Case Report

Agraphia for Kana Predominance Induced by a Cerebral Infarction Involving the Left Middle Frontal Gyrus (Exner’s Area)

Kengo Maeda* and Nobuhiro Ogawa
Department of Neurology, National Hospital Organization Higashi-ohmi General Medical Center, Japan

*Corresponding author: Kengo Maeda, Department of Neurology, National Hospital Organization Higashi-ohmi General Medical Center, 255 Goishi, Higashi-ohmi, Shiga 527-8505, Japan, Tel: +81-748-22-3030; Fax: +81-748-23-3383; Email: maeda-kengo@shiga-hosp.jp

Received: July 20, 2014; Accepted: August 19, 2014; Published: August 20, 2014

Abstract

The foot of the left middle frontal gyrus has been considered as the site of graphemic motor image center since Sigmund Exner’s work in 1881. However, there have been only a few cases supporting the hypothesis. Recently, direct electrical stimulation and studies using functional MRI or PET have indicated the functional role of the area in handwriting. We herein report an ischemic stroke patient who showed apraxia of speech and agraphia. In her handwriting, kana (Japanese syllabograms) was predominantly disturbed compared with kanji (Japanese ideograms). Lesion analysis with MRI clearly showed the involvement of the middle part of the left precentral gyrus expanding to the caudal part of the middle frontal gyrus (rostral to the primary motor hand area). The former was considered to be responsible for her apraxia of speech, and the latter for her agraphia. In Japanese handwriting, “the writing center” of kana and that of kanji might be separate in the Exner’s area, as was in the case in left postero-inferior temporal lobe lesions.

Keywords: Frontal agraphia; Kana; Kanji; Exner’s area

Introduction

Agraphia is manifested as inability to write letters even though there is no paralysis or ataxia of the hand or aphasia. This symptom arises from lesions in the left postero-inferior temporal lobe or from left frontal lesions. As to frontal agraphia, Sigmund Exner reported the foot of the middle frontal gyrus as the “graphemic motor image center” in 1881 [1,2]. However, there have been only a few cases to support this hypothesis [3-5]. We herein report a single stroke case in which the patient showed apraxia of speech and agraphia for kana (Japanese syllabograms) predominant over kanji (Japanese ideograms).

Case Presentation

An 89-year-old right-handed woman presented with difficulty in speaking. She had a medical history of congestive heart failure, chronic kidney disease, and cerebral infarction at the right occipital lobe. When she woke up in the morning, her son noticed that he could not understand what she said. She was brought to our hospital that evening. Her blood pressure and body temperature were 147/97 mmHg and 36.9°C, respectively. She was alert and could walk by herself. There was no vascular bruit on her neck. On neurological examination, there was a slight weakness of her right mouth and right hand. She did not have dysphagia and could use tools with her right hand. There was no involuntary movement or sensory disturbance. Tendon reflexes were symmetric and normal. No pathological reflex was evoked. On neuropsychological examination, her auditory comprehension was normal. However, her spontaneous speech was rare, but not hesitant. She could not correctly pronounce words and attempted to correct her words several times. She could repeat single words but could not repeat sentences. Although there were incorrect pronunciations, she could read aloud sentences including kana and kanji. She could obey written simple commands. In spontaneous handwriting, she could write her own name in kanji (Figure 1-A). When she was asked to write anything she thought, she wrote using kana. The figure of each kana was correct, but the sentence was meaningless (Figure 1-B). Copying of sentences was almost normal for both kana and kanji (Figure 1-C). Dictation revealed marked paragraphia of kana (Figure 1-D).

A supraventricular contraction was demonstrated on the electrocardiogram. Brain magnetic resonance imaging (MRI) showed a fresh infarction in the left middle part of the precentral gyrus on diffusion-weighted images (Figure 2, upper panel). The infarction extended to the caudal part of the left middle frontal gyrus (just rostral to the primary motor hand area, Exner’s area) (Figure 2, lower panel). There was no stenosis in the intracranial major arteries. Ultrasound cardiogram showed no thrombus in the left atrium or valvular diseases of the heart. Echogram revealed some atherosclerotic changes at the left carotid artery.

Standard language test of aphasia (SLTA) performed on the seventh day revealed impaired writing. Although kanji writing had seemed to be preserved on the onset day, the ratio of the correct answers in kanji writing was almost the same as that in kana writing. There was also impairment of sentence repetition, explanation of a picture, and word fluency in speaking (Figure 3). Single photon emission computed tomography using 123I-N-isopropyl-P-iodoamphetamine showed hypoperfusion in the right temporoparietal region (the previous cerebral infarction) and the left frontal area corresponding to the fresh infarction. There was no widespread hypoperfusion in the left hemisphere (Figure 4).
She was treated with anticoagulants. In a few days, the weakness of her right mouth and right hand was improved. Although her handwriting was also improved after two weeks, speech difficulty remained.

**Discussion**

In the first examination, we wondered if she had motor aphasia. In general, patients with motor aphasia show hesitation or need effort in speaking. The patient presented above showed some attempts to correct her own pronunciation, but no hesitation or efforts. In addition, she could repeat single words. Although her free handwriting was meaningless and she could not express her thinking both in speaking or writing, figures of her letters were correct and especially the use of kanji letters was not disturbed as was in kana letters. SLTA revealed the low score in sentence comprehension, follow verbal commands, and sentence repetition. We considered that the low score in these points had been due to the impairment of auditory retention span rather than generalized aphasia. We diagnosed her as having apraxia of speech and agraphia. In her handwritings, kana was more deeply disturbed than kanji. The left middle part of the precentral gyrus was responsible for her apraxia of speech [6]. Agraphia was considered to be due to the lesion expanding to the left middle frontal gyrus, rostral to the primary motor hand area. Apraxia of speech and agraphia due to this lesion has been also reported in amyotrophic lateral sclerosis [7]. Her agraphia for kana was mainly phonological errors (correct...
kana was substituted by incorrect kana letter). Since the letter figures of her handwriting of kana were not impaired, visual images of kana were considered to be preserved. The site of frontal agraphia has been considered to be localized in the left middle frontal gyrus also known as “Exner’s area”. Recently, “writing center” or “graphic motor image center” was further examined using direct cortical electrical stimulation [8,9], functional MRI (fMRI) or PET [9,10]. Studies using fMRI or PET showed the specific activation in the anterior part of the superior parietal lobe and the posterior part of the superior and middle frontal gyrus during handwriting, suggesting that these areas correspond to a "writing center” [9,10].

Japanese people use two kinds of letters. One is kana, which express the sound only, and the other is kanji, which were Chinese letters originally and express both sound and meaning. In reading and writing of Japanese letters, sometimes dissociation between kana and kanji can be seen. In the cases of left posteroinferior temporal lobe lesions, writing of kanji can be preferentially disturbed [11-17]. Sakurai et al. reported two cases of frontal pure agraphia for kanji or kana [18]. In their study, a patient with agraphia for kana had a relatively large lesion (the posterior two thirds of the middle frontal gyrus). Compared with their case, in our case the lesion size was relatively large lesion (the posterior two thirds of the middle frontal gyrus). Compared with their case, in our case the lesion size was smaller and restricted to the posterior part of the middle frontal gyrus. In our case, the lesion was located in the left precentral gyrus.

References