Case Reports

Mixed Transcortical Aphasia Without Anatomic Isolation of the Speech Area

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We report two patients with mixed transcortical aphasia following left frontal lobe infarctions. Although there was no evidence of anatomic isolation of the speech area on computed tomograms or magnetic resonance imaging scans, single-photon emission computed tomography in one case demonstrated diminished blood flow over the left parietal convexity suggestive of “functional isolation” of the posterior perisylvian language zone. (Stroke 1990;21:953–956)

Mixed transcortical aphasia is characterized by reduced or absent spontaneous speech, severely impaired language comprehension, and preserved repetition.1–3 Although the localization of lesions producing mixed transcortical aphasia is variable,4,5 the syndrome is seen most often with diffuse or multifocal pathologic changes that involve both anterior and posterior left hemisphere cortical association areas but spare the perisylvian language core.6 According to Goldstein,1 the critically placed lesions produce an “isolation of the speech area” posteriorly from the “ideational field” of the parietal association cortex and anteriorly from frontal areas important for the production of volitional speech. Since Broca’s area, Wernicke’s area, and the arcuate fasciculus are usually spared, repetition remains intact.

Acute mixed transcortical aphasia is uncommon.6 Recently, Bogousslavsky et al6 described four cases of acute mixed transcortical aphasia in the setting of internal carotid artery occlusion with computed tomographic (CT) evidence of simultaneous anterior precentral-central sulcus artery territory embolic infarction and posterior watershed infarction related to hemodynamic insufficiency. These cases support Goldstein’s contention by demonstrating both anterior and posterior anatomic isolation of the speech area.

We report two cases of mixed transcortical aphasia in which CT and magnetic resonance imaging (MRI) showed only frontal left hemisphere infarcts.
FIGURE 1. Computed tomograms and diagram showing left frontal lesion in Case 1.

FIGURE 2. Single-photon emission computed tomograms of Case 1 showing decreased blood flow in area corresponding to left frontal lesion seen on computed tomograms and magnetic resonance images (left, right, and bottom). There is also area of diminished blood flow over left parietal convexity (right and bottom).
died of a cardiac arrest 4 days later. Request for autopsy was denied.

Case 2

A 62-year-old right-handed man was admitted for the sudden onset of the inability to speak. On admission, spontaneous speech was absent. Although alert and attentive, he did not follow any commands and could not repeat or name. Mild right facial weakness and right upper extremity drift were present. Two days later he was noted to produce single meaningless repetitive utterances and occasional echolalic repetition. Propositional speech was still virtually absent, but he could now follow axial commands. By the fourth day he accurately repeated words, sentences, and meaningless phrases and completed open-ended sentences. Except for axial commands, auditory comprehension remained severely impaired even at the single-word level. He occasionally correctly named a few items on the Boston Naming Test, but would soon start to perseverate. Although he could read words and sentences aloud, he gave no indication that he understood what he read. Spontaneous writing was impossible, but he correctly spelled to dictation several words he did not understand.

CT 4 days after admission and MRI 3 weeks later (Figure 3) showed a large left frontal infarct in the territory of the middle cerebral artery. The lesion destroyed Broca’s area and involved the dorsolateral premotor and prefrontal cortex anterior and superior to it. The primary motor cortex was spared, and there were no retrorolandic lesions.

Although some improvement of language comprehension was observed, the patient’s condition remained essentially unchanged for the first 6 weeks.

At 3 months’ follow-up, spontaneous speech was still sparse but he was able to produce occasional meaningful utterances. A significant improvement of auditory and reading comprehension was noted.

Discussion

These two cases had very similar language profiles. After a brief initial period of global aphasia, the language deficit rapidly evolved into classical mixed transcortical aphasia. CT and MRI showed only left frontal lobe infarcts in both cases. The lesion destroyed Broca’s area and involved premotor and prefrontal regions anterior and superior to it. Partial or complete destruction of Broca’s area has been described in reports of transcortical aphasia, leading some investigators to believe that repetition was mediated by the intact right hemisphere in these cases. The abolition of repetition following the right hemispheric stroke in Case 1 is consistent with this view.

The lack of CT or MRI evidence for posterior anatomic isolation of the speech area is an unexpected finding in mixed transcortical aphasia. However, in Case 1 SPECT demonstrated an area of diminished blood flow over the left parietal convexity, suggesting that decreased functional activity of this cortical region results in language deficits typically seen with structural lesions that anatomically isolate the posterior perisylvian language zone. Based on these observations, we propose that “functional isolation” of the speech area may be a mechanism of mixed transcortical aphasia in cases without anatomic isolation.
Acknowledgments

The authors wish to thank Harold Atkins, MD, for his help with the SPECT study, Patricia A. Coleman, RMA, and Bari Searles for preparing the manuscript.

References


KEY WORDS • aphasia • cerebral infarction
Mixed transcortical aphasia without anatomic isolation of the speech area.
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Stroke. 1990;21:953-956
doi: 10.1161/01.STR.21.6.953

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/21/6/953

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