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Severe Aphasia following Infarction in the Territory of the Left Anterior Choroidal Artery

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We present a case of severe aphasia resulting from infarction in the territory of the left anterior choroidal artery (AChA). The AChA typically originates from the internal carotid artery, although it may also arise from the middle cerebral artery or posterior communicating artery [1]. Regions supplied by the AChA include the optic tract, posterior limb of the internal capsule, cerebral peduncle and choroid plexus. Additional areas often include portions of the paraventricular corona radiata, lateral geniculate body, medial temporal lobe, hippocampus, thalamus and basal ganglia [2]. Infarction of the AChA was initially associated with a syndrome of hemiparesis, hemianopia and hemianesthesia [1, 3], but more recently with some degree of neglect and language deficit with right and left hemisphere infarction, respectively [4–7]. The language deficits, when described, tended to be fairly mild.

Our patient is a 74-year-old, right-handed woman hospitalized with acute onset of aphasia and right hemiparesis. Brain MRI revealed acute infarction in the area of the left AChA, including the posterior limb of the left internal capsule and thalamus, as seen on diffusion-weighted imaging in figure 1. The patient was referred for functional neuroimaging but, unfortunately, was considered too agitated to participate. She had no neurological deficits prior to hospitalization. After transfer to an inpatient rehabilitation program, follow-up neurological examination revealed right hemianopia with adequate attention to the right, and spontaneous crossing of midline, right facial asymmetry at rest, 2/5 in right-upper and right-lower extremity strength, 5/5 in left-upper and left-lower extremity strength, and sensory exam was intact to light touch, temperature, and vibration throughout. Neuropsychological examination revealed possible signs of right neglect but this was difficult to verify. Otherwise fully alert, she made no spontaneous verbalizations, and responses to prompts were delayed and sparse. Because her communication skills were so impaired, language was evaluated with the Mississippi Aphasia Screening Test. She was able to name 2/5 objects and perform 3/5 automatic speech tasks (expressive subscale 10/50). She was accurate for 6/10 yes/no questions and followed 2/5 auditory commands (receptive subscale 16/50). Her total Mississippi Aphasia Screening Test score was 26/100. Repetition was impaired with



Fig. 1. A 74-year-old, right-handed woman hospitalized with acute onset of aphasia and right hemiparesis. Brain MRI revealed acute infarction in the area of the left AChA, including the posterior limb of the left internal capsule and thalamus.

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Accessible online at: www.karger.com/ced verbal perseverations. She was diagnosed with severe, mixed nonfluent aphasia and made no improvement in the following month.

The degree of aphasia following the stroke in the left AChA territory significantly exceeds previous reports of language disturbance. The mechanism for her aphasia (and neglect) was likely a disconnection between the thalamus and the cortex, as has been suggested by previous reports [4, 5, 7, 8]. Although this patient's presentation does not fit into the most common classifications of aphasia, it is consistent with a recent survey of stroke aphasia that found a high rate of exceptions to the classic clinical-topographic aphasia rules [9]. This case suggests that aphasia following left AChA infarction can be more severe than previously described.

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