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Transcortical Sensory Aphasia Following the Unilateral Left Thalamic Infarction— A Case Report

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ABSTRACT

We reported a 60-year old right handed woman who had transcortical sensory aphasia caused by left thalamic cerebral infarction. She had a mild right hemiparesis involving the face, with hyperactive tendon reflexes. Tactile and pain sensations were decreased in the right side involving the face without visual field defect. Spontaneous speech was remarkably reduced, and she had a remarkable disturbance in object naming. Her auditory comprehension and writing were severely disturbed. This contrasted with full preservation of repetition of words and short sentences (5∼6 words). A CT scan 3 days later showed an infarct in the left ventral region of the thalamus. In the CBF (cerebral blood flow) study, ^{123}I-IMP SPECT (single photon emission CT) showed a low flow in the left temporal and occipital lobes. We concluded that there might be thalamic aphasia caused by an occlusion of the left posterior communicating artery directly affecting the area served by left posterior cerebral artery.

INTRODUCTION

Involvement of the thalamus in language function is a subject that has attracted great interest and debate following Fisher's report (1959) of a case of aphasia caused by lesions in the thalamus. Primary characteristics mentioned in cases of aphasia as a result of injury to the thalamus are transcortical aphasia marked by retention of repetition (Alexander and LoVerme 1980), and transitory aphasia which involves decreased awareness and loss of concentration (Mohr *et al.* 1975; Luria 1977). Cited causes mainly include thalamic bleeding and thalamic infarction with the latter appearing far more commonly (Cohen *et al.* 1980; Demeurisse *et al.* 1979; Mc Farling *et al.* 1982; Graff-Radford *et al.* 1984; Archer *et al.* 1981 Bogousslavsky *et al.* 1986a,b). Heretofore there has been considerable discussion of correlations between lesion localization and impairments subjectly, but very few reports have considered the role of regional cerebral circulation in aphasias caused by thalamic thrombosis. Therefore, we performed CT, angiography and ^{123}I-IMP SPECT (single photon emission CT) in a case of transcortical sensory aphasia caused by left thalamic cerebral infarction in order to determine the area and mechanism responsible for appearance of functional impairments.

CASE REPORT

S.Y. is a 60 year-old right-handed woman with 9 years of school education, and is a housewife. Her past history revealed that 10 years earlier she had undergone (encephalo-duro-arterio-synangiosis) EDAS for Moyamoya disease. She had experienced no neurological problems such as motor paralysis or speech impairment.

On 30 May 1990 in the course of conversation the patient suddenly began to exhibit speech disturbances. She was referred for diagnosis to the Neurological Surgery Unit, Wakayama Rosai Hospital. Initially, the patient was completely conscious with normal orientation. No anomalies were detected in eyesight or field of vision. Slight sensory paralysis in the right half of the face was accompanied by minor right hemiparesis, but there were no apparent sensory disturbance.

Neuropsychological Findings

The patient spoke little of her own volition, answering only when spoken to and not volunteering speech. When presented with picture-description problems, she exhibited literal paraphasia and neologism of this nature: *Kore soki* (Kore wa inu; this is a dog), *kore namiita yo* (kore wa yotto; this is a yacht). On word-finding tests (naming objects), she almost invariably replied "I don't know." On occasions where she did speak, however, nominal paraphasia was evident in substitutions such as *choucyo*

(butterfly) for *kingyo* (goldfish). There was no echolalia, and phonological structure was clear. Meanwhile, repetition was fluent up to short, five-word sentences. There was minor difficulty with visual recognition of words and with aural comprehension at the word level, but she was able to read aloud words in both *kanji* (Chinese characters) and in *kana* (syllabary). Agraphia was almost total for *kanji* but was 50% for single *kana*. This was shown by mistakes in *kana* vocabulary such as *tokei* (clock) becoming *tohai*, and *enpitsu* (pencil) being *genbitsu*, (Standard Language Test for Aphasia; Fig. 1). There was no oral apraxia, ideomotor apraxia or unilateral spatial agnosia.

