

Nonfluent Aphasia and Cognitive Impairment Caused by Anterior Cerebral Artery Infarction

Xin-Yu Zhou,^{1,2} Li Chen^{1,3} & She-Qing Zhang¹

¹ Department of Neurology, Shanghai Hospital, Shanghai, China

² Department of Neurology, The First People's Hospital of Lianyungang City, Jiangsu, China

³ Department of Neurology, PLA 154 Hospital, Xinyang, Henan, China

Correspondence

She-Qing Zhang, Department of Neurology,
Changhai Hospital,
No. 168, Shanghai Rd.
Shanghai 200433, China.

Tel.: +86-21-8187 6719;

Fax: +86-21-5523 7129;

E-mail: sheqingzhang@126.com

Received 6 August 2013; revision 21 August

2013; accepted 21 August 2013

doi: 10.1111/cns.12180

The first two authors contributed equally to this work.

Anterior cerebral artery (ACA) supplies the anterior four-fifths of the medial surface of the cerebral hemisphere, including the medial surface of the frontal, parietal lobes, the frontal pole, the anterior four-fifths of the corpus callosum, and a narrow strip of the superior, lateral surface of the cerebral hemisphere. The clinical pictures of ACA infarction will depend on the location and size of the infarct and which is associated with the site of the occlusion and pattern of the Willis circle. Well-studied cases of ACA infarction are not numerous, and so, the ACA syndrome has not been elucidated completely. Kang and Kim reviewed the clinical, MRI, and angiographic findings of 100 patients with ACA infarctions. It was demonstrated that the most frequently occurring symptoms were motor dysfunction (91%), including severe motor dysfunction (43%), hypobulbia/apathy/indifference (43%), urinary incontinence (30%), grasp reflex (25%), sensory deficit (20%), and aphasia (18%). Among the eighteen patients with aphasia are eight global aphasia, eight transcortical mixed aphasia, and two motor aphasia [1].

Traditionally, it was believed that transcortical aphasia caused by ACA infarction occurred with occlusions of the Heubner branches of the left ACA or occurred with the infarction at the junction between two arterial territories, so-called watershed infarction, which might effectively isolate the intact motor and sensory language areas from the rest of the cortex of the same hemisphere [2].

However, recently, we encountered a patient with partial ACA infarction who presented with nonfluent aphasia. This aphasia is different from the above-mentioned features.

A 67-year-old right-handed man suddenly developed right-sided weakness. His relatives noticed that he became silent since

then. He was a well-educated man with a history of hypertension, diabetes mellitus, and hyperlipidemia. He used to take antihypertensive drugs and had his blood pressure controlled under a normal level. On examination, he was conscious, but he did not tell doctors actively about his onset course. When repeatedly prompted, he only uttered a few words, but not fluently, nor understandable easily. He was able to track objects and occasionally nodded his head to questions. His muscle strength of the right arm is slightly weaker than the left and that of the right leg is grade 4/5. The plantar reflex on the right is extensor. On mini-mental status test, his date orientation is poor and the place orientation is right. He was able to register three objects, and his recent memory is good. His calculation ability is poor, for example, he could not get the right result of 93 minus 7. His repeating ability is poor. The verbal and written commands can be executed correctly. His writing and copying abilities are nearly normal. Unenhanced MRI with diffusion weighted sequence of the brain showed acute cerebral infarction in the territory of anterior cerebral artery, including corpus callosum, cingulated gyrus, paracentral lobe, and superior frontal gyrus. (Figure 1) He was administered aspirin 100 mg, qd and clopidogrel 75 mg, qd.

During his hospitalization, he did not talk with others, and he lay on the bed all day. He ate food, but did not talk about its taste or quality. After eating, he would lie on the bed again, not moving.

The presentation of speech and language in our patient is quite peculiar. Firstly, it was different from akinetic mutism (AM). The main features of AM are mute, unresponsive, motionless, with no speech and gestures, while the patient is apparently alert. Since its first description by Cairns et al. in 1941, AM has been reported in

