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Acute anterior choroidal artery infarction: Clinical predictors of prognosis

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Abstract

Objectives: The clinical and prognostic features of acute anterior choroidal artery (AChA) stroke have not been sufficiently investigated in the context of new treatment approaches. *Methods:* This AChA stroke study was a prospective, observational cohort study conducted at a stroke center from July 1, 2020, to September 30, 2024, enrolling patients aged 28 to 85 years within 48 hours of symptom onset. Demographic and clinical characteristics were analyzed for all patients. Clinical characteristics were compared between groups with good outcomes (modified Rankin Scale score, 0-2) and poor outcomes (modified Rankin Scale score, 3-6), as well as between those treated with or without intravenous rt-PA or endovascular treatment. *Results:* 115 patients were studied. An NIHSS score of 6 or more at discharge increased the risk of poor outcome (P <0.001). Multivariable regression analysis showed that age (P = 0.04), smoking (P = 0.009), infarct size >15 mm (P = 0.036), and clinical progression (P < 0.001) were positively associated with poor outcome, whereas dual antiplatelet treatment was negatively associated with poor outcome (P = 0.014). Despite thrombolysis, 16 of 30 patients (53%) had an mRS score >2 at 90 days (P <0.005). Thrombectomy was performed in 18 patients, and 10 of them (56%) had mRS scores >2 at 90 days, compared to 8 (44%) with mRS scores ≤2 (P =0.023). *Conclusion:* There was no benefit from the thrombolysis within the therapeutic window in patients with AChA strokes. Additional treatments are needed and therapeutic trials should be designed, for example, new dual antiplatelet agents, bridging strategies such as thrombectomy after thrombolysis.

Keywords: Anterior choroidal artery, cardioembolic stroke, dual antiplatelet treatment, intravenous thrombolysis, endovascular treatment

INTRODUCTION

Strokes in the anterior choroidal artery (AChA) territory have been known for a long time and have a unique clinical course.[1] The AChA typically arises from the posterior wall of the internal carotid artery, although in some cases, it originates from the posterior communicating artery. The branches of the AChA follow an extensive course, supplying blood to multiple anatomical structures. Originating from the distal part of the internal carotid artery, the AChA travels along the optic tract and temporal lobe, ultimately reaching the choroid fissure at its medial edge.[2-5]

The AChA is divided into two segments: the superficial and the deep. The deep segment extends from its origin to the choroidal fissure, supplying deep brain structures such as the posterior limb and retrolenticular part of the internal capsule (including optic radiations), the lateral thalamus (including the lateral geniculate nucleus), the optic tract, the lateral cerebral peduncle, the globus pallidus internus, the tail of the caudate nucleus, mesial temporal structures (including the uncus), the head of the hippocampus, and the amygdala. The superficial territory includes the uncus, head of the hippocampus, amygdala, piriform cortex, and the lateral portion of the lateral geniculate nucleus.[4-7]

Infarctions in the AChA territory often result in a wide spectrum of neurological deficits, with typical symptoms including the triad of hemiparesis, hemianesthesia, and hemianopia. Although there are anastomotic branches between the AChA and the middle cerebral artery, posterior communicating artery, and posterior cerebral artery, the collateral circulation of the perforators supplying blood to the posterior limb of the internal capsule is very poor. As a result,

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the posterior limb of the internal capsule is the most commonly affected infarction territory of the AChA, rather than other areas with richer collateral circulation. Due to its rarity, only a few series have specifically addressed infarctions in the AChA territory. In the last decade, only three large case studies have been reported.[8-10] More recently, frequent clinical progression has been noted, but detailed analyses have been limited in recent studies.[9,11] Only two studies have focused on the response to intravenous thrombolysis (IVT).[10,12] However, to our knowledge, there have been no clinical observations or trials after thrombectomy in AChA strokes.

Due to the lack of data on this subject, we conducted a single-center prospective cohort study to investigate the demographic and clinical characteristics, treatment outcomes, and the prognosis of acute ischemic stroke (AIS) in the anterior choroidal artery territory.

METHODS

We report our study in accordance with the STROBE guidelines (Strengthening the Reporting of Observational Studies in Epidemiology). Among the 1,350 patients diagnosed with acute cerebral infarcts and consecutively included in our acute stroke registry between July 1, 2020, and September 30, 2024, the study group comprised 60 men and 55 women, aged 28 to 85 years. They were examined upon admission by a neurology resident and within 24 hours by a senior neurologist. Only infarcts involving the AChA territory were included in this study. Structures previously described as connected to the AChA blood supply, including both deep and superficial regions of the artery, were considered in the analysis. Patients with post-thrombectomy anterior choroidal artery (AChA) stroke were also included, with the specific aim of investigating outcomes in cases where the AChA stroke occurred subsequent to thrombectomy. Infarcts affecting areas beyond the AChA territory were excluded. Our MRI protocol included at least gradient echo T2*, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted (DW) imaging within the first week after stroke onset to demonstrate deep, superficial, or combined territory infarcts. We performed transesophageal echocardiography within 4 weeks after stroke onset in all patients, with a systematic search for aortic arch plaques. The Ethics Committee of Ege University Medical School approved this study, and patients provided informed consent.

