

5. Goyal M, Kucharczyk W, Keystone E. Granulomatous hypophysitis due to Wegener's granulomatosis. *AJNR Am J Neuroradiol* 2000;**21**:1466–9.
6. Buxton N, Robertson I. Lymphocytic and granulocytic hypophysitis: a single centre experience. *Br J Neurosurg* 2001;**15**:242–6.
7. Higuchi M, Arita N, Mori S, et al. Pituitary granuloma and chronic inflammation of hypophysis: clinical and immunohistochemical studies. *Acta Neurochir (Wien)* 1993;**121**:152–8.
8. Vasile M, Marsot-Dupuch K, Kujas M, et al. Idiopathic granulomatous hypophysitis: clinical and imaging features. *Neuroradiology* 1997;**39**:7–11.
9. Rickards AG, Harvey PW. Giant cell granulomas and other pituitary granulomata. *Q J Med* 1954;**18**:319–78.
10. Albin CH, MacGillivray MH, Fisher JE, et al. Triad of hypopituitarism, granulomatous hypophysitis, and ruptured Rathke's cleft cyst. *Neurosurgery* 1988;**22**:133–6.
11. de Bruin WI, van 't Verlaat JW, Graamans K, et al. Sellar granulomatous mass in a pregnant woman with active Crohn's disease. *Neth J Med* 1991;**39**:136–41.
12. Kristoff RA, van Roost D, Schramm J, et al. Primary granulomatous hypophysitis not responsive to pulsed high dose prednisolone therapy: case report. *J Neurol Neurosurg Psychiatry* 1998;**64**:693–4.

doi:10.1016/j.jocn.2005.11.046

Acquired stuttering in a patient with Wernicke's aphasia

Aiko Osawa ^a, Shinichiro Maeshima ^{b,*}, Takako Yoshimura ^c

^a Department of Rehabilitation Medicine, Kawasaki Medical School, Kawasaki Hospital, Okayama, Japan

^b Department of Sensory Science, Kawasaki University of Medical Welfare, 577 Matsushima, Kurashiki, Okayama 701-0193, Japan

^c Speech Therapy, Osaka College of Medical Welfare, Osaka, Japan

Received 24 January 2005; accepted 10 January 2006

Abstract

We report a patient with aphasia caused by cerebral infarction in the left temporal, parietal and occipital lobes that was complicated by acquired stuttering (AS), which is an unusual association. Our findings indicated that Wernicke's area might be involved in the development of AS.

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Keywords: Acquired stuttering; Wernicke's aphasia; Cerebrovascular accident

1. Introduction

Stuttering is defined as speech characterized by frequent lengthening or repeating of a single sound, syllabic sounds and words, or by frequent faltering and pauses that interrupt the rhythmic conversation flow.¹ Stuttering that is caused by cerebral vascular disease or head injury is known as acquired stuttering (AS). The supplementary motor area, basal ganglia or corpus callosum are thought to be involved in lesions causing AS. Although various AS lesions have been previously reported, the responsible lesion or mechanisms underlying AS have still not been clearly identified. In the present report, a patient with aphasia caused by cerebral infarction in the left temporal, parietal and occipital lobes that was complicated with AS is described.

2. Case report

The patient was a 51-year-old right-handed security guard with 12 years of school education. His parents, brothers and sisters were all right handed. He had a 10-year history of hypertension, but no prior history of neurological problems. He developed acute speech disturbance with headache. He was referred to our hospital for diagnosis two days later.

Initially, the patient was completely conscious with normal orientation. Homonymous hemianopsia was detected on testing visual fields. He had no paresis or cerebellar dysfunction. Deep tendon reflexes were normal and pathological reflexes were absent.

At a neuropsychological examination, he made no articulation errors and his spontaneous speech was fluent in the parts of the conversation in which he was not stuttering. However, he had clear problems with oral reading, repetition and object naming. In addition, he had severe

* Corresponding author. Tel.: +81 86 225 1111; fax: +81 86 232 8343.
E-mail address: maeshima@mw.kawasaki-m.ac.jp (S. Maeshima).

Table 1
Western aphasia battery scores in a patient with Wernicke's aphasia and acquired stuttering after cerebral infarction

	Admission	4 Weeks after onset
Spontaneous speech	13	13
Speech comprehension	5.15	7.1
Repetition	2.1	4.6
Naming	5.8	7.2
Reading	5.85	6.45
Writing	0.8	5.0
Praxis (right hand)	5.8	6.7
Praxis (left hand)	5.3	6.7
Composition	5.2	7.4
Aphasia quotient	52.1	63.8
Right	37.5	63.6
Left	38.0	63.6

problems with auditory and letter comprehension. Therefore, he was diagnosed as having Wernicke's aphasia. Western Aphasia Battery (WAB) scores are shown in Table 1. The patient's utterance speed was normal, and he did not speak quietly. Examples of his characteristic reiteration

of syllabic sounds and words are shown below (underlined):

Examiner: "Gokibun wa ikagadesuka?" [How are you? (greeting)].

