

## Clinical Study of the Responsible Lesion for Dysarthria in the Cerebellum

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### Abstract

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There have been few reports describing the lesion for cerebellar dysarthria. We compared MRI findings of 4 reported patients (including our previously reported patient) to that of our patient who showed ataxic speech and ataxic gait. The lesions of 4 patients involved lobulus quadrangularis and lobulus simplex, and the lesion of the present patient involved lobulus semilunaris superior and lobulus simplex. Since lobulus simplex and lobulus quadrangularis were involved in many patients, we speculated that the cerebellar dysarthria of the present patient was due to the damage of these areas in the upper cerebellum.

**Key words:** cerebellar dysarthria, cerebellar infarction, lobulus simplex, lobulus quadrangularis

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### Introduction

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Cerebellar dysarthria is characterized by ataxic speech, and is mainly caused by impairment of the upper cerebellar hemisphere in the distribution of the superior cerebellar artery (SCA) (1-4). However, there have been only a few reports about the responsible lesion for ataxic speech in the upper cerebellar hemisphere (1, 5-7). Previously, we reported a patient who showed isolated cerebellar dysarthria by the infarct of the upper cerebellar hemisphere (7) (Patient 2), and speculated that lobulus simplex was the responsible lesion of ataxic speech in the upper cerebellar hemisphere, based on the MRI findings of 3 reported patients and our patient (1, 5-7). We encountered another patient with cerebellar infarction showing ataxic speech, although there were slight cerebellar signs and symptoms (Patient 1). We speculated on the responsible lesion in the cerebellum for cerebellar dysarthria, based on MRI findings of 4 previously reported patients (1, 5-7) and a newly encountered patient.

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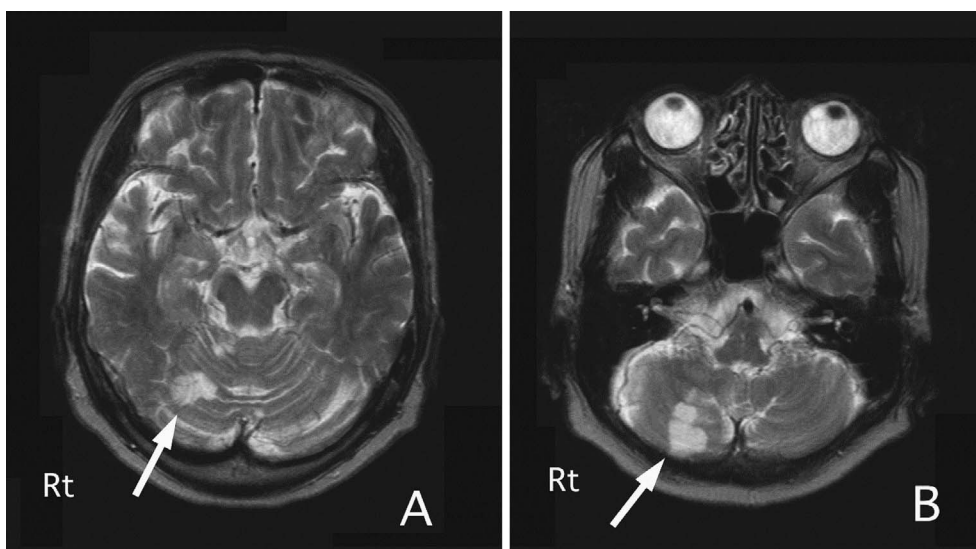
### Case Report

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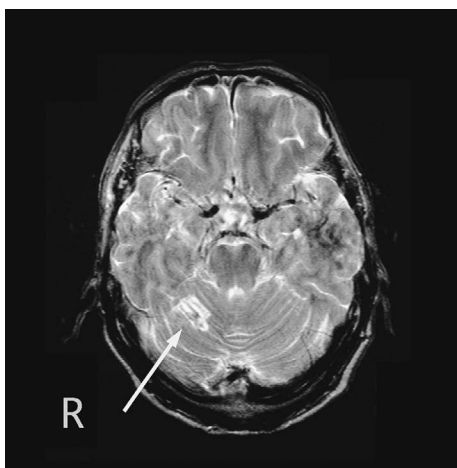
#### ***Patient 1 (Patient 1 was a newly encountered patient)***