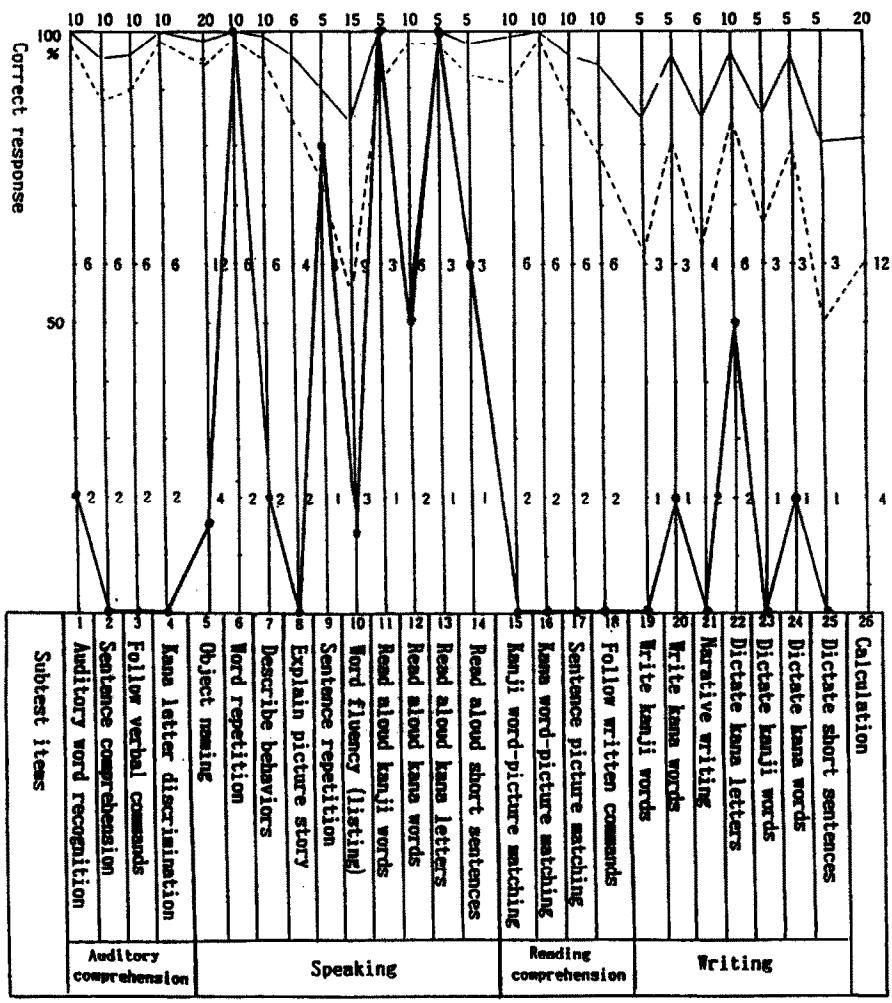

Figure 1. Standard Language Test for Aphasia at 3 Weeks after Onset.

Spontaneous speech was still limited 3 months later with the patient rarely initiating speech. However, there was some improvement in problems of naming and scene-description. Neologism and paraphasia had also disappeared. Repetition was good in sentences of 5–6 words. Although the patient could read short sentences aloud, visual recognition of individual letters even in vocabulary items remained below 50%. Auditory verbal understanding had improved somewhat, but was still poor at the word level. Agraphia had not improved.

Neuroradiological Findings

CT performed on the third day showed a low density area in the left ventral region of the thalamus (Fig. 2a). Cerebral angiogram taken on the eighth day revealed an occlusion of the left posterior communicating artery, so that peripheral circulation in the cerebral arteries was limited to minimal volume entering from the left intermediate cerebral artery (Fig. 2b). One month later ^{123}I-IMP SPECT was conducted and showed a decline in blood flow to the left hemisphere, particularly in the region extending from the left temporal lobe to parieto-occipital lobe (Fig. 3).

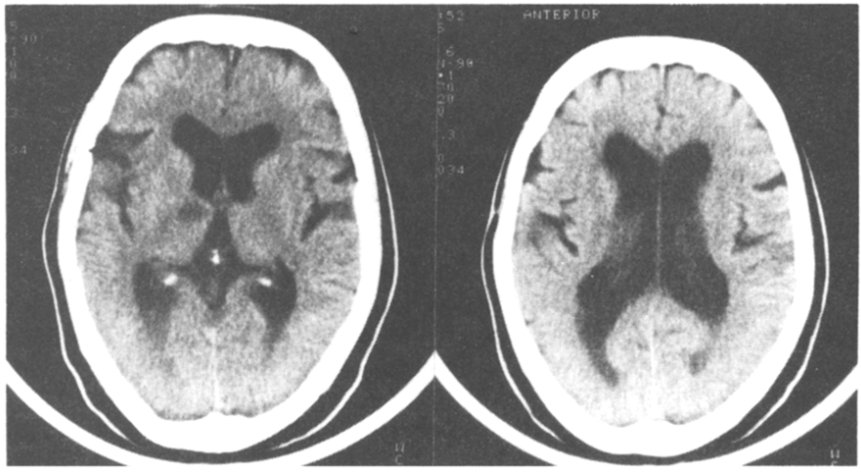

Figure 2a. CT performed on the third day showed a low density area in the left ventral region of the thalamus.