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 neurological examination at admission; lesion infarct size (<15 mm 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) and at discharge (NIHSS-2), and the modified Rankin Scale (mRS) score at 30 and 90 days of follow-up. Poor outcome was defined as an mRS score greater than 2 (dependence or death). In addition, the stenosis of intracranial and extracranial arteries was recorded. Subsequent follow-up data at 7, 30, and 90 days were obtained locally and reviewed centrally. A good outcome was defined as an mRS score of 0 to 2, and a poor outcome as an mRS score of 3 to 6. Clinical progression was traditionally defined as the worsening by 4 points or more on the NIHSS scale, or 2 or more NIHSS points worsening limited to motor function.[13,14] However, since NIHSS scores are usually low in AChA infarcts, with only a few scale items (mainly motor) being scored, the suggestion by Chausson was adopted in this study.[13] It was considered that any persistent neurological worsening should be defined as clinical progression, regardless of its possible impact on the NIHSS score.

The Ethic's Committe of the Ege University gave approval for this study. Patients gave informed consent for the study.

Statistical analysis

Categorical variables were presented as frequencies and percentages. Continuous variables with nonnormal distributions were summarized as median (interquartile range). The Pearson χ^2 or Fisher exact test and Wilcoxon tests were used for the statistical significance analysis of the outcomes, where appropriate. The Cox proportional hazards model was used to explore the factors associated with clinical progression, where NIHSS score at admission was grouped by 6. Multivariable logistic regression was conducted to define independent predictors of good and excellent outcomes, respectively.

For both the logistic regression model (predicting 90-day outcomes) and the Cox proportional hazards model (for time to clinical progression), the initial full models incorporated all clinically relevant baseline covariates. Variable selection was performed using a backward stepwise procedure. Predictors with a univariable association of $p \geq 0.20$ were considered for exclusion. Final model selection was guided by the Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC) to optimize parsimony and goodness-of-fit. Potential multicollinearity was assessed by examining pairwise correlations; variables with a correlation coefficient of $|r| \geq 0.7$ were not retained in the same model.

Odds ratios (ORs) and hazard ratios with 95% CIs were presented. The missing data were deleted from the analysis. All analyses were performed using SPSS 27.0 software (SPSS Inc., Chicago, IL, USA) and 2-sided P<0.05 was considered significant.

RESULTS

Demographic and clinical characteristics

In our analysis, 115 patients (8.5%) with isolated AChA were included. The main characteristics of these patients (Table 1) were median age, 68.57 (66- 69) and median NIHSS-admission score, 5 (4- 7); and median NIHSS-discharge score 4 (3- 7). MRI revealed that 37 (32.2%) patients had an infarct size greater than 15 mm. The frequent risk factors were hypertension in 64.3%, diabetes in 44.31%, hyperlipidemia in 49.6% and smoking in 26.1; characterized by lacunar syndromes in 60.9%% of the patients, mainly with impaired motor function (88.7%); hemianopia found in 6.9% of patients; the superficial AChA territory damage was less frequent than deep territory, detected in 11.3% vs 88.7; cardioembolic (19,1%) and severe carotid stenosis (>70%) and occlusion (17,4%) were rare despite a relatively comprehensive etiologic work-up; and the 3% mortality and neurological deterioration was more common in the first 7 days (21.7%). The different clinical features are reported in Table 1. For all patients with poor outcomes, the disability was because of residual motor deficit and/or walking diffculties with spasticity.

Clinical outcome

Progression of stroke was observed in 36 of 115 patients (31.3%). They were prominently more common in the first week (21.7%) but could be associated with the latter (9.6%). In Cox regression analysis, NIHSS-2 score ≥6 was the only independent predictor of clinical progression (HR 11.56, 95% CI 3.46–38.65, $p < 0.001$). Kaplan–Meier analysis showed significantly shorter survival without neurological deterioration among patients with NIHSS-2 score ≥6 (median 4.0 days, 95% CI: 2.2–5.8) compared with those with NIHSS -2 score <6 (log-rank $\chi^2 = 111.0$, $p < 0.001$). Table 2 shows the Cox regression analysis. The mean length of hospitalization was longer for progressive stroke patients than others (14.9$\pm$5.4 vs 9.7 $\pm$ 3.1; $P < 0.001$). There were no group differences between the initial clinical motor and sensory presentation and IV rtPA use (90% vs 88% of 115) or thrombectomy (16.7% vs 7.7% of 115). The lesion size on MRI was larger than >15 mm in patients with progressive stroke patients than others (66.7% vs 33.3%) (OR, 15.56; 95%CI, 5.83 - 41.48; $P < 0.001$).

