Rehabilitation Practice and Science

Volume 24
Issue 1 *Taiwan Journal of Physical Medicine and Rehabilitation (TJPMR)*

Article 15

6-1-1996

Global Aphasia without Hemiplegia: Three casereport

Szu-Fu Chen

Tsan-Hon Liou

Zong-Jing Wang

I-Nan Lien

Global Aphasia without Hemiplegia - Three Case Report

Szu-Fu Chen, Tsan-Hon Liou[1], Zong-Jing Wang,
I-Nan Lien[2]

Global aphasia is the most severe form of language disorder. It is characterized by an extensive lesion involving the entire left perisylvian lesion and is usually accompanied by a dense right hemiplegia. However, it may be associated with only minimal and transient motor deficit. Most of the previous reports indicated that embolism was the most possible etiology of global aphasia without hemiplegia.

Here we present three patients who acutely developed global aphasia without hemiplegia as a result of stroke. These patients were examined in both acute and chronic stages. Computerized tomography and magnetic resonance imaging studies demonstrated that one patient had a single temporoparietal infarct, one had a single frontotemporopareital infarct and one had a temporoparietal hematoma. There was good recovery of verbal communication abilities. By three months after onset, two could be classified as having a residual sensory aphasia and another a nearly normal speech.

Our cases indicate that global aphasia without hemiplegia predicts a particularly good recovery of speech .

Keywords : global aphasia, hemiplegia, stroke

Submitted for publication, Feb 13, 1996. Resubmitted in revised form, Mar 20, 1996. Accepted in revised form, Mar 27, 1996.

Department of Physical Medicine and Rehabilitation, Taipei Municipal Yang-Ming Hospital
[1] Department of Physical Medicine and Rehabilitation, Peng-Hu Provincial Hospital
[2] Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital
Address reprint requests to: Szu-Fu Chen, Department of Physical Medicine and Rehabilitation, Taipei Municipal Yang-Ming, Hospital 105, Yu Sheng Street, Shih-Lin, Taipei, Taiwan, R.O.C. TEL:8353456-6773

Global aphasia is the most severe form of language impairment that occurs following focal brain damage. It is characterized by severe impairment of speech production, auditory comprehension, and repetition. Hemiplegia is considered to be an almost constant component of the clinical presentation. [1] Global aphasia may, however, be present with minimal and transient motor deficit. Global aphasia is classically caused by a large perisylvian region covering the posterior frontal gyrus (Brqca's area) as well as the posterior temporoparietal gyrus (Wernick's area). Global aphasia without hemiplegia may be caused either by a single large frontotemporoparietal lesion [2] or by 2 discrete lesions in the dominant hemisphere , one affecting the posterior and the other the anterior left language area. [3] It may even result from a single temporoparietal infarct [4] or prerolandic infarct in the left hemisphere[5]. Cerebral embolism was reported as the most possible etiology of the syndrome and it seems to indicate a better outcome of verbal communication abilities than classical global aphasia. [3,6] We present three patients who acutely developed global aphasia without hemiplegia as results of cerebral thrombosis and cerebral hemorrhage.

CASE REPORT

Case 1. A 52-year-old, right handed man who had past history of diabetes, hypertension and hyperlipidemia, suddenly begun to speak incoherently one day prior to admission. On examination, he was alert. When he attempted to speak, he said only "yes". He was unable to follow command, name objects, repeat simple words, read aloud, or write his name. Neurological examination showed a discrete right facial weakness and a minimal right pronator drift ,both of which resolved completely by the seventh day after onset. The muscle strength was otherwise normal. Tendon reflexes, plantar responses, and gait were also normal. Sensory examination was impossible to interpret.

Computerized tomography (CT) obtained 8 days after onset showed an area of decreased density in the left temporoparietal lobe with heterogenous enhancement (Fig. 1). The echocardiogram revealed left ventricular septal hypertrophy with normal contractility. Valvular motility was fair and there was no evidence of mural thrombus, valvular vegetation, or other source of cardiac embolism. B-mode and duplex Doppler ultrasonographic examnation of the carotid arteries revealed patent carotid arteries, with decreased flow over left anterior and posterior cerebral artery.

