Global aphasia due to left thalamic hemorrhage

Ali Ozeren, Filiz Koc, Meltem Demirkiran, Abdurrahman Sönmezler, Mustafa Kibar*

*Departments of Neurology, and *Nuclear Medicine, Cukurova University School of Medicine, Adana, Turkey*

Global aphasia is an acquired language disorder characterized by severe impairments in all modalities of language. The specific sites of injury commonly include Wernike's and Broca's areas and result from large strokes - particularly those involving the internal carotid or middle cerebral arteries. Rarely, deep subcortical lesions may cause global aphasia. We present three cases with global aphasia due to a more rare cause: left thalamic hemorrhage. Their common feature was the large size of the hemorrhage and its extension to the third ventricule. HMPAO-SPECT in one of the cases revealed ipsilateral subcortical, frontotemporal cortical and right frontal cortical hypoperfusion. Left thalamic hemorrhage should be considered in the differential diagnosis of global aphasia.

Key words: Global aphasia, thalamic hemorrhage

This PDF is available for free download from a site hosted by Medknow Publications (www.medknow.com).

Introduction

Global aphasia is the most severe aphasia type characterized by loss of all aspects of speech and language and develops due to large "anterior-posterior" lesions that affect the Broca and Wernicke areas together. The lesion generally involves white matter, too.[1] The typical examples are occlusions in the proximal portion of the middle cerebral artery. But there are also cases with global aphasia due to lesions other than "anterior-posterior" lesions.[2] The ratio of global aphasia cases without anterior-posterior lesions is variable in different series, ranging between 42-47 %.[2,3] In our study, it is 43.5 %.[4] We report three right-handed patients with global aphasia due to left thalamic hemorrhage.

Case Reports

Case 1

A 60-year-old, right-handed woman was admitted to our department for right hemiparesis and absence of speech. Her past medical history was significant for hypertension of 12 years. During admission, her blood pressure was 200/110 mmHg and pulse was 100/bpm. Neurological examination revealed confusion, central facial paresis, hemiparesis and positive Babinski's sign on the right. Her confusion improved after a few days. Sensation, visual fields, praxis and visuospatial examinations could not be evaluated due to her language disorder. CT scan showed a large hemorrhage in the left thalamus extending to the anterolateral, posterolateral and medial areas of the thalamus and also anterior-superior and superior periventricular white matter (PVWM) slightly compressing the third ventricle [Figure 1].

First detailed language evaluation was done with the Gülhane aphasia test and the Turkish version of the Boston diagnostic aphasia examination (BDAE) one month after admission. Gülhane aphasia test for Turkish citizens, which is still in use for clinical evaluation of language disorders throughout Turkey, is modified from Mayo clinic aphasia test and BDAE.[5] She had no spontaneous speech. Other language modalities such as auditory and reading comprehension, naming, repetition and writing were severely impaired. The patient was diagnosed with global aphasia. Follow-up language evaluation at the third and sixth months showed only minimal improvement in her language abilities.

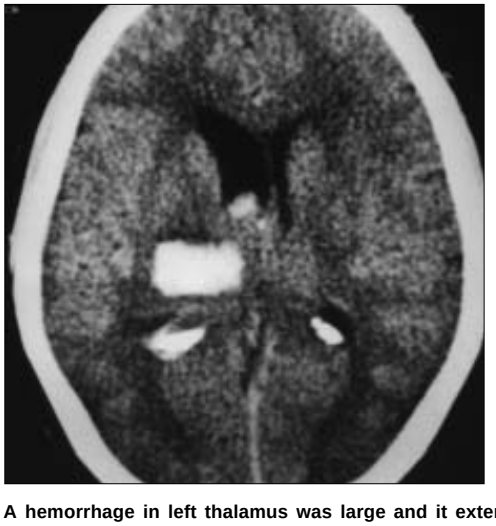


Figure 1: A hemorrhage in left thalamus was large and it extended to anterolateral, posterolateral and medial areas of thalamus and also anterior superior and PVWM and it slightly compressed the third ventricle

Filiz Koc
Department of Neurology, Cukurova University School of Medicine, Adana, Turkey. E-mail: zaferkoc@superonline.com

Case 2

A 65-year-old right-handed female was admitted to our department with sudden confusion, right hemiparesis and absence of speech. She had had hypertension for 10 years and congestive heart failure for three years. Her blood pressure was 200/110 mmHg, pulse was 90/bpm. Neurological examination showed confusion, right central facial paralysis, right flaccid hemiplegia. Babinski's sign was positive on the right. Her confusion resolved in several days and she became alert on the fourth day. On the 15th day of admission, she had severe articulation difficulties and severe agrammatism in speech output. Other language modalities were severely impaired. Her aphasia was classified as global aphasia. The initial CT scan showed left thalamic (large posterolateral) hemorrhage ruptured into the adjacent third ventricle resulting in obstructive hydrocephalus [Figure 2]. Follow-up examination revealed no change in language functions six months later.