Predictive factors affecting the outcome

Patients with poor outcomes at 90 days (n = 37) were compared with those with good outcomes (n = 78). Univariate analysis showed the following parameters associated with poor outcome: Hypertension, smoking, clinical progression, carotid stenosis>70% and occlusion, atrial fibrillation, NIHSS score ≥6 at admission and discharge, and admission infarct size greater than 15 mm, deep territory involvement, IV thrombolysis and thrombectomy (Table 3). Multivariable logistic regression analyses showed that hypertension ($P = 0.03$), smoking ($P < 0.01$), severe carotid stenosis ($P = 0.04$) and clinical progression ($P < 0.001$) and dual antiplatelet treatment (P = 0.01) were independently associated with outcome (Table 4).

Outcome after IV thrombolysis

IV rtPA was administered in 30 patients; 21 (%60) of them had NIHSS-1 scores ≥6 and 9 (11.3%) had NIHSS-1 scores <6 and were treated 265 ± 48 minutes after symptom onset. Despite thrombolysis, NHISS-2 score ≥6 was higher at discharge (60% vs 30%; $P < 0.001$) and 19 of 30 strokes (63.3%) continued to progress and 16 of 30 patients had 90 days mRS scores greater than 2 (OR, 3.48; 95%CI, 1.46- 8.32; $P < 0.005$). No hemorrhages were observed.

Outcome after IV thrombectomy

The 18 patients who received thrombectomy for carotid artery thrombus were more severely impaired than those who did not undergo

Table 1: Demographic and Clinical Characteristics of Anterior Choroidal Artery Territory Stroke at Baseline

Characteristics	All Patients (n = 115)
Age (y), median (IQR)	68.57 (66-69)
Sex, n (%)	
Male	59 (51.3)
Female	56 (48.7)
Risk factors, n (%)	
Hypertension	74 (64.3)
Diabetes	51 (44.3)
Hyperlipidemia	57 (49.6)
Ischemic Heart Disease	26 (22.6)
Atrial Fibrillation	22 (19.1)
Smoking	30 (26.1)
Clinical course, n (%)	
Prior transient ischemic event	20 (17.4)
Clinical progression	36 (31.3)
Progression in 7 days	25 (21.7)
Progression in >7days	11 (9.6)
NIHSS score, mean±(SD)	
At admission	5.3 ± 1.9
At discharge	4.7 ± 2.4
Symptom, n (%)	
Hemiparesis	102 (88.7)
Hemianesthesia	41 (35.7)
Aphasia	19 (16.4)
Hemianopia	8 (6.9)
Location, n (%)	
Superficial territory	13 (11.3)
Deep territory	102 (88.7)
Affected areas in the deep location, n (%)	
Posterior limb of the internal capsule	98 (85.2)
Posterolateral striatum	57 (49.6)
Paraventricular corona radiata	62 (53.9)
Infarct size≥15 mm, n (%)	37 (32.2)
Carotid artery disease, n (%)	
Occlusion	9 (7.8)
Severe stenosis >70%	11 (9.6)
Plaques <50%	82 (71.3)
None	13 (11.3)
IV thrombolysis, n (%)	30 (26.1)
Time to needle, mean ± (SD)	265 ± 48
EVT, n (%)	18 (15.7)
Time to intervention (mins) mean ± (SD)	302 ± 24

EVT. Endovascular treatment; IVT. Intravenous thrombolysis

Table 2: Cox Regression Analysis for Clinical Progression in Patients With Anterior Choroidal Artery Territory Stroke

Variable	HR	95% Confidence Interval	P Value
Age	1.01	0.98-1.05	0.31
Smoking	1.19	0.57-2.48	0.64
Atrial fibrillation	2.58	0.94-5.29	0.10
NIHSS-2	14.58	4.72-45.04	**<0.001**
Endovascular therapy	1.51	0.53-4.27	0.43
Intravenous thrombolysis	0.33	0.12-1.03	0.052

HR: hazard ratio, NIHSS: National Institutes of Health Stroke Scale

thrombectomy (respective NIHSS-2 scores ≥6, 45.2% vs 54.8%; *P* <0.001) and were treated 303 ± 24 minutes after symptom onset. After thrombectomy, 10 of 18 patients (55.6%) had 90-day mRS scores ≥2, while 8 (44.4%) had mRS scores ≤2 (*P* = 0.023). When mRS scores were evaluated by multivariable regression analysis at 90 days, it was found that neither thrombolysis nor thrombectomy had any effect on the outcome. No reperfusion injury was observed.

