A Case of Amnestic Syndrome Due to Right Thalamic Infarction

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A 66-year-old right-handed woman with amnestic syndrome due to right thalamic infarction, is described. Amnestic syndrome in this case was both anterograde verbal and non-verbal (visuospatial) memory impairment in the acute stage and only non-verbal impairment in the chronic stage. On the basis of these observations and previous reports, it was concluded that her memory impairment may be the result of cortical hypometabolism following thalamic infarction.

Key words: Memory impairment, Cortical hypometabolism

It is well known that bilateral thalamic lesions cause memory impairment (1) and some cases with amnestic syndrome due to unilateral thalamic lesion have been reported (2-9). Recently, Baron et al (10) suggested from their study by positron emission tomography (PET) that unilateral thalamic lesion causes ipsilateral cortical hypometabolism and that this cortical hypometabolism and neuropsychological deficit, including memory impairment, are related causally.

We describe a case of amnestic syndrome due to right thalamic infarction, with both anterograde verbal and non-verbal (for visuospatial materials) memory impairment in the acute stage and only non-verbal impairment in the chronic stage.

REPORT OF A CASE

A 66-year-old, completely right-handed woman was admitted to our clinic because of memory impairment on December 27, 1985. Three days before admission, her niece talked to her by telephone and noticed that she said incoherent things and that she could not recall the name of her acquaintance. When her husband came home in the evening, she complained of a mild headache and fatigue. Two days before admission, she could neither name nor use an electric washing machine or a dryer, so her husband brought her to our clinic for closed examination and further therapy. She had been receiving treatment for non-insulin dependent diabetes mellitus for eight years and for angina pectoris for two years. Her parents, brothers and sisters are all right handed.

Physical examination on admission showed her to be generally well. She had normal blood pressure (120/62 mmHg). She was alert but reticent. She answered our questions sluggishly. On neurological examination, neither motor, sensory disturbance, ataxia nor pathological reflexes were disclosed but deep tendon reflexes of the lower extremities were decreased. Grasping power of the right and left hand was 13 kg and 9 kg, respectively. Neuropsychological evaluations are shown in Table 1. Result of Wechsler Adult Intelligence Scale showed her to be at average intellectual ability and to have a discrepancy of 23 points between verbal and performance intelligence quotient. Scores of Standard Language Test of Aphasia (11) showed her to be normal except for mild impairment of word recall. Reading and writing were smooth and correct but the naming of some objects (a watch and an electric washing machine, etc.) was impossible. Digit span

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Table 1. Results of neuropsychological examinations.

1. Intelligence
   WAIS: VIQ 109, PIQ 86, FSIQ 101

2. Language
   SLTA: within normal range except for mild impairment of word recall
   Reading: smooth and correct
   Writing: smooth and correct
   Naming: partially impossible

3. Memory
   1) Immediate: Digit span was five digits forward and four back
   2) Recent:
      a) Learning ability: Recall of three pairs of unrelated words after three minutes was two pairs and one additional pair after receiving a hint
      b) Miyake’s test (Recall of ten pairs of related words) first trial: four pairs, second trial: eight pairs
   3) Visual:
      Benton visual retention test: three correct with twelve errors
      Copy of the figure: correct
      Immediate reproduction of the figures: incorrect
   3) Remote: correct

WAIS: Wechsler Adult Intelligence Scale, VIQ: verbal intelligence quotient, PIQ: performance intelligence quotient, FSIQ: full scale intelligence quotient, SLTA: Standard Language Test of Aphasia (9).

was five digits forward and four back, and recall of three pairs of unrelated words after three minutes was two pairs with one addition after receiving a hint. Recall of ten pairs of related words was four pairs at the first trial and eight pairs at the second trial by Miyake’s test (12). Score of the Benton visual retention test was three correct with twelve errors and an indication of low visual retentive ability. Copy of figures was correct but their immediate reproduction was incorrect. Recognition of proverbs and calculation were correct. Neither apraxia, agnosia nor disorientation for time and place were shown.