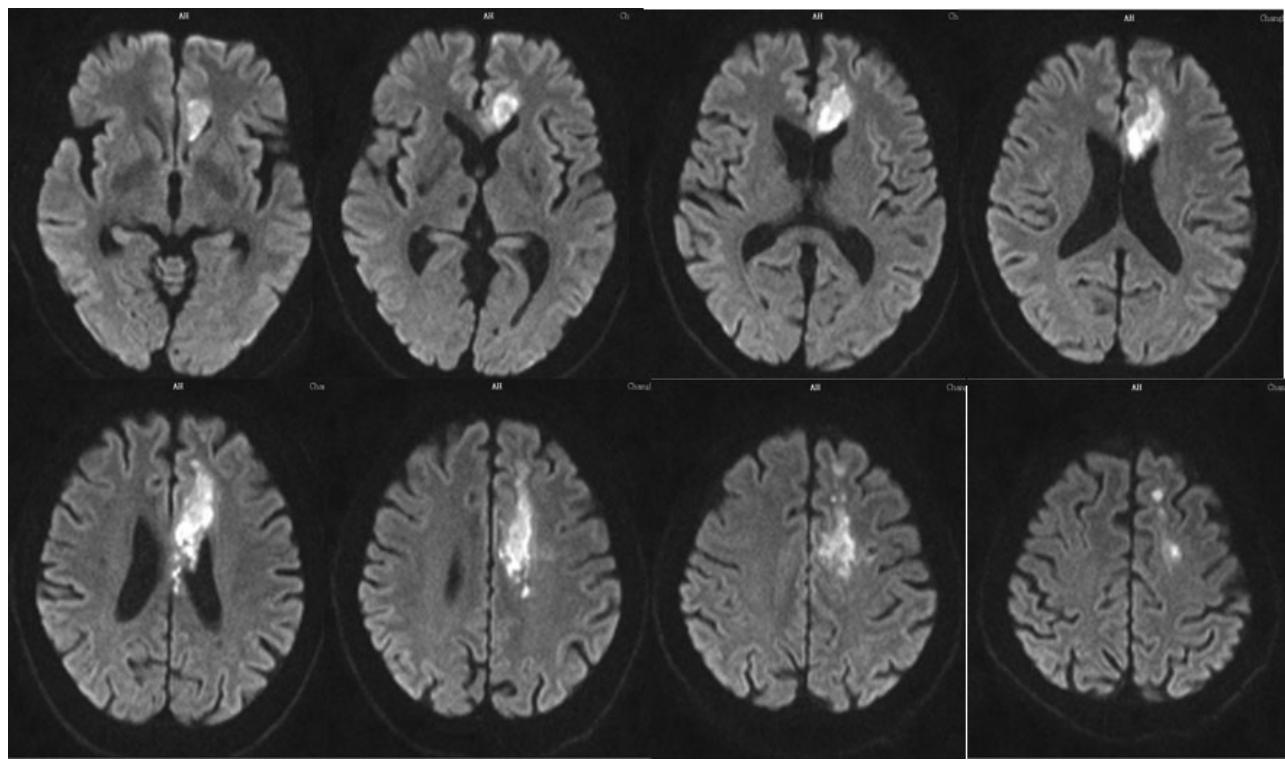


Figure 1 Diffusion-weighted MRI shows acute infarction in the territory of anterior cerebral artery, including cingulated gyrus, corpus callosum, paracentral lobe, and superior frontal gyrus.

a variety of clinical conditions, such as lesions to the anterior thalamus, fornix, and right internal watershed infarction, or infarction caused by unilateral anterior cerebral artery, etc. [3–6]. Although our patient always kept silent, he did spoke one or two words after repeatedly prompted. However, his responses are limited to single words, short phrases with unclear articulation. He

always lay on the bed, but he was able to walk if repeatedly ordered by the nurse or relatives.

Secondly, it was different from transcortical motor aphasia (TCMA). Transcortical motor aphasia is characterized by the decreased fluency of spontaneous speech, with relatively spared speech comprehension and repetition. The main lesions are asso-