Table 3: Differences between patients with good (mRS score ≤2) (n = 78) and poor (mRS score >2) (n = 37) outcomes at 90 days

Characteristic	mRS score		*P*	OR (95% CI)
	≤2 (n = 78)	>2 (n = 37)		
Age (y), mean ± SD	67.31 ± 11.94	71.22 ± 11.43	0.099	
Male sex, n	34 (436)	22 (59.5)	0.082	1.89 (0.86-4.2)
Risk factors				
Hypertension	45 (57.7)	29 (78.4)	0.024	2.66 (1.08-6.55)
Diabetes	32 (41)	19 (51.4)	0.20	1.52 (0.69-3.33)
Hyperlipidemia	40(51.3)	17 (45.9)	0.37	0.81 (0.37-1.77)
Smoking	12 (154)	18 (48.6)	0.001	5.21 (2.14-12.70)
Carotid stenosis>70% and occlusion	8 (40)	12 (60)	0.005	4.20 (1.54-11.47)
Coronary heart disease	14 (58.3)	10 (41.7)	0.19	1.69 (0.67-4.28)
Atrial fibrillation	9 (40.9)	13 (59.1)	0.004	4.15 (1.58-10.94)
Symptoms				
Motor	66 (84.6)	36 (97.3)	0.29	6.55 (0.82-52.39)
Sensory	26 (33.3)	15 (40.5)	0.32	1.36 (0.61-3.06)
Clinical progression	7 (9)	29 (78.4)	0.001	36.77 (12.21-110.73)
NIHSS score, mean ± SD				
Admission >6	14 (17.9)	20 (54.1)	0.001	5.38 (2.26-12.80)
Discharge >6	7 (9)	24 (64.9)	0.001	18.72 (6.69-52.39)
Admission infarct size ≥ 15 mm	15 (19.2)	18 (48.6)	0.001	3.98 (1.69-9.37)
Superficial territory	12 (15.4)	1 (2.1)	0.037	0.15 (0.02-1.22)
Deep territory	66 (84.6)	36 (97.3)	0.037	6.54 (0.82-52.39)
Single antiplatelet treatment	46 (59)	20 (54)	0.333	0.78 (0.35-1.71)
Dual antiplatelet treatment	30 (38.5)	17 (46)	0.135	1.69 (0.77-3.72)
IV thrombolysis	14 (17.9)	16 (43.2)	0.005	3.48 (1.46-8.32)
EVT	8 (10.3)	10 (27)	0.023	3.24 (1.16-9.08)
Death	0	4 (11)	0.01	1.12 (1.00-1.25)

Values in parentheses are percentage of column.
Abbreviations: mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; EVT. Endovascular treatment; IVT. Intravenous thrombolysis

Table 4: Multivariable Logistic Regression for Relative Factors Associated With 90-Days Clinical Outcome in Patients with Anterior Choroidal Artery Territory Stroke

Variable	OR	95% Confidence Interval	P Value
Age	0.94	0.88-1.03	0.06
Hypertension	6.93	1.11-13.38	**0.03**
Smoking	8.09	3.54-12.46	**0.003**
Carotid stenosis (>70%)	2.18	1.56-4.50	**0.04**
Coronary heart disease	6.33	0.74-15.31	0.09
NIHSS-2	1.87	0.11-30.71	0.66
Clinical progression	13.09	6.51-19.80	**<0.001**
Admission infarct size ≥ 15 mm	0.80	0.40-1.57	0.09
Dual antiplatelet therapy	0.14	0.08-0.71	**0.01**
Endovascular therapy	0.11	0.06-1.98	0.13
Intravenous thrombolysis	0.14	0.80-2.41	0.17

DISCUSSION

Our study includes a large series of patients with stroke in the AChA vascular territory, diagnosed with all MRI sequences. We included a significant number of patients with AChA-limited stroke, diagnosed and measured by MRI. These patients represented 8.5% of those with acute ischemic stroke admitted to our hospital, a rate consistent with other studies focusing on AChA infarctions.[9,10] In line with the literature on AChA strokes, we found that the clinical picture of isolated AChA infarctions is almost always a lacunar syndrome with predominant motor impairment, and that etiologies such as advanced atherosclerosis of the carotid artery or cardioembolism are common.[2,4,13,14] In terms of prognosis at day 90, there was a 3% mortality rate and 26% had clinical progression. These results suggest that patients with AChA stroke have lower mortality compared to patients with hemispheric lesions, although significant clinical deterioration may occur during the first week. This is largely parallel to the results of previous studies.[8-10] The increase in the dependency rate of patients at 90 days can be explained by clinical deterioration, especially in motor functions.