One week after onset, formal language testing using Boston Diagnostic Aphasia Examination (BDAE) revealed a typical pattern of global aphasia. His speech was severely noninfluent, and he showed profound deficits in most aspects of linguistic functions. He did produce

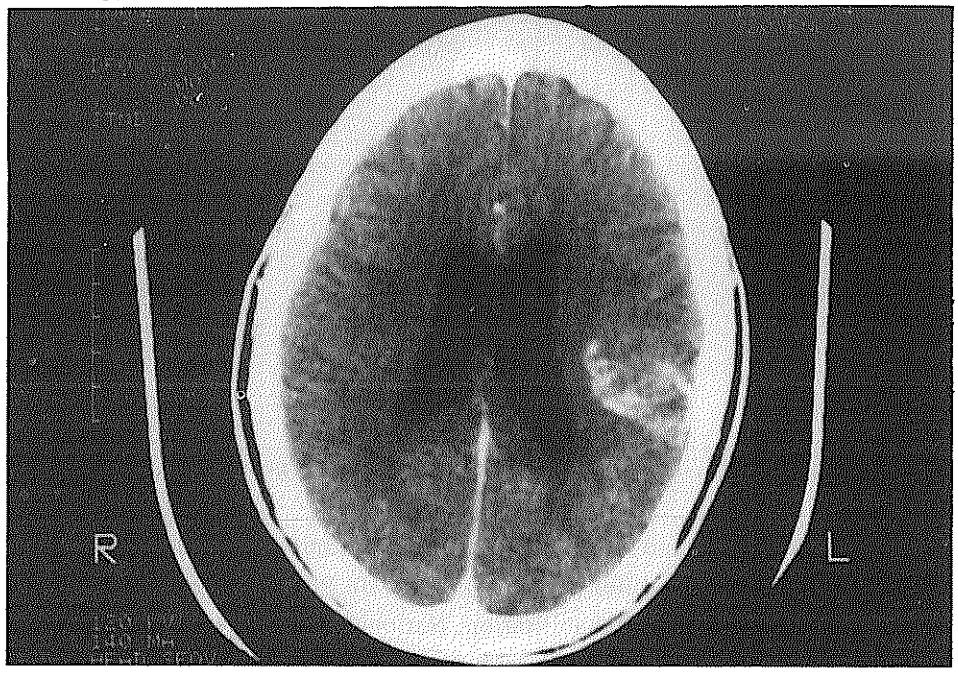

Fig 1: Contrast CT of patient 1 obtained eight days after onset: A single area of decreased density in the left temporoparietal lobe with heterogenous enhancement.

some short utterances of two or three words. Marked paraphasic errors were noted. Naming and repetition were not possible. He showed marked impairment of aural and reading comprehension, even for single word . There was no oral or ideomotor apraxia.

The patient was followed up at weekly interval. By the 14th hospital day, he was able to give his full name and some phrases consisting of several words, with a great deal of effort. Comprehension of conversational speech was restricted to very simple commands. He still could not name, repeat, read or write. Over the next few weeks, the patient continued to have more spontaneous speech production.

The language evaluation performed on the 12th week revealed good recovery of spontaneous speech. He had grammatical speech but with occasional pauses. Verbal paraphasia was still noted. He was unable to name in either the visual, auditory, or tactile modalities. Repetition was not possible. He could follow simple commands and show some preservation of simple words and short phrases; however, aural and reading comprehension of more complex material was severely defective. Language testing using BDAE revealed Wernick's aphasia. One year after stroke, he still presented a Wernick's aphasia by BDAE.

Case 2. A 60-year-old, right handed male was found at home with unintelligible speech. Past history was unremarkable except benign prostatic hypertrophy was noted at a physical check-up. On examination, he was awake and attentive, producing a few phrases repetitively; he was unable to follow commands, repeat, read, write or name objects. He had slight drift of the outstretched right arm, a right homonymous hemianopsia and mild right hyperreflexia. No weakness was found on manual muscle examination. There was no carotid bruit, heart murmur, gallop, or arrhythmia.