Patient: "Ki, ki, ki, kibun, kibun wa ii ii iidesu" [I am fine].

Examiner: "Kore wa nandesuka?" [What is this? (explain a picture story)].

Patient: "I, ito, ito, ano, ito, ano, ano, i, ito ga agatte imasu." [A kite is being flown].

"Inu, inu, inu ga . . . i, inu, inu ga, i, i, ite, itemasu" [There is a dog].

Examiner: "Kore wa nandesuka?" [What is it? (naming)].

Patient: "Ki, ki, ki, kitte, kitte desu." [It is a stamp.]

"Fu, fo, fo, fukubu, chigauna, fu, fo, fohku desu." [It is a fork].

Examiner: "Watashi no mane wo site itte kudasai." [Please repeat after me. (repetition)].

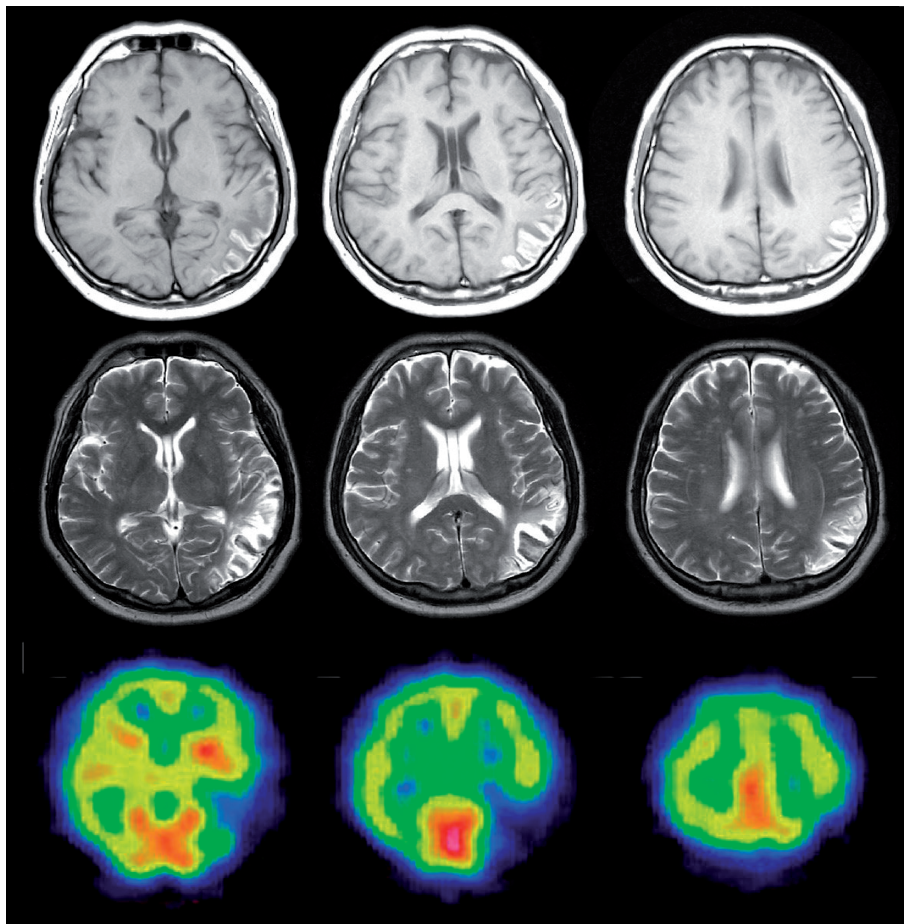


Fig. 1. Imaging findings in a patient with Wernicke's aphasia and acquired stuttering after cerebral infarction. Axial brain MRI revealed a low-intensity area surrounded by a high-intensity area in the region from the posterior part of the left temporal lobe to the parieto-occipital lobe on T1-weighted images (top row), and a high-intensity area on T2-weighted images (middle row). Single photon emission CT showed a low perfusion area corresponding to the infarction (bottom row).

“Ani wa mada modorimasen” [Brother has not returned yet].

Patient: “Ani, ani, aniwa, aniwa, aniwa, aniwa modorimasen.”

There were no differences in the rate of occurrence of “spontaneous speech”, “repetition” and “naming”, and AS appeared regardless of the length of words or sentences.

Buccofacial apraxia and ideomotor and ideational apraxia were observed in both hands. A sufficient evaluation could not be performed due to his aphasia, but he seemed not to have callosal disconnection since there was no difference in the rates of apraxia errors in the right and left hands. No unilateral spatial neglect or constructional apraxia was observed. According to the Revised Wechsler Adult Intelligence Scale (WAIS-R), performance intelligence quotient (PIQ) was 52, and verbal IQ could not be estimated due to his aphasia. His Raven’s coloured progressive matrices result was 23/36.

