) demonstrated an acute infarction of the left retrosplenium.

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INTRODUCTION

The main structures related to human memory are the Papez circuit, the basolateral limbic circuit, and the basal forebrain, which communicate with each other through white-matter tracts. Damage to these structures (including the communication tracts) from hemorrhages, infarctions, and tumors can result in memory disturbances.^{1,2} In addition to these structures, Valenstein et al. suggested that the retrosplenium could be a supplementary pathway of the limbic system connecting the anterior thalamus and medial temporal lobe structures.³ The retrosplenium is located in the posterior cingulate cortex surrounding the splenium, and is a cytoarchitecturally distinct structure forming Brodmann areas 29 and 30⁴ (Fig. 1).

We report on a patient who developed both verbal and visual memory deficits after an acute infarction of the retrosplenial cortex.

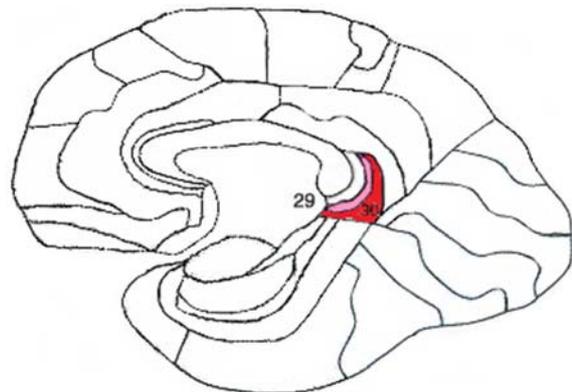


Figure 1. Location of the retrosplenium (Brodmann areas 29 and 30) in a schematic of a brain sagittal section.

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Department of Neurology, Samsung Medical Center Sungkyunkwan University School of Medicine Ilwon-Dong 50, Kangnam-gu, Seoul, 135-710, Korea
Tel: +82-2-3410-3598,3599, Fax: +82-2-3410-0052, E-mail: kimgm@smc.samsung.co.kr

CASE REPORT

A 57-year-old right-handed man who had suffered from diabetes mellitus for 10 years was admitted to an emergency room due to acute memory loss. He had no previous history of cognitive impairment. One month prior to admission he was diagnosed with syphilis and began treatment with penicillin G. On the day of his symptom onset, his daughter noted that he asked

repeatedly over the phone about an appointment time for a clinic visit. After the conversation, he asked his daughter what day of the week it was. Two days later, accompanied by his daughter, he went to the hospital in order to receive treatment for syphilis, but he did not know why he was there or where he was. Therefore, he was transferred to the emergency room for further evaluation.

On initial evaluation at the emergency room, his blood pressure was 103/63 mmHg and his heart rate was

Table 1. Neuropsychological-test scores

Neuropsychological test	Time interval after onset		Control subjects, mean (SD)
	4 days	2 months	
Attention Digit span (front/back)	5*/3	8/4	6.00 (1.73)/3.67 (1.32)
Language and related functions			
Spontaneous speech	NL	NL	
Comprehension	NL	NL	
Repetition	NL	NL	
K-BNT	42	53	47.56 (6.82)
Reading	NL	NL	
Writing	NL	NL	
Praxis	NL	NL	
Right-left orientation	NL	NL	
Calculation	NL	NL	
Body-part identification	NL	NL	
Visuospatial function			
Interlocking pentagons	NL	NL	
Copying Rey-Osterrieth Complex Figure	31*	32*	33.33 (1.12)
Memory			
Orientation (time/place)	4/5	5/5	5/5
Three-word recall	1	2	2
Rey AVLT (1st/2nd/3rd)	13* (3/5/5)	16 (4/6/7)	19.78 (3.80)
Delayed recall	0*	5	6.22 (1.86)
Recognition discriminability index 100	79	87.5	84.78 (6.40)
Rey-Osterrieth Complex Figure Immediate recall	2*	8.5	14.39 (6.63)
Delayed recall	3*	5.5*	16.06 (5.68)
Recognition discriminability index 100	62.5*	87.5	79.63 (8.45)
Frontal executive function			
Contrasting program	NL	NL	
Square and triangle	NL	NL	
Luria loop	NL	NL	
Word fluency	NL	NL	
Animal/supermarket	20/11	19/12	15 (2.00)/17.44 (6.98)
Stroop test			
Word reading	83	112	112
Color naming	74	75	89.78 (15.16)
MMSE	27	29	27.44 (1.24)