A right-handed 73-year-old woman exhibited rotatory vertigo and dysarthria. Although vertigo improved the next day, the speech disorder persisted. On that same day, she was admitted to our hospital. On admission, neurological examinations showed that she was mentally alert, but her speech was mildly slow and slurred, and the rhythm of her speech was mildly poor. The natural gait was normal. Her trunk deviated to the right side occasionally during tandem gait, although she was able to stand on one foot. Otherwise, there was no sign of incoordination on finger-to-nose test and heel-knee-shin test on both sides. General and neurological examinations were normal, including the cranial nerves, muscle strength and tone, and sensation.

Examinations of blood cell count and chemistry, urine, electrocardiogram and cardioechogram were normal. Cranial MRI demonstrated a cerebellar infarct that was localized from the lobulus simplex to lobulus quadrangularis on the right side (Fig. 1A), and another infarct in the distribution of the right medial branch of the posterior inferior cerebellar



**Figure 1.** T2-weighted brain MRI (TR 3,600 / TE 96). An infarct was located from the lobulus quadrangularis to the lobulus simplex on the right side (A). Another infarct was located in the area of the distribution of the right medial branch of the posterior inferior cerebellar artery (B).



**Figure 2.** T2-weighted brain MRI (TR 3,600 / TE 96). An infarct was located in the lobulus quadrangularis and lobulus simplex of the upper cerebellar hemisphere on the right side, consistent with the distribution of the medial branch of the superior cerebellar artery.

artery (mPICA) (Fig. 1B). Dysarthria improved gradually, and disappeared after 2 weeks of hospitalization.

**Patient 2 (7) (Patient 2 was a previously reported patient)**

A right-handed 69-year-old man was admitted to the cardiosurgery service of our hospital due to exercise-induced pain in the right upper extremity. He was diagnosed as having arteriosclerotic obliteration (ASO) of the right upper extremity. Pain of the right upper extremity improved with intravenous administration of prostaglandin. Two months after the onset of ASO, he complained of pain in the right upper extremity again. He was admitted to our hospital and underwent emergent angiography of the right extremity, and was

discharged on the same day. However, speech disturbance developed that night. He was admitted to our neurology ward the following day because the speech disorder persisted. Computed tomography (CT) of the brain demonstrated an infarct in the right cerebellar hemisphere.

On admission, the rhythm of his speech was moderately poor. The initial words of his speech occasionally became explosive. The speed of his speech was moderately slow. Scanning speech was also present. Finger-to-nose test and heel-knee-shin test were normal on both sides. General and neurological examinations were normal, including the cranial nerves, muscle strength and tone, and sensation. There were no involuntary movements, orthostatic hypotension or vesicorectal dysfunction. Findings on blood cell count and chemistry, urine, electrocardiogram and cardioechogram were normal. Cranial MRI demonstrated an infarct that was localized from the lobulus simplex to the lobulus quadrangularis in the cerebellum on the right side (Fig. 2). Dysarthria improved gradually, and disappeared on day 5 of hospitalization.