CT scan of brain on admission showed no focal lesion. By the 9th day, Magnetic resonance imaging (MRI) revealed a large contrast enhancing infarct in the left frontotemporoparietal lobe (Figure 2). There was mild mass effect on the left hemisphere as obliterated sulci, fissures and diminished size of left lateral ventricle. Besides, no definite missing or stenosis of intracranial vessels was detected in Magnetic resonance angiography (MRA) study. However, atherosclerotic change was noted at the bifurcation of carotid arteries. The echocardiogram revealed fair valvular mobility. The left ventricle had normal contractility and was not hypertrophic. There was no evidence of mural thrombus, valvular vegetation, or other source of cardiac embolism. B-mode and duplex Doppler ultrasonographic examination of the carotid arteries showed a modest plaque at the lower end of the internal carotid artery, with decreased blood flow over left middle and posterior cerebral artery.

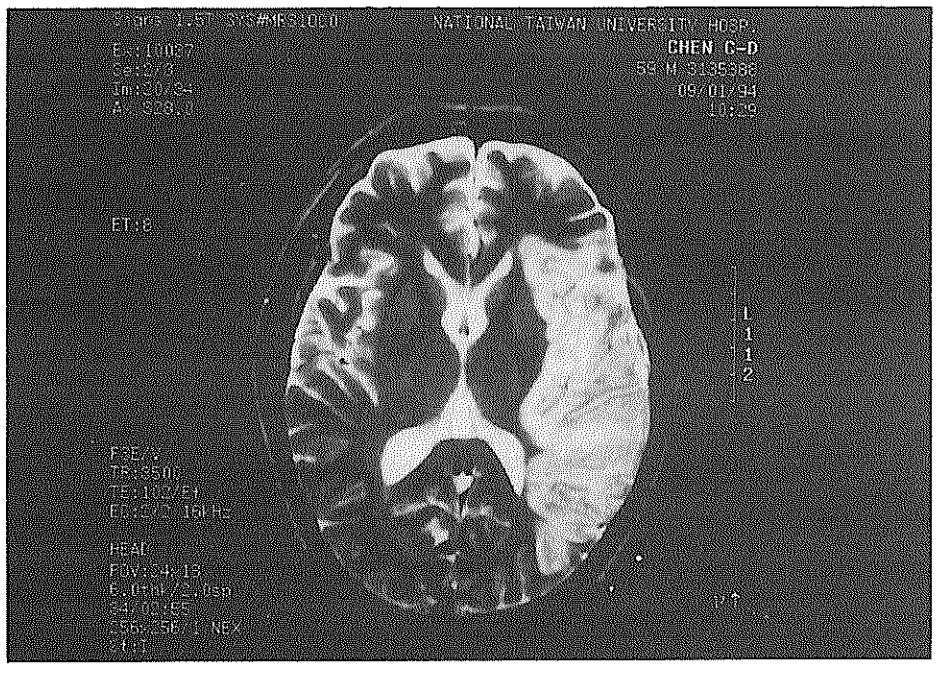

Fig 2: T2-weighted image of MRI of patient 2 nine days after onset: A single area of increased signal was found in the left fron to temporoparietal lobe.

One week after onset, formal language testing using BDAE revealed the pattern of global aphasia. Besides, he had severe alexia, agraphia and gestural apraxia of the right arm. He also showed constructional apraxia and impaired right-left discrimination. Visual perception was relatively intact. He improved slightly during the first 2 weeks in the hospital, the most notable recovery took place in the domain of oral expression of conversational speech. Follow-up aphasia evaluation 3 months after the stroke showed recovery of speech and language. He could speak fluently with mild hesitations. Speech content was filled with empty phrases and circumlotions, with a paucity of meaningful nouns and verbs. Paraphasic error of both literal and verbal type was also noted. Naming was deficient, often with bizarre paraphasic substitutions for the correct time. Auditory comprehension and repetition were also impaired. The result of BDAE revealed Wernick's aphasia. There was also very significant improvement in gesture and constructional apraxia. One year after onset, a repeated BDAE still showed Wernick's aphasia.

