Perfusion Deficits and Association with Clinical Outcome in Patients with Anterior Choroidal Artery Stroke

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Background and Purpose: Anterior choroidal artery (AChA) strokes have a varied pattern of tissue injury, prognosis, and clinical outcome. It is unclear whether perfusion deficit in AChA stroke is associated with the clinical outcome. This study aims to determine the frequency of perfusion abnormalities in AChA stroke and association with clinical outcome. *Methods:* The study cohort was derived from ischemic stroke patients admitted to 2 stroke centers between July 2001 and July 2014. All patients received an acute magnetic resonance imaging (MRI) scan. Patients with ischemic stroke restricted to the AChA territory were included in the study. Lesion size was measured as the largest diameter on diffusion-weighted imaging (DWI) or apparent diffusion coefficient and divided into 2 groups (<20 mm or ≥20 mm). Group comparisons were performed among patients with and without perfusion abnormalities and based on diffusion diameter. Favorable clinical outcome was defined as discharge to home. *Results:* A total of 120 patients were included in the study. Perfusion deficits were identified in 67% of patients. The admission National Institutes of Health Stroke Scale (NIHSS) was higher in patients with perfusion abnormalities ($P = .027$). Diameter lesion size on DWI was larger among patients with a perfusion deficit median [interquartile range], 1.63 [1.3-2.0], as compared with those without, 1.18 [1.0-1.7], $P < .0001$. Patients with a perfusion deficit were less likely to be discharged to home than those without (36% versus 60%, $P = .013$). *Conclusions:* Two thirds of patients with an AChA stroke have a perfusion deficit on MRI, higher admission NIHSS, and larger DWI lesion size at presentation.
Key Words: Stroke—magnetic resonance imaging—tissue plasminogen activator—anterior choroidal artery infarction.

Introduction

Anterior choroidal artery (AChA) typically arises from the posterior wall of the internal carotid artery distal to the origin of the posterior communicating artery and proximal to the intracranial carotid bifurcation.[1] The AChA has perforating branches that supply several anatomic regions including the internal capsule, posterior part of putamen, medial part of globus pallidum, posterior corona radiata, optic radiations, optic tract, crus cerebri, and medial temporal lobe.[2] The pattern of tissue injury and clinical syndromes of AChA strokes vary widely.[3]

The most common clinical presentation is motor hemiparesis and lacunar-type sensorimotor syndrome.[4] Higher cortical symptoms can result from AChA lesion. Ischemic

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Received December 1, 2016; accepted April 2, 2017.

This research was supported by the Intramural Research Program of the NIH, National Institute of Neurological Disorders and Stroke.

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1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2017.04.001

strokes of the right AChA territory may cause contralateral spatial hemineglect and constructional apraxia; those on the dominant hemisphere may result in a speech and language disorder. Bilateral AChA stroke may cause pseudobulbar mutism, bilateral facial weakness, and changes in affect.[5-8] Bilateral involvement of the lateral geniculate bodies may cause distinctive visual field abnormality resembling hourglass-shaped defects.[9]

The outcome after AChA stroke has been associated with severity of the stroke at presentation including higher admission (National Institutes of Health Stroke Scale [NIHSS]), infarct size, infarcts involving cortical regions, superficial versus deep territories, bilateral lesions, and fiber disruption as determined by fractional anisotropy.[10,11] To date, there are limited data on the prevalence of perfusion abnormalities in patients with AChA strokes and whether these abnormalities are associated with worse clinical outcome.[10-14] The primary goal of this study was to determine the frequency of perfusion abnormalities in patients with AChA stroke and the association with clinical outcome.

