Infarction in the Anterior Choroidal Artery Territory: Clinical Progression and Prognosis Factors

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This study was undertaken to describe the still poorly known evolving profile of anterior choroidal artery (AChA) infarctions, identify their prognosis factors, and evaluate responses to intravenous (IV) thrombolysis. During 42 months, we prospectively enrolled patients with an isolated AChA stroke. Clinical and radiologic parameters were compared between patients with or without progression, defined as any clinical worsening. Factors associated with poor outcome (dependence or death) were tested, and IV thrombolysis responses were assessed. For the 100 of 1234 (8.1%) analyzed patients with AChA stroke (predominantly lacunar syndrome [88%]), mean admission and maximum National Institutes of Health Stroke Scale (NIHSS) scores were 4.4 and 5.2, respectively. Arterial hypertension (78%) and diabetes (30%) were the main vascular risk factors. Despite low 3-month mortality (3%), 26% of the patients were dependent; 46 patients with progressive stroke (over 56 ± 56 hours, 1.6 mean successive plateaus) had higher risks of dependence ($P < .0001$). An acute-phase NIHSS score of 6 or more significantly increased the risk of poor outcome ($P < .0001$). Maximum NIHSS score and progression were independently associated with poor outcome. Among 21 patients given IV thrombolysis, 12 AChA strokes continued to progress, leaving 8 disabled at 3 months. Almost half of AChA strokes progress during the first 2 to 3 days. Maximum acute-phase NIHSS scores and progression were independently associated with poor outcome, also strongly predicted by an NIHSS score of 6 or more at any time. Our unconvincing experience with IV thrombolysis means new therapeutic options and trials are needed, especially for patients with clinical progression and/or NIHSS score of 6 or more. **Key Words:** Anterior choroidal artery—stroke—progression—prognosis.

Introduction

The anterior choroidal artery (AChA) usually originates from the internal carotid artery and, rarely, from the middle cerebral or posterior communicating artery. The extent and boundaries of the AChA territory are still being debated. However, most authors agree with the following description[1-5]: the deep, or perforating, AChA territory includes the posterior two thirds of the internal capsule, the adjacent optic and auditory radiations, the medial portion of the globus pallidus, the tail of the caudate nucleus, and the posterior paraventricular part of the corona radiate, with the latter being the most debated.[6] The superficial territory includes the uncus,

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Received December 11, 2013; revision received January 23, 2014; accepted February 18, 2014.

Conflicts of interest: The authors have no interests to disclose.

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1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2014.02.013

the head of the hippocampus, the amygdala, the pyriform cortex, and the lateral portion of the lateral geniculate nucleus.[1] Isolated infarction in the AChA territory was considered rather rare, until the emergence of diffusion-weighted (DW) magnetic resonance imaging (MRI).

To date, only a few series specifically addressed infarctions in the AChA territory.[2-4,7,8] During the last decade, only 2 large case studies were reported, including 127[2] and 112 patients.[3] More recently, frequent clinical progression was mentioned but thoroughly analyzed in only 1 small study on 30 patients.[4] Only 1 other small study focused on the response to intravenous (IV) recombinant tissue plasminogen activator (rtPA).[9] However, because the motor pathway in the posterior part of the internal capsule is usually involved, residual motor disability can result from AChA stroke. The aims of our study were to better define clinical progression, prognosis factors, and response to IV thrombolysis.

Patients and Methods

Patients

This study was conducted at the Stroke Center of the Fort-de-France University Hospital, in Martinique (French West Indies). The mainly Afro-Caribbean population of Martinique has a relatively high stroke incidence.[10] Among the 1282 patients diagnosed with acute cerebral infarcts and consecutively included in a prospective acute stroke registry between July 1, 2007 and December 31, 2010, 48 were excluded because an acute phase MRI was not obtained. Hence, this analysis concerned 1234 patients.

Infarctions involving only the AChA territory were retained for this study. The structures described earlier were considered to be dependent on the AChA blood supply, with the deep and superficial territories of this artery being distinguished. Infarctions affecting other structures in addition to the AChA territory were not considered here. Our MRI protocol included, at least, gradient T2* echo, fluid-attenuated inversion recovery, DW imaging, and time of flight images of the circle of Willis sequences for all patients and was performed between days 0 and 3 after stroke onset.

Data Collected and Definitions

The following data were collected prospectively for all patients: demographic information; vascular risk factors (smoking, hypertension, diabetes mellitus, dyslipidemia, heart disease and history of stroke); current treatments; symptoms and neurologic examination at admission; lesion infarct size (<15 or >15 mm); localization of the acute infarctions; associated brain lesions; and the etiologic classification according to the Trial of ORG 10172 in Acute Stroke Treatment criteria, National Institutes of Health Stroke Scale (NIHSS) at admission (NIHSS-1), at the peak neurologic deficit (NIHSS-2), and at discharge (NIHSS-3), and the modified Rankin Scale (mRS) score at 3 months of follow-up. Poor outcome was defined as an mRS score greater than 2 (dependence or death).

During hospitalization, every neurologic deterioration was recorded. However, the classical definition of deterioration, that is, an increase of 4 points or more on the NIHSS scale[2,4] or 2 or more NIHSS points for motor function,[4] is not suitable in the setting of AChA infarctions as it is unable to detect even significant worsening. Indeed, in patients with AChA stroke, NIHSS scores are usually low, with only a few scale items, mainly only motor, being scored. Here, any persistent neurologic worsening was considered clinical progression, regardless of its possible impact on the NIHSS score: this designation was possible because our AChA stroke database explicitly includes this item. Progressive stroke was also diagnosed when patients reliably reported worsening before admission. We distinguished successive progressive plateaus from clinical fluctuations, whose deterioration was merely transient and fully reversible. Some patients exhibiting clinical progression underwent follow-up MRI for confirmation.

Statistical Analyses

Univariate analysis used a χ^2 test for dichotomous variables. Continuous variables were tested with Student *t* test or the Mann–Whitney *U* test, when normality was difficult to determine. First, we compared the recorded variables according to progressive stroke status. Then, multivariate logistic regression analyses were used to identify factors of dependence for activities of daily living. Variables with *P* less than .1 in univariate analyses were considered to represent explanatory variables and were evaluated together in subsequent multivariate analyses. *P* value less than .05 was defined as significance. All statistical analyses were computed with the SPSS package v13.0 for Windows.

Results

Patient Demographics

Among cerebral infarcts, 100 of 1234 (8.1%) corresponded to isolated AChA strokes and were included in the analysis. The main characteristics of those 100 included patients (Table 1) were age, 64 ± 17 years (mean ± standard deviation), and mean NIHSS-1 score, 4.4 ± 1.5; 79% were hypertensive and 31% had diabetes mellitus; the overwhelming predominance of lacunar syndrome in 88% of the patients, mainly with impaired motor function; the surprising paucity of superficial AChA territory damage, detected in only 3%; the rarity of hemianopia, observed in only 4 patients; the rarity of cardioembolic and atherothrombotic causes, despite a relatively comprehensive etiologic work-up including