*; <16%ile, AB; abnormal, NL; normal, AVLT; Auditory Verbal Learning Test, K-BNT; Korean version of the Boston Naming Test, MMSE; Mini-Mental State Examination, SD; standard deviation

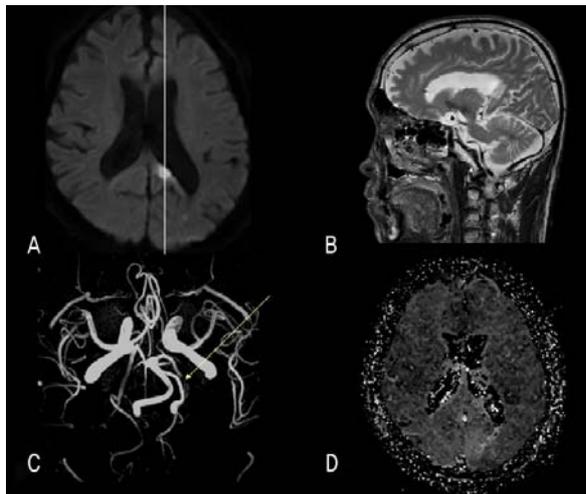


Figure 2. Axial diffusion-weighted magnetic resonance imaging (MRI) demonstrated an acute infarction in the left splenium and retrosplenium. A. White vertical line indicates the plane of the sagittal T2-weighted MRI imaging in panel B. B. sagittal T2-weighted MRI demonstrates that the infarction is restricted to the splenium and retrosplenium. C. Occlusion in the left posterior cerebral artery (PCA) is marked by an arrow in magnetic resonance angiography (MRA). Time-to-peak (TTP) perfusion map reveals a mild perfusion delay to the left occipital region.

75 beats/min and regular. He was alert and cooperative, but disoriented to time and place. He demonstrated alexia without agraphia, and retro- and antegrade amnesia. His score on the initial Mini-Mental State Examination (MMSE) was 22/30 (memory registration, 3/3; memory recall, 0/3; orientation to time, 4/5; orientation to place, 3/5; calculation and concentration, 4/5; and reading, 0/1). The visual fields of both eyes were constricted due to a previous panretinal photocoagulation procedure for diabetic retinopathy, but all the other cranial nerve examinations were normal. There were no motor, sensory, or cerebellar function abnormalities. A cerebrospinal fluid (CSF) analysis showed a normal WBC count (2 /ml) and mild elevated protein (81.5 mg/ml). In addition, the CSF/serum glucose ratio was 0.474 and the CSF VDRL test was negative.

The memory of the patient began to improve on the second day of hospitalization, but he could not remember his home address and phone number. He underwent a formal neuropsychological test four days

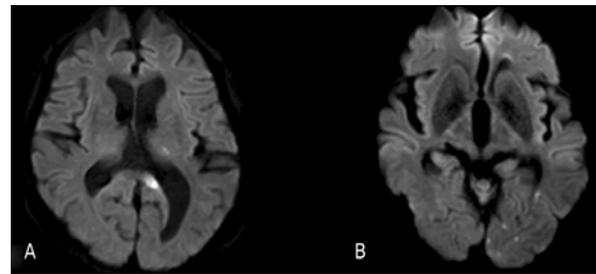


Figure 3. Other lesions were evident in the center of the thalamus (A), and small scattered lesions were evident in the area supplied by the left PCA (B).

after the onset of symptoms (Table 1), at which time the score on the MMSE was 27/30 (memory registration, 3/3; memory recall, 1/3; and calculation and concentration, 4/5). On the Rey Auditory Verbal Learning Test (AVLT), the score for free recall was 13 (3.75%ile of age-matched control subjects) and that for 20 min delayed recalls was 0 (0.04%ile). There were eight true positives of recognition and one false positive. The discrimination index of recognition was 7 (18.94%ile). The total score on the Rey copying test was 31/36. The scores for immediate and delayed recalls were 2/36 (3.07%ile) and 3/36 (1.07%ile), respectively. On recognition of the Rey copying test, there were four true positives and one false positive. The discrimination index was 3 (2.12%ile). Five days later, his memory appeared to have recovered considerably, but he still could not remember his home address.