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**Discussion**

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Cerebellar dysarthria, called ataxic speech, is characterized by slurred speech, explosive staccato vocalization and wavering modulation (8). The symptoms of dysarthria in our 2 patients (previously reported patient (7) and the present patient) were consistent in the characteristics of ataxic speech. Previous reports about the responsible lesions of cerebellar dysarthria have emphasized the paravermal zone of the rostral cerebellum, including the lobulus semilunaris superior and lobulus simplex, supplied by the medial superior cerebellar artery (mSCA) in the development of cerebellar dysarthria (9, 10). However, there have been only a few reports which discuss the responsible lesions for cere-

**Table 1. Previously Reported Patients with Cerebellar Infarction Demonstrating Isolated Dysarthria and Our 2 Patients**

Author	age (year)/sex	site of infarct	neurological finding
Kuwabara et al (1)	58/male	lobulus quadrangularis to lobulus simplex (right)	ataxic speech
Amarenco et al (5)	69/male	paravermal zone of the rostral cerebellar hemisphere (left) (lobulus semilunaris superior to lobulus simplex)	ataxic speech
Gironell et al (6)	78/male	paravermal zone of the rostral cerebellar hemisphere (right) (lobulus quadrangularis to lobulus simplex)	ataxic speech
Patient 1	73/female	lobulus quadrangularis to lobulus simplex (right) mPICA distribution area (right)	ataxic speech slightly ataxic gait
Patient 2 (7)	69/male	lobulus quadrangularis to lobulus simplex (right)	ataxic speech

PICA, posterior inferior cerebellar artery; mPICA, medial branch of the posterior inferior cerebellar artery.

bellar dysarthria in the upper cerebellar hemisphere (1, 5-7).

The symptoms of cerebellar dysarthria in our 2 patients were considered to have been caused by the infarct in the upper cerebellar hemisphere. Although Patient 2 demonstrated isolated ataxic speech, Patient 1 demonstrated slightly ataxic gait in addition to ataxic speech. The mPICA supplies the inferior vermis (nodulus, uvula, pyramis, tuber, and sometimes clivus), the internal parts of the lobulus semilunaris inferior, lobulus gracilis and tonsil (11). The prominent neurological characteristics of mPICA infarction are reported to cause both ataxic gait and lateropulsion to the ipsilateral direction without affecting coordination of the four extremities (11, 12). Thus, the ataxic gait in Patient 1 was considered to be caused by the infarct in the distribution of mPICA.

Four patients with isolated cerebellar dysarthria due to cerebellar infarction, including Patient 2 (7), have been reported previously (1, 5, 6). We compared the location of the infarcted lesions in these 4 reported patients with that of the infarct in our newly encountered patient (Patient 1) in order to determine the responsible lesion for cerebellar dysarthria (Table 1). The responsible lesions for cerebellar dysarthria in all 5 patients were all located in the upper cerebellum (1, 5-7). This finding was consistent with previous reports in which upper cerebellar lesions caused cerebellar dysarthria (1-4).

The infarct of the reported patient of Amarenco et al (5)

was located in the area from the lobulus semilunaris superior to the lobulus simplex. This lesion was consistent with the paravermal zone of the upper cerebellar hemisphere supplied by mSCA. The infarct reported by Gironell et al (6) also existed in the paravermal zone of the upper cerebellar hemisphere. This region of the infarct was anatomically localized in the area from the lobulus quadrangularis to the lobulus simplex. The infarct reported by Kuwabara et al (1) was located in the area from the lobulus quadrangularis to lobulus simplex, as was the infarct reported by Gironell et al (6). The lobulus simplex was damaged in the 5 patients discussed here, and the lobulus quadrangularis was also damaged in 4 of these patients (Table 1). We speculated that cerebellar dysarthria in the present patients occurred due to damage of the area from the lobulus simplex to lobulus quadrangularis, based on the localization of each infarct in these 5 patients (Table 1).

The association between speech function and cerebellar hemisphere dominance has been discussed previously (6, 9, 13). Ackermann et al (13) reported that the right cerebellar hemisphere connects with the lower precentral gyrus in the left frontal lobe, and contributes to language production, based on functional MRI findings (13, 14). In the 5 patients (1, 5-7), the right cerebellar hemisphere was more impaired (Table 1). This result was consistent with the report in which the right cerebellar hemisphere is dominant for speech production (13).

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