Case 3. The patient, a 61-year-old housewife, was a naturally right-handed woman. At the age of 54, hypertension was noted during a routine examination. On the day of admission, she suddenly developed right limbs weakness, stopped speaking and appeared unable to comprehend speech. On examination, she was slightly drowsy. She could follow only a few simple verbal commands and perseverated on these. She was unable to name, repeat, or follow written commands. There was slight right facial weakness, right hemiparesis (manual muscle test revealed "good" grade) and right hemianopsia. Cutaneous and deep sensation, and coordination were all intact. CT showed nonenhancing areas of increased density at the left temporoparietal subcortical area (3×2.5×4cm) which was considered as hypertensive intracranial hemorrhage . The standard language test using BDAE performed 7 days after the onset indicated global aphasia. Spontaneous speech was limited to only two words. These were uttered with extreme effort and frequent initial shuttering. The patint could not name or repeat. Comprehension was limited to very simple, one-step commands (e.g. "raise your hand"). Visual perception was relatively intact. There was no ideomotor, oral or constructional aphasia.

The patient improved significantly during the hospitalization course. Follow-up evaluation 1 month after onset showed excellent recovery of speech and language. She had grammatical speech with occasional pauses and word-finding difficulty, but formal linguistic assessment demonstrated normal or near-normal levels of performance in most areas tested. Neurological examnation performed at this time revealed mild right central facial weakness, slight positive pronator and

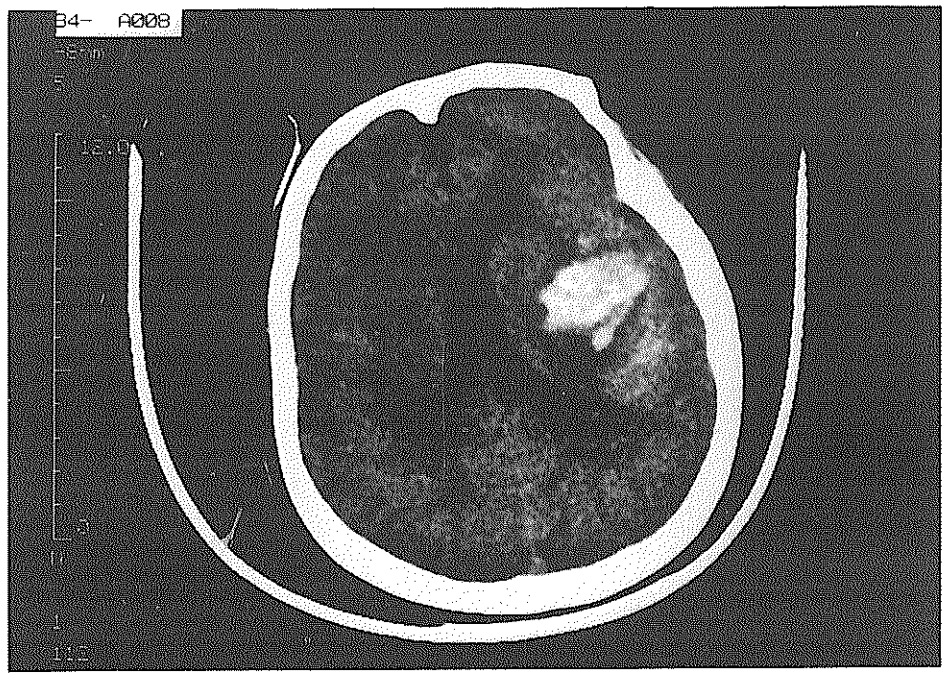

Fig 3: Noncontrast CT of patient 3 obtained at the day of onset: A hematoma in the left temporoparietal lobe.

Babinski sign on the right . There were no other signs of motor weakness. Brain CT performed 6 months after onset showed hypodense area at left temporoparietal area without contrast enhancement.

DISCUSSION

The percentage of aphasic stroke patient who have global aphasia range from 10 to 40% in various studies. [8-11] The figure obtained depends on the time of examination, since global aphasia may evolves into other aphasic patterns. In global aphasia, all aspects of language are so severely impaired. Speech is mute or nonfluent, comprehension is also severely deficient. In general, all six items of language evaluation (spontaneous speech, naming, auditory comprehension, repetition, reading comprehension, writing) are impaired. Patients with global aphasia typically have a dense right hemiplegia and often hemisensory loss and hemianopsia.