Case 3

A 65-year-old right-handed female was admitted to our department with lethargy and right hemiparesis. Her past medical history was significant for a 20-year nephrolithiasis and a five-year hypertension. Her blood pressure was 190/110 mmHg and pulse was 120/bpm. Neurological examination showed lethargia, confusion, right central facial paralysis and moderate right hemiparesis. Babinski was positive bilaterally. First language evaluation was done at one month after admission. She had no spontaneous speech and was only capable of occasional undifferentiated grunts in conversation. She could only repeat meaningless phrases. Other language modalities were severely impaired. The patient was diagnosed as global aphasia. The CT scan showed left thalamic hemorrhage, spreading to medial, posterolateral and anterior superior PVWM, which ruptured into the third ventricle resulting in hydrocephalus [Figure 3]. HMPAO-SPECT of brain showed hypoperfusion in the left frontotemporal cortical, subcortical and right frontal cortical areas [Figure 4]. Control examination revealed minimal improvement in her communication five months later.

Discussion

For many years a debate has raged over the role the dominant thalamus plays in aphasia. In 1959, Penfield and Roberts suggested a connection center between the anterior and posterior language areas of the thalamus supported by electrical stimulation studies.[6] In addition, Fisher reported aphasia as one of the main signs of left thalamic hemorrhage.[7] This aphasia is characterized by fluent and sometimes hypophonic speech associated with paraphasia, moderate to mild disturbance in auditory and reading comprehension. Repetition and naming are well preserved. Kumar *et al* were the only authors reporting global aphasia due to left thalamic lesion.[8]

In the acute stages of stroke, it is hard to differentiate between akinetic mutism and global aphasia. Akinetic mutism occurs due to lesions of the dorsomedial and ventromedial thalamus.[9]

This PDF is available for free download from a site hosted by Medknow Publications (www.medknow.com).

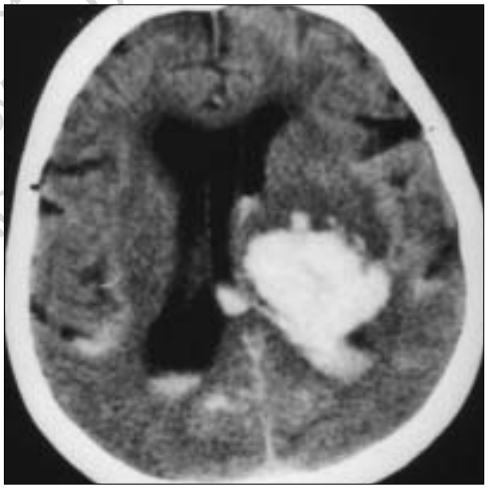


Figure 3: Spreading to medial, posterolateral and anterior superior PVWM hemorrhage that ruptured into third ventricle resulting in hydrocephalus

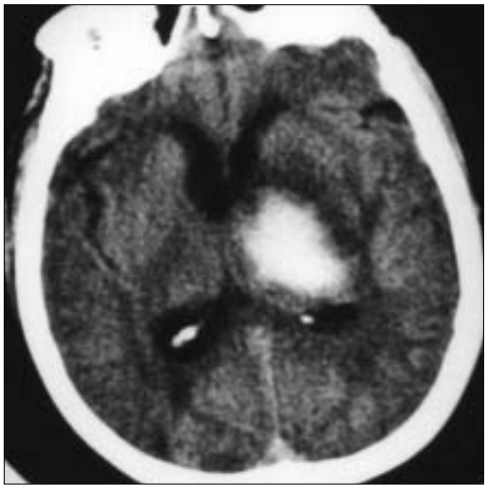


Figure 2: Large posterolateral hemorrhage ruptured into the adjacent third ventricle resulting in obstructive hydrocephalus

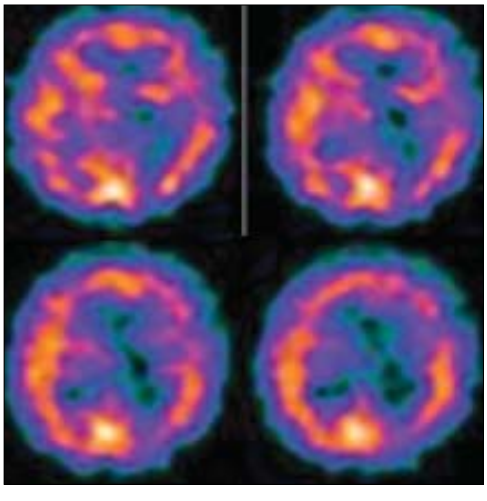


Figure 4: Hypoperfusion in left frontotemporal cortical, subcortical and right frontal cortical areas was seen at the HMPAO-SPECT of brain

Mutism generally develops in the acute period of thalamic hemorrhage and tends to show improvement. In all our cases, speech output resembled mutism, but no akinesia was observed and their aphasia lasted without much change in the follow-up period.