Methods

The study population was derived from a cohort of ischemic stroke patients admitted to 1 of 2 stroke centers (MedStar Washington Hospital Center, Washington, DC; Suburban Hospital, Bethesda, MD) between July 2001 and July 2014. Magnetic resonance imaging was performed using 1.5T (Twinspeed, General Electric, Chicago, IL), 3T (Skyra, Siemens, Malvern, PA) or 3T (Achieva, Philips, Netherlands) clinical scanners. All patients received magnetic resonance imaging (MRI) studies before any acute intervention including intravenous (IV) thrombolysis. The MRI studies were used for treatment decision making. The acute MRI studies included diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC), and perfusion-weighted imaging (PWI), including scanner-produced mean transit time (MTT) or time to peak (TTP) maps MRI studies (Figs 1, 2). The parameters for the PWI gradient-echo planar series included 20 contiguous axial oblique slices with single-dose gadolinium contrast injection of .1 mmol/kg of gadolinium (gadolinium-DTPA; Magnevist, Bayer Schering Pharma, Whippany, NJ) through a power injector using 25-40 phase measurements, TR/TE = 1500-2200/45 ms, acquisition matrix of 64×64-128×128-256×256, 7-mm slice thickness, and 22- to 24-cm field of view. The MTT maps were calculated as the first moment of the time concentration curves divided by the zeroeth moment with no arterial input correction or deconvolution. The TTP maps were calculated as the time from gadolinium arrival to the time of maximal parenchymal concentration. The DWI and PWI series were acquired co-localized over the entire brain with a superior to inferior coverage of 14 cm. Patients with DWI or ADC changes restricted to the AChA territory and consistent with acute ischemic stroke according to previously published territory maps were included in the study.[2] Patients were excluded if the DWI was negative for acute ischemia, not restricted to the AChA territory, or the MTT

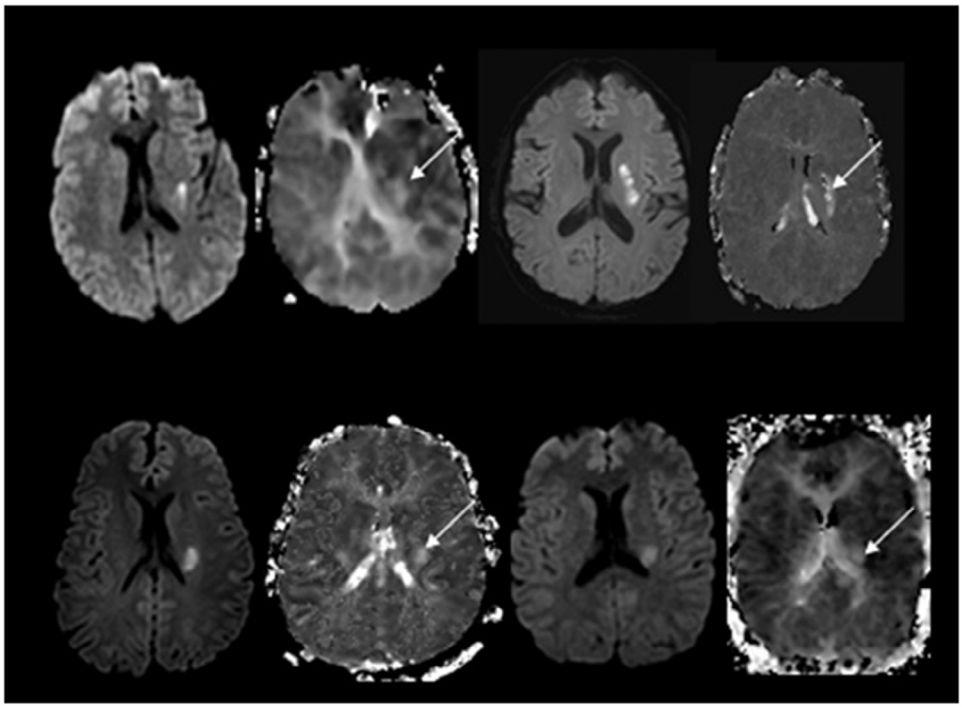

Figure 1. *DWI and paired PWI (MTT) images showing AChA ischemic strokes with positive perfusion deficits (arrows). Abbreviations: DWI, diffusion-weighted imaging; MTT, mean transit time; PWI, perfusion-weighted imaging.*