The lesions causing global aphasia were different in both extent and location. Commonly, global aphasia is associated with a large lesion in the left perisylvian and basal ganglion regions, usually results from an anterior trunk occlusion (middle cerebral or internal carotid artery) which also causes infarction of the precentral cortex. Therefore, almost all patients with global aphasia have an associated right hemiplegia. [12] The particularity of our observation is that our patients acutely developed a syndrome of global aphasia without the expected hemiplegia.

Van Horn and Hawes [3] described three patients who developed global aphasia without hemiplegia, all of whom had two separate ischemic lesions in the left hemisphere. One is over the anterior language cortex or language related subcortical areas, and the other over the posterior language cortex. Jeanine Delevel [5] presented 2 cases of global without hemiplegia whose brain CT revealed only a single lesion of the prerolandic area. He claimed that functional disconnection of posterior language area seemed responsible for this syndrome in such cases. Bogousslarsky [4] found that global aphasia without hemiplegia may even result from a single temporoparietal infarct in the left hemisphere. Other reports suggested that the syndrome can also be related to a single frontotemporoparietal lesion, which can sometimes be hemorrhagic. [2] Our cases were also consistent with above findings. Case 1 had CT evidence of a single temporoparietal infarct in the left hemisphere. The initial picture of global aphasia may be due to adjacent edema. However, the lesion spared the motor area in the frontal lobe. Owing to the posterior lesion and the location of motor function in the frontal lobe, the individual would demonstrate no obvious hemiplegia and gradually got recovery in spontaneous expression. MRI study in case 2 demonstrated a single large infarct of both Broca's and Wernick's area, partly sparing the posterior limb of the internal capsule and corona radiata, caused by middle cerebral artery occlusion without involvement of the lenticulostriate artery or the anterior choroidal artery. As for case 3, the syndrome was caused by a single temporoparietal hemorrhagic lesion. Since initial deficit may also be caused by edematous effect, the patient showed rapid recovery of the verbal communicative abilities .

The pathological events that lead to such a syndrome are of special interest. Van Horn and Hawes [3] described three patients who developed global aphasia without hemiplegia, all of whom had two discrete lesions in the dominant hemisphere. The author concluded that the presence of such double lesions suggested the possibility of an embolic stroke. Ferro [2] presented a case report, in which the syndrome was related to a single frontotemporoparietal lesion, and could be thrombotic origin. Legatt et al [6] also presented six cases with similar symptoms and signs. Four patients most likely had embolic infarcts, but the other two clearly had strokes of other etiology. One patient had a subarachnoid hemorrhage with hematoma near cortical language area. In the other one, aphasia was caused by a hematoma in the sylvian fissure with intraparenchymal extension and edema. The above mentioned causes are the commonest causes of the syndrome. However, it may also result from pathological events other than vascular origin. One patient has been reported in whom the syndrome was due to complex partial status epilepticus; the aphasia persisted for days reflecting both ictal and postictal effects. [13] Traumatic brain injury can also be responsible as in 5 of 280 patients with aphasia due to head injury in one study. [14] In summary, patients with global aphasia without hemiplegia frequently have cerebral emboli, but other

etiologies are also possible.

As for our patient, case 1 and 2 were shown to have had a single infarct. There was also no evidence of systemic emboli or an embolus visualized by angiography. Besides, the echocardiogram revealed no specific findings. So there was no distinct clinical evidence in the establishment of embolic cerebral infarction. Case 3 had a single temporoparietal hemorrhagic lesion.

A final point of importance is the issue of recovery. Previous research showed that global aphasia had poor prognosis. [11] A comparison between recoveries from different types of aphasia revealed that the patients with global aphasia invariably face the poorest outcome. [11] In Gail's study [15], 41% of patients presenting global aphasia at onset evolved to less severe aphasic syndrome. Jose [16] suggested the greatest change in the evolution of global aphasia occurred in the first 3 months. Nevertheless, these patients almost remained severely defective in communication skills 3 months after onset.