Brain MRI (3.0-tesla device, PHILIPS) was performed two days after the onset of symptoms. Diffusion-weighted and sagittal T2-weighted MRI images (Fig. 2-A and 2-B) revealed a high-signal-intensity lesion in the left splenium and retrosplenium that appeared as a low-intensity signal on the apparent diffusion coefficient (ADC) map of b 1000 values. In addition, there was a point lesion in the center of the thalamus and small scattered lesions in the area supplied by the left posterior cerebral artery (PCA) (Fig. 3). On magnetic resonance angiography (MRA) images, there was an occlusion in the P2 portion of the left PCA (Fig. 2-C). The blood flow to the PCA region was delayed slightly based on the images of the time-to-peak (TTP) maps (Fig. 2-D).

After discharge, the patient demonstrated a marked

improvement in both his visual and verbal memory impairments and did not complain of any cognitive dysfunctions. Repeated neuropsychological tests - which demonstrated impairment at the time of admission - at two months after symptom onset demonstrated improvement on the Rey AVLT and a near-normal recall score on the Rey-Osterrieth Complex Figure test (Table 1).

DISCUSSION

Figure 2-A shows that the cerebral infarction was restricted to the left splenium with a little area of the retrosplenium. We considered that the amnesia of our patient was due to the retrosplenial lesion, because memory-related structures other than the retrosplenium were intact. Although the mild perfusion delay to the left PCA territory might have contributed to his amnesia, there were no significant changes in the relative cerebral blood volume and cerebral blood flow compared with the corresponding contralateral regions (Fig. 2-D). However, we could not exclude the possibility of other small infarctions being present in the areas related to the memory function because we did observe point lesions in the left central thalamus and left PCA territory.

Our patient showed visual and verbal amnesia irrespective of the unilateral left lesion. In contrast, there are reported cases of a single right retrosplenial lesion causing both visual and verbal memory deficits.⁵ There is strong evidence that the right medial temporal lobe is involved in navigation, and it now appears that input of the hippocampus and related structures receive from and convey to the right retrosplenial cortex has a similar spatial preference, while the left medial temporal and left retrosplenial cortices appear primarily concerned with more-general aspects of episodic memory.^{3,4,6} However, there are recent studies that can explain the presence of both visual and verbal amnesias in our case. Most functional neuroimaging studies have shown that navigation or orientation in a large-scale space activate the retrosplenial cortex, usually bilaterally.⁴ In addition, some functional MRI studies have shown that during episodic memory, bilateral retrosplenial areas were activated.⁷ Although there is a tendency for predomi-

nance, each retrosplenium must be involved in both visual and verbal memories.

Our patient showed considerable improvement in memory testing (Table 1). Although bilateral retrosplenial lesions tend to have a poor prognosis, retrosplenial amnesia generally recovers rapidly.^{4,8} Each retrosplenium receives major inputs from the contralateral retrosplenium, the orbital and dorsolateral prefrontal cortex, the anterior cingulate cortex, parahippocampal cortex, superior temporal sulcus, precuneus, claustrum, and the anterior and lateral thalamic nuclei.⁹ Therefore, a good prognosis may be partly due to functional substitution of the counterpart retrosplenium or other parts of the cerebrum. In addition, the prognosis may be influenced by the location of the retrosplenial lesion. Saito, et al.¹ reported a case of transient global amnesia (TGA) resulting from a retrosplenial infarction at a similar location to that in our case. However, in the case of Yasuda, et al.,⁵ which showed a poor prognosis, the lesion encroached the retrosplenium more posteriorly, although the size and location of the cerebral infarction were similar to those in our case. In contrast, Takahashi, et al.⁶ reported three cases of retrosplenial lesions that extended even to the inferior precuneus, all of which showed a good prognosis. Therefore, specific areas in the retrosplenium may critically affect the prognosis, and these could be elucidated by further functional neuroimaging, which would improve our understanding of the function of the retrosplenium.

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