The relatively high risk of clinical progression in AChA infarctions within the first few days after symptom onset has been noted in previous publications.[8-10,15] The clinical progression of AChA infarctions was significantly higher than that of hemispheric and deep infarctions that developed after hospitalization. Nearly two-thirds of our patients experienced significant clinical progression during the first week. As we observed, patients with progressive stroke have more pronounced initial and final deficits, with odds ratios being significantly higher at 90 days in these patients. Previous studies have shown that this progression is not due to lesion

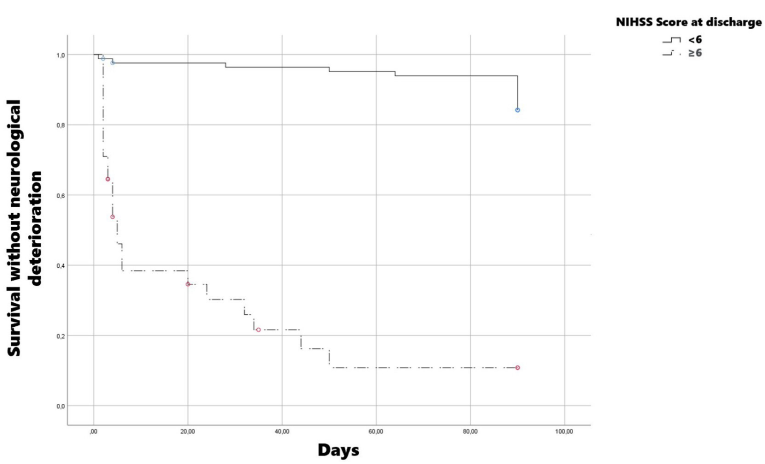

Figure 1. Kaplan-Meier analysis of clinical progression regarding to NIHSS score ≥6 versus NIHSS score <6.

volume, but rather fiber reduction during the acute stroke phase, as demonstrated by diffusion tensor MRI. This reduction is an important parameter predicting adverse motor outcomes in patients with AChA stroke.[16] Furthermore, there was significant infarct expansion in patients with progressive stroke, which appears to be the main mechanism of clinical progression. A DWI and perfusion study explained the higher rate of clinical worsening due to the expansion of the initial perfusion deficit, which subsequently leads to further growth of the ischemic zone.[17]

Recent studies have attempted to determine prognostic factors in AChA strokes. In the Cox regression analysis, where multiple covariates were evaluated, no independent factor affecting progression was identified. However, in univariate analyses, NIHSS scores ≥6 were associated with worse outcomes. Our results demonstrate the prognostic value of clinical progression and acute-phase NIHSS scores. The finding that patients with ischemic lesions larger than 15 mm had approximately twice the risk of a worse prognosis compared to the other patient group supports data from a previous series.[8-10] In addition, hypertension, smoking, atrial fibrillation, and deep/superficial territory infarctions were associated with a significantly increased risk of poor outcomes. According to our results, although NIHSS-1 and NIHSS-2 scores ≥6 indicate a poor prognosis in single-factor analyses, they were not found to be independent factors in multivariable regression and Cox regression analyses. However, when the results of conventional IV thrombolysis were evaluated in univariate analyses, almost half of the treated patients continued to progress, and one-third of them had poor outcomes. In multivariable analyses, IV thrombolysis was not found to be an independent prognostic factor. Regardless of whether thrombolysis or EVT was used, dual antiplatelet therapy was associated with a better prognosis. IV thrombolysis has long been considered a unique treatment option within the therapeutic window, and the benefit of early dual antiplatelet administration for small occlusive thrombi was demonstrated in this study. In AChA strokes, where multiple factors may predict outcomes, additional treatments are needed, and therapeutic trials should be designed, such as new dual antiplatelet agents or bridging strategies like thrombectomy after thrombolysis.

In conlusion, there was no benefit from the thrombolysis within the therapeutic window in patients with AChA strokes. Additional treatments are needed and therapeutic trials should be designed, for example, new dual antiplatelet agents, bridging strategies such as thrombectomy after thrombolysis.

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DISCLOSURE

Data availability: The data that support the findings of this study are available from the corresponding author, upon reasonable request.

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