In contrast, our patients showed rapid recovery of their verbal communicative abilities. This was particularly evident in case 3, in whom had a nearly normal speech 2 months later. By 3 months after onset, case 1 and 2 both could be classified as having a residual sensory aphasia. In case 2 constructional and gesture apraxia also resolved. Trantel et al [7] suggested the reason for such a rapid recovery, in patients with global aphasia without hemiplegia is that there is only a circumscribed lesion involving a small portion of this area. Although the initial insults sufficiently produced a picture of global aphasia, such a situation could allow for compensatory activity of the intact cortex to develop.

In summary, patients with acute global aphasia without hemiplegia frequently have a embolic stroke, but other etiologies are possible. The syndrome often predicts an unusually good recovery of speech and language.

REFERENCES

1. Geschwind N: Current concepts: Aphasia. N Engl J Med 1971; **284**: 654-656.
2. Ferro JM : Global phasia without hemiparesis. Neurology 1983; **33**: 1106.
3. Van Horn G, Hawes A: Global aphasia without hemiparesis: A sign of embolic encephalopathy. Neurology 1982; **32**: 403-406.
4. Bogousslarsky J: Global aphasia without other lateralizing signs. Arch Neurol 1988; **45**: 143.
5. Deleval J, Leonard A, Marroudakis N, Rodesch G: Global aphasia without hemiparesis following prerolandic infarction. Neurology 1989; **39**: 1532-1574.
6. Legatt AD, Rubin MJ, Kaplan LR, et al: Global aphasia without hemiparesis. A sign of embolic encephalopathy. Neurology 1987; **37**: 201-205.
7. Trantel D, Biller J, Damasio H, Adams HP, Cornell SH: Global aphasia without hemiparesis. Arch Neurol 1987; **44**: 304-308.
8. Boller F: Strokes and behavior: disorders of higher cortical functions following cerebral disease. Stroke 1981; **12**: 532-4.
9. Brust JCM, Shafer SQ, Richer RW, Bruun B: Aphasia in acute stroke. Stroke 1976; **7**: 167-74.
10. Davis GA: A survey of adult aphasia. Englewood Cliffs, NJ: Prentice Hall,1983.
11. Kertesz A, McCable P: Recovery patterns and prognosis in aphasia. Brain 1977; **100**: 1-18.
12. Damasio AR: Aphasia. N Engl J Med 1992; **326**: 531-539.
13. Dinner DS, Leuders H, Lederman R, Gretter TE: Aphasic status epilepticus: a case report. Neurology 31.
14. Russel WR, Espir MLE: Traumatic aphasia: a study of aphasia in war wounds of the brain. London: Oxford University 1980; **30**: 1273-9.
15. Gail VP, Hollan AL: Evolution of aphasia in the first year post-onset. Cortex 1988; **24**: 411-423.
16. Jose MR: The influence of infarct location on recovery from global aphasia. Aphasiology 1992; **6(4)**: 415-43.

全失性失語症無合併半身偏癱-三病例報告

陳思甫　劉燦宏[1]　王榮俊　連倚南[2]

全失性失語症是失語症中最嚴重的一型，臨床上多肇因於左大腦病變；且病人通常都有合併右半身偏癱。然而，病人也可能並無運動功能障礙。過去的文獻報告指出，大部份全失性失語症而無合併半身癱瘓的病例多肇因於腦栓塞。

本研究提出三例全失性失語症而無合併半身癱瘓之個案；三例皆肇因於腦血管病變。電腦斷層及核磁共振檢查顯示其病變區域如下：一為左顳頂葉梗塞，一為左額顳頂葉梗塞，另一位則為左顳頂葉出血；三例皆非腦栓塞之病例。三位患者在語言功能都有顯著的進步。在發病三個月後，第一、二位病患轉型為接受型失語症，第三位病患在語言功能方面則近乎正常。

由本研究經驗，全失性失語症而無合併半身癱瘓的病例在語言功能方面預後極佳。

關鍵字：全失性失語症，偏癱，腦血管病變

台北市立陽明醫院復健科
[1] 省立澎湖醫院復健科
[2] 國立台灣大學附設醫院復健部
抽印本索取地址：陳思甫，台北市立陽明醫院，台北市士林區雨聲街105號