Laboratory data on admission were within normal ranges for peripheral blood and biochemical routine examination. However, fasting plasma glucose level was elevated (269 mg/dl) and glucosuria (4+) was found. Chest radiogram showed mild cardiomegaly (CTR 57%) and electrocardiogram demonstrated ST-segment depressions in II, III, aVF, V5 and V6 leads. Electroencephalogram showed that the background rhythm consisted of normal waves but high voltage slow waves occasionally occurred in the right hemisphere. Computed tomogram (CT) of the head a day after admission revealed a small well-defined low-density area [5 x 5mm (Fig. 1 left), 5 x 15mm (Fig. 1 right)] in the anteromedial aspect of the right thalamus, but
no abnormality was disclosed in other regions. Serial CT twelve days after admission showed a slight enhancement in the margin of the low density area. **Clinical course**

Two days after admission, naming objects and word recall had become smooth and correct. During the first month of admission, apathetic and sluggish behavior gradually improved. Serial CT thirty-eight days after admission similarly showed a low-density area in the anteromedial aspect of the right thalamus and a slight decrease in the size of the low density area. Two months after admission, the results of verbal memory test showed her to be normal (digit span was five forward and five back, and recall of three pairs of unrelated words was three). However, the score of the Benton visual retention test was four correct with eleven errors and an indication of the persistent low visual retentive ability. Seventy-four days after admission, she was discharged and followed as an outpatient. At eleven months after the attack, the score of Benton visual retention test (three correct with ten errors) showed persistent low visual retentive ability.

**DISCUSSION**

The primary feature in our patient was recent memory impairment without clouded consciousness and general loss of intellectual ability. On the basis of the diagnostic criteria from DSM III (13), we considered her case to be amnestic syndrome. It is characteristic of amnestic syndrome in the acute stage to involve both verbal and non-verbal memory impairment (for visuospatial materials). However, the chronic stage involved only non-verbal memory impairment.

CT of the head showed a well-defined low-density area with enhancement in the anteromedial aspect of the right thalamus. This finding was compatible with thalamic infarction, and involved the dorsomedial, intralaminar and ventrolateral nuclei.

It is well known that bilateral thalamic lesions cause memory impairment (1). Recently, some cases with amnestic syndrome due to unilateral thalamic lesion have been reported (2–9). In those cases, amnestic syndrome was considered to be due to damage in the anterior and/or the dorsomedial nuclei. However, Castaigne et al (14) reported that it is unlikely that lesioning of only the unilateral dorsomedial nucleus of the thalamus causes memory impairment. Regarding this problem, Baron et al (10) studied the energy metabolism of the cerebral cortex in patients with unilateral vascular thalamic lesions by PET. They proposed that unilateral thalamic lesions cause ipsilateral cortical hypometabolism and that this cortical hypometabolism and neuropsychological deficit, including memory impairment, were related causally.

Regarding the functional asymmetry of the hemisphere in memory impairment, it has been generally accepted that there is domination of the left hemisphere in verbal memory and the right hemisphere in non-verbal memory. Though PET was not performed in our patient, on the basis of these reports, her non-verbal memory impairment in the chronic stage may have been the result of ipsilateral cortical hypometabolism due to damage of the thalamocortical connection rather than from damage to only the right dorsomedial nucleus of the thalamus.

In amnestic syndrome due to right thalamic lesion, Speedie and Heilman (7) and Sato et al (8) have reported that patients have non-verbal memory impairment. These reports are similar to our patients in the chronic stage. However Akiguchi et al (4) and Tsoi et al (9) reported cases with both verbal and non-verbal memory impairment due to a right thalamic lesion. And also in our patient, both verbal and non-verbal memory impairment occurred only in the acute stage. Recently, Lenzi et al (15) reported a reduction in cerebral blood flow and metabolism in the hemisphere contralateral to cerebral infarction and that these changes were most pronounced within the first two weeks following infarction. Baron et al (10) reported thalamic lesion-induced hypometabolism of the contralateral cortex in addition to the ipsilateral cortical hypometabolism; Girault et al (16) reported the same observation in rats. Based on these reports, the memory impairment in the acute stage of the present case may have resulted from bilateral cortical hypometabolism following right thalamic infarction. In the memory impairment of our patient, symptomatic variability (verbal and non-verbal) may be responsible for the difference in the severity of cortical hypometabolism in right and left hemispheres during the course.

Hereafter, in order to clarify the pathophysio-
logical mechanism involved in the development of bilateral and ipsilateral cortical hypometabolism associated with the thalamic lesion, it is necessary to investigate the relationship of the thalamic nucleus (especially the anterior and the dorsomedial nuclei) and cortical hypometabolism (bilateral and ipsilateral), and the relationship between cortical hypometabolism and the neuropsychological deficit, including memory impairment.

REFERENCES