On neuroradiological examinations, CT scan on admission showed a low-density area in the region from the posterior part of the left temporal lobe to the parieto-occipital lobe, and moderate oedema was observed around this area. MRI performed on the day after onset revealed a low-intensity area surrounded by a high-intensity area in the same region as in the CT scan on T1-weighted images, and a high-intensity area on T2-weighted images. Cerebral angiography on the day after onset revealed no occluded vessels, and the vessels were thought to have recanalized after cerebral infarction. Single photon emission CT (SPECT) scan showed a low-perfusion area corresponding to the infarction (Fig. 1).

The patient received speech therapy to treat the aphasia and AS. Four weeks after admission, the aphasia gradually improved (Table 1) but the AS persisted.

3. Discussion

Our patient’s condition was characterized by development of Wernicke’s aphasia due to cerebral infarction, accompanied by AS. Acquired stuttering should be discriminated from other speech disorders involving constant reiteration of words or phrases, such as cluttering, palilalia and echolalia. In patients with cluttering, reiteration of words or phrases is similar, but the utterance speed is fast, compared with that in patients with AS. Patients with palilalia involuntarily repeat the same words or phrases. They tend to shorten paragraphs, and speak hurriedly or in a quiet voice, as with patients with cluttering. Unlike patients with echolalia, our patient did not repeat everything the person speaking to him said, but mainly repeated the initial sounds. In addition, his utterance speed and sound volume were normal. Therefore, he was thought to have AS.

AS due to cerebrovascular disorders is rare,² and only a few cases of AS accompanied by aphasia have been reported.^{3,4} This may be because it is difficult to discriminate between aphasia and AS as the non-fluency, including parenthetical expressions for filling the silence and reiteration for correcting utterances, observed in patients with aphasia is similar to that in patients with AS. There have been some reports of transcortical motor aphasia accompanied by AS.⁵ In these cases, AS can be recognized even though the patient has motor aphasia, as the patient has only mild apraxia of speech or distortion of words. On the other hand, in our patient with Wernicke’s aphasia, the reiteration of initial sounds was remarkable, although the spontaneous speech except for the AS was fluent. Therefore, it was relatively easy to make a diagnosis of AS. This unusual association should not be overlooked.

There have been some reports concerning the lesions of patients with AS, and the involvement of a closed-loop motor system (that is, brain cortex–thalamus–basal ganglia–brain cortex) has been suggested.⁶ In the cortex, the occurrence of AS has been reported after infarcts of the left frontal lobe, temporal lobe, parietal lobe and occipital lobe, and the right frontal lobe.^{2,3} A recent study using positron emission tomography (PET) suggested a phonological loop in the left frontal lobe and the anterior part of the temporal lobe based on decreased metabolism in these regions in AS patients.⁷ However, a patient with AS whose lesion was in the posterior part of the temporal lobe has also been reported,⁸ but in this case was not accompanied by aphasia. Our patient, who had a lesion in the posterior part of the temporal lobe, and in whom AS was accompanied by Wernicke’s aphasia, might provide important data for investigating lesions responsible for AS. Kariyasu et al.⁹ noted that non-fluent aphasia was linguistically and phonetically similar to AS. In our patient, difficulty in word recall or search and self-correction of the target sound was observed. Therefore, the underlying mechanism of AS in our patient was considered a linguistic disorder involving Wernicke’s area rather than a disorder of the pre-supplementary motor area that controls and inhibits the functions of the supplementary motor area.

References

1. WHO. *The ICD-10 Classification of Mental and Behavioural Disorders. Diagnostic Criteria for Research*. Geneva: WHO; 1993.
2. Helm NA, Butler RB, Benson FD. Acquired stuttering. *Neurology* 1987;**28**:1159–65.
3. Ciabarra AM, Elkind MS, Roberts JM, et al. Subcortical infarction resulting in acquired stuttering. *J Neurol Neurosurg Psychiatry* 2000;**69**:546–9.
4. Grant AC, Biousse V, Cook AA, et al. Stroke-associated stuttering. *Arch Neurol* 1999;**56**:624–47.
5. Freedman M, Alexander MP, Naeser MA. Anatomic basis of transcortical motor aphasia. *Neurology* 1984;**34**:409–17.

6. Alexander GE, Crutcher MD. Functional architecture of basal ganglia circuits: neural substrates of parallel processing. *TINS* 1990;**13**:266–71.
7. Fox PT, Ingham RJ, Ingham JC, et al. A PET study of the neural systems of stuttering. *Nature* 1996;**382**:158–62.
8. Turgut N, Utku U, Balci K. A case of acquired stuttering resulting from left parietal infarction. *Acta Neurol Scand* 2002;**105**:408–10.
9. Kariyasu M, Ohdaira Y, Shibamoto I. Speech fluency characteristics following damage-comparison among acquired stuttering, aphasia, and developmental stuttering. *Onsei Gengo* 1991;**32**:354–9.

doi:10.1016/j.jocn.2006.01.041