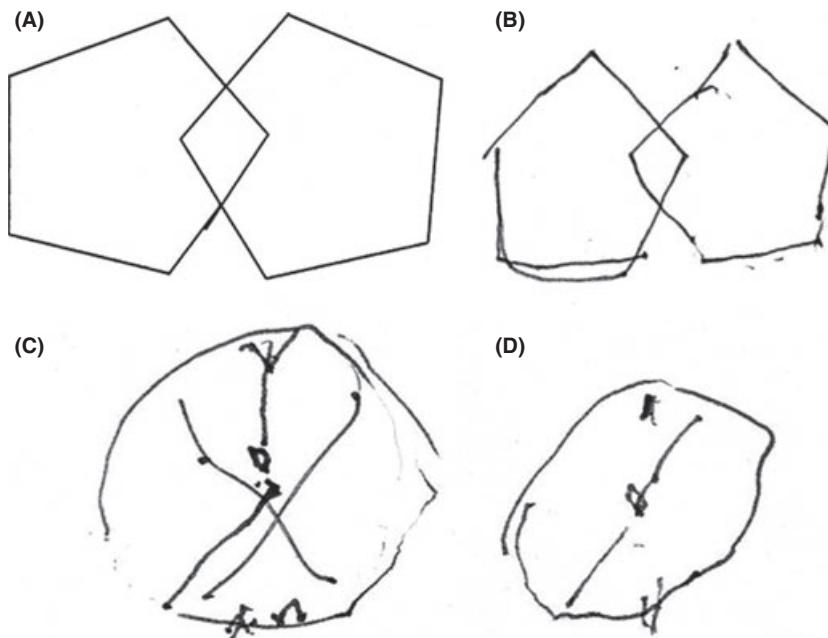


Figure 2 The patient's reproduction ability is normal: (A) is the original picture, and (B) is his drawing. However, his original drawing ability is poor: (C) and (D) is his drawing of a clock with its hands pointing to 8:20.

ciated with the left supplementary motor area (SMA) and the regions either anterior or superior to the dominant frontal operculum. Subcortical lesions associated with TCMA include the white matter deep to the frontal operculum, portions of the anterior limb of the internal capsule, the caudate, and the putamen [7]. Obviously, the lesions of our patient are not within the above-mentioned areas, and moreover, the patient does not possess the ability of well repetition.

Luria et al. have noticed the "dynamic aphasia" patients, who were able to improve their spontaneous output by provided with nonverbal cues, such as a piece of paper [8]. Patients with dynamic aphasia have the following features: they do not try to communicate in situations under which language is usually used; repetition is fast and well articulated; comprehension is scarcely compromised; reading is performed more easily than writing [9]. They suggested that these patients were unable to transform spontaneously a concept into a linear sequence of lexical items: failure of inner speech [8]. However, Luria's dynamic aphasia is just the prefrontal syndrome (apathy, adynamia, lack of initiative

and programming, attention disturbance, etc.) Although our patient has lesions in the left anterior cingulated gyrus, he could neither speak fluently nor did he preserve the well repetition ability.

Lack of initiative and spontaneity is the most common result of extensive and bilateral frontal lobes. It is characterized by the reduced psychomotor activity. The number of movements, spoken words, and thoughts per unit time diminished dramatically. With relatively mild forms of this disorder, patients show an idleness of thought, speech, and action. They are tolerant of most conditions in which they are placed [10]. This condition is called abulia. We have wondered if our patient developed this abulia. But, his visual-spatial cognition impairment (shown in Figure 2C,D) is certainly not associated with abulia.

In conclusion, ACA infarction may lead to a variety of clinical syndromes. Deep insight into the symptoms and signs caused by ACA infarction may help to elucidate the complex function of the forebrain, which has the name of "the organ of civilization."

References

1. Kang SY, Kim JS. Anterior cerebral artery infarction, stroke mechanism and clinical-imaging study in 100 patients. *Neurology* 2008; **70**:2386-2393.
2. Cecile CM, Constance FR, Christian D. Borderzone strokes and transcortical aphasia. *Curr Neurol Neurosci Rep* 2011; **11**:570-577.
3. Shetty AC, Morris J, O'mahony P. Akinetic mutism—not coma. *Age Ageing* 2009; **38**:350-351.
4. Oberndorfer S, Urbanits S, Lahrmann H, et al. Akinetic mutism caused by bilateral infiltration of the fornix in a patient with astrocytoma. *Eur J Neurol* 2002; **9**:311-313.
5. Anderson CA, Arciniegas DB, Huddle DC, et al. Akinetic mutism following unilateral anterior artery occlusion. *J Neuropsychiatr Clin Neurosci* 2003; **15**:385-386.
6. Lim YC, Ding CSL, Kong KH. Akinetic mutism after right internal watershed infarction. *Singapore Med J* 2007; **48**:466-468.
7. Gold M, Nadeau SE, Jacobs DH, et al. Adynamic aphasia: A transcortical motor aphasia with defective semantic strategy formation. *Brain Lang* 1997; **57**:374-393.
8. Luria AR, Hutton JT. A modern assessment of the basic forms of aphasia. *Brain Lang* 1977; **4**:129-151.
9. Ardila A, Lopez MV. Transcortical motor aphasia: One or two aphasias? *Brain Lang* 1984; **22**:350-353.
10. Ropper AH, Brown RH. *Neurological disorders caused by lesions in particular parts of the cerebrum in Adams and Victor's Principles of Neurology*. 8th edn, New York: McGraw-Hill, 2005:390-395.