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Acute ischemic stroke in anterior choroidal artery territory

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ABSTRACT

Objective: The aim of the study was to describe a series of patients with acute ischemic infarct in the anterior choroidal artery (AChA) territory. Moreover, we analyzed the prevalence of these strokes and compared them with hemispheric and deep infarcts. Finally, we hypothesized that the size of the infarct could be related to aetiology and prognosis.
Methods: We studied a prospective series of 1350 patients with acute ischemic stroke. We analyzed the following factors: age, gender, diabetes mellitus, hypertension, hyperlipidaemia, current smoking, ischemic heart disease, previous stroke, peripheral arterial disease, prior antithrombotic treatment, major cardioembolic source, severe arterial stenosis, initial severity, progression, mortality, disability, and recurrence rate at three months. AChA strokes were classified as small (<20 mm) or large (≥20 mm), as measured by diffusion-weighted MRI, and compared by size in the analysis.
Results: 112 patients (8.3%) had an ischemic lesion restricted to the AChA territory (large: 42 patients, small: 70 patients). Patients with AChA infarcts were younger, more likely to be diabetic, and predominantly male. We found significant differences in the rate of major embolic sources, recurrence, progression and prognosis. Large AChA strokes were associated with embolic pathologies and had worse prognosis than small AChA strokes.
Interpretation: Infarcts in the AChA territory have different aetiological mechanisms and outcome than other territories. Large AChA infarcts have a higher association with an embolic source and worse prognosis than small lesions.

1. Introduction

In 1925, Foix [1] described the Anterior choroidal artery (AChA) syndrome, which includes, in its complete form, the triad of hemiparesis, hemianaesthesia, and hemianopia. The AChA is a small artery that commonly originates in the posterior wall of the internal carotid, 2 to 5 mm distal to the posterior communicant artery (PCoA) and 2 to 5 mm proximal to the intracranial carotid bifurcation in 96–99.5% of cases [2]. The AChA territory shows large variations amongst individuals. The most reported supply areas include: the posterior limb of the internal capsule, optical tract, lateral geniculate body, medial temporal lobe, and medial part of the pallidum [3–12]. Other territories, such as the lateral thalamic border and the medial part of the lentiform nucleus, are still subject to debate, although the most controversial territory is the posterior paraventricular territory [5,13–15]. Despite its small size (0.5–2.0 mm), the AChA has perforating branches (between 2 and 9 AChA perforators with a diameter that varies from 90 to 600 μM) that have been identified in microdissection studies [16].

The origin and incidence of AChA infarcts is controversial [17–35]. It