The most extensive theoretical model associated with the roles of subcortical structures in linguistics has been suggested by en Crosson.[10] According to this model, the striatum and cortico-striato-pallido-thalamo-cortical loop are as important as anterior and posterior linguistics structures. In this theory, striatal and thalamic areas are important in regulating rightness of speech regarding meaning.

In all our cases, hemorrhage was large extending posterolaterally. In all of them secondary subarachnoid hemorrhage was present and signs of hydrocephalus were seen in the acute phase, but it was more prominent in Case 3. The following explanations could be the possible causes of the aphasia due to thalamic lesions: a) Direct or indirect compression of perithalamic structures such as striatum, capsula interna, arcuate fasciculus and temporal lobe,[9] b) Perifocal edema (our patients had their brain CTs performed in acute phase when the effect of edema is the most), c) Remote functional depression (diaschisis) may be responsible for the development of aphasia.[11] The role of the subcortical structures in the development of the aphasia has been investigated by various functional imaging techniques. Compression of surrounding cortical structures or remote functional depression may be responsible for the development of aphasia.[12] This is crucial for the development of cortical hypoperfusion in the perisylvian language areas presumably due to undercutting of the white matter that would lead to the development of subcortical global aphasia.[13] Sequential imaging and autopsy studies suggest that in this type of aphasia, the cerebral cortex does not suffer structure damage, although there are some studies demonstrating morphological damage in the cortex of patients with deep lesion on CT scan.[14] We had the opportunity to perform cerebral SPECT in only one patient (Case 3), which showed hypoperfusion in the left frontotemporal cortical, subcortical and right frontal cortical areas in SPECT. This condition may be associated with compression of surrounding cortical structure and/or diaschisis and we can speculate that global aphasia could be due to the disconnection of the cortico-striato-pallido-thalamo-cortical loop in this patient. Any one of the causes mentioned above or any combination of them may be the reason of global aphasia associated with thalamic hemorrhage.

In conclusion, left thalamic lesions should be kept in mind in the differential diagnosis of global aphasia.

References

1. Albert ML, Helm-Estabrooks N. Diagnosis and treatment of aphasia. Part II. JAMA 1988;259:1205-10.
2. Vignolo LA, Boccardi E, Caverni L. Unexpected CT-scan findings in global aphasia. Cortex 1986;22:55-69.
3. Scarpa M, Colombo A, Sorgato P, De Renzi E. The incidence of aphasia and global aphasia in left brain brain-damaged patients. Cortex 1987;23:331-6.
4. Özeren A, Saryca Y, Efe R. Global aphasia due to left hemispheric stroke. Turk J Neurol 1995;1:73-6 (in Turkish)
5. Tanrýdað O. Aphasia. 2nd ed, GATA Press: Ankara; 1993 (in Turkish).
6. Penfield W, Roberts L. Speech and Brain Mechanisms. Princeton University Press: Princeton; 1959.
7. Fisher CM. The pathologic and clinical aspects of thalamic hemorrhage. Trans Am Neurol Assoc 1959;84:56-9.
8. Kumar R, Masih AK, Pardo J. Global aphasia due to thalamic hemorrhage: A case report and review of the literature. Arch Phys Med Rehabil 1996;77:1312-5.
9. Alexander MP, LoVerme SR Jr. Aphasia after left hemispheric intracerebral hemorrhage. Neurology 1980;30:1193-202.
10. Crosson B. Subcortical functions in language: A working model. Brain Lang 1985;25;257-92.
11. Olsen TS, Bruhn P, Oberg RG. Cortical hypoperfusion as a possible cause of "subcortical aphasia". Brain 1986;109:393-410.
12. Hillis AE, Barker PB, Wityk RJ, Aldrich EM, Restrepo L, Breese EL, *et al.* Variability in subcortical aphasia is due to variable sites of cortical hypoperfusion. Brain Lang 2004;89:524-30.
13. Okuda B, Tanaka H, Tachibana H, Kawabata K, Sugita M. Cerebral blood flow in subcortical global aphasia. Perisylvian cortical hypoperfusion as a crucial role. Stroke 1994;25:1495-9.
14. Godefroy O, Rousseaux M, Pruvo JP, Cabaret M, Leys D. Neurophysiological changes related to unilateral lenticulastriate infarcts. J Neurol Neurosurg Physchiatr 1994;57:480-5.

Accepted on 09-06-2006

Source of Support: Nil, **Conflict of Interest:** None declared.

This PDF is available for free download from a site hosted by Medknow Publications (www.medknow.com).