DISCUSSION

Because cases of aphasia caused by lesions in the thalamus usually exhibit signs of spatial or temporal disorientation, it has been assumed that a lower level of awareness and impaired attention are also involved (Mohr *et al.* 1975; Luria 1977). Moreover,

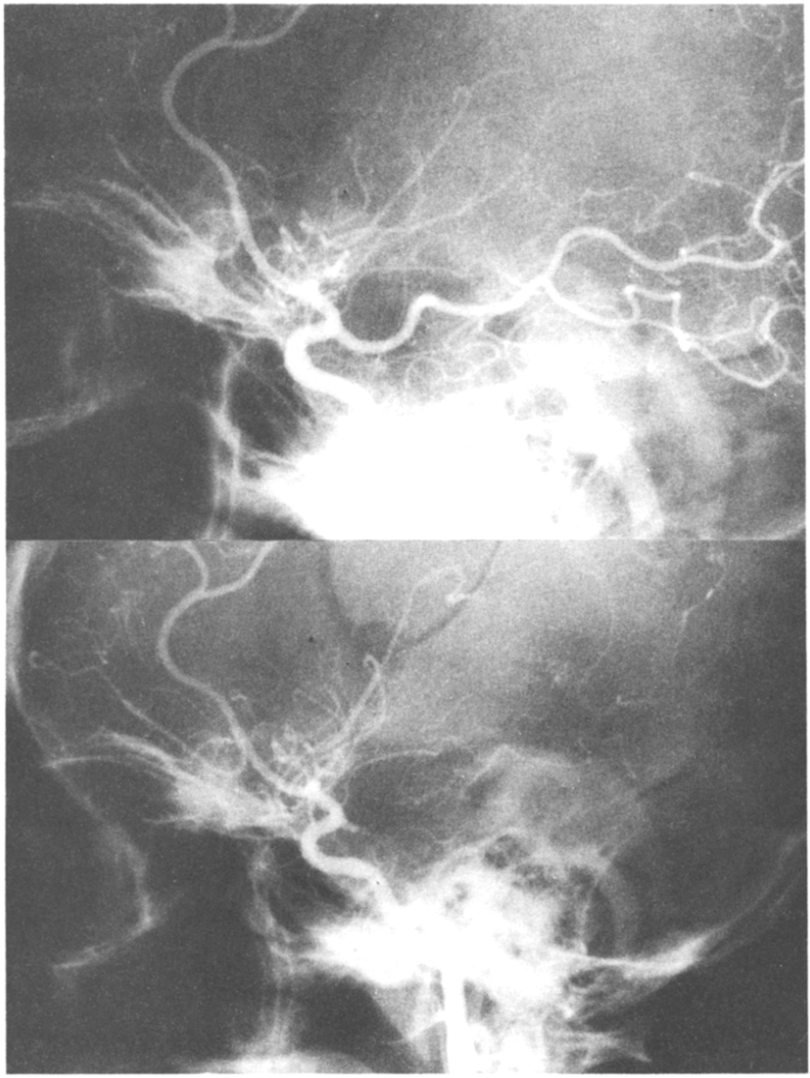

Figure 2b. Cerebral angiogram of 10 years earlier (upper) and the 8th day from onset (lower). Cerebral angiogram taken on the 8th day revealed an occlusion of the left posterior communicating artery, so that peripheral circulation in the cerebral arteries was limited to minimal volume entering from the left intermediate cerebral artery.

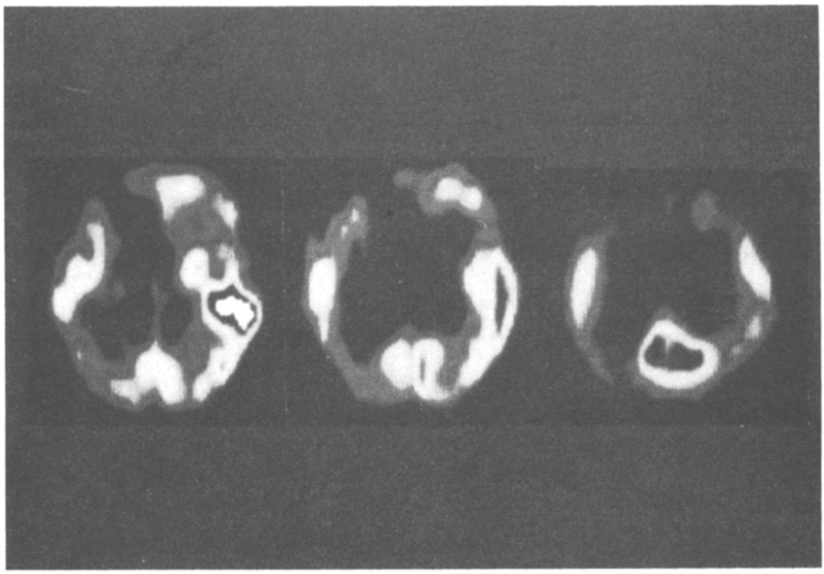

Figure 3. ^{123}I-IMP SPECT at 1 month showed a decline in blood flow to the left hemisphere, particularly in the region extending from the left temporal lobe to parieto-occipital lobe.

in many cases it is difficult to distinguish between impairments of mental faculties and those of awareness. In this particular case, however, consistent symptoms were observed for 3 months after onset of the condition. The patient was independent in all contexts of hospital life aside from language, so it is inadmissible to presuppose impairments in sensory awareness or mental faculties.

Some symptoms commonly cited as characteristic of speech impairment caused by disorders in the thalamus are loss of fluency, inappropriate volume, intonation or phonological structure, paraphasia, perseveration, slight difficulty with comprehension, and an array of problems related to written language such as dyslexia or agraphia (Mazaux *et al.* 1979). It has also been said that aphasias of the transcortical type in which repetition is retained are common (Alexander *et al.* 1980). In this particular case there was no echolalia or jargon, but the patient had severe difficulty in naming objects despite remarkable understanding of the spoken language. Also, despite the fact that extreme impairments were seen in her ability to recognize written words, she retained the ability to read aloud. Agraphia was also present. Finally, the fact that the patient had good repetition for short sentences further indicated that it was a transcortical aphasia.

Cerebral hemorrhage is the most frequently cited cause of aphasia due to lesion in the thalamus, while thrombosis is mentioned more rarely (Cohn *et al.* 1980;

Demeurisse *et al.* 1979; McFarling *et al.* 1982; Graff-Radford *et al.* 1984; Archer *et al.* 1981, Bogousslavsky *et al.* 1986a,b). In cases of hemorrhaging, aphasia is thought to be complicated by the effect of the subcortical fibers, including the capsula interna, or pressure placed on the cortex by masses such as hematoma. In cases of infarction, on the other hand, the lesion localization is clearly in the thalamus. Earlier reports have noted that cases of thalamic thrombosis which produce aphasia may be divided into occlusions of the posterior thalamosubthalamic paramedian artery, which supplies blood to the lateral medial corpus of the thalamus (Davous *et al.* 1984; Bogousslavsky *et al.* 1986b; Mori *et al.* 1986), and of the tuberothalamic artery, which supplies the anterior and central thalamic corpi (Cohen *et al.* 1980; Graff-Radford *et al.* 1984; Archer *et al.* 1981). However, recent discoveries indicated that damage to the thalamus may cause secondary impairment to cortical function. Araki *et al.* (1990) used PET (positron emission topography) in a case of infarction in the left anterior thalamic nucleus which was marked by agraphia and dyslexia, and reported finding secondary lesions in the left temporal and parietal lobe. In the case we experienced, the lesion as seen in CT appeared restricted to the anterior lateral section of thalamus. A cerebral angiogram confirmed that there was an occlusion of the left posterior communicating artery. From these facts we concluded that the infarction was in the area perfused by a branch of the tuberothalamic artery. However, only minimal circulation could be detected in the branch of the posterior cerebral artery that should have been supplied by the left posterior communicating artery, this small amount coming from the left medial cerebral artery. Examination by ^{123}I-IMP SPECT of the area extending from the left temporal lobe to the parietal and occipital lobes also confirmed a regional decline in cerebral circulation.

We believe the reason for this is that occlusion of the left posterior communicating artery directly affected the area served by left posterior cerebral artery. Kertez *et al.* (1982) have also reported that lesions in the left temporal–parietal–occipital junction were responsible in a case of transcortical sensory aphasia. Such an explanation would corroborate well with the type of aphasia observed in this case.

In other words, rather than viewing this case as an example of secondary impairment of cortical function resulting from the lesion in the thalamus, we should consider the symptoms as having a single cause: thalamic occlusion coexisting with damage caused by the shutdown of blood to the region perfused by the left posterior communicating artery.

Therefore, when studying cases of aphasia resulting from thalamic thrombosis, before trying to assign responsibility for various linguistic dysfunction damage to language centers in the thalamus or to thalamic way stations which attain the cortex, we should first examine the functional level of contiguous areas of perfusion which might be affected by the occluded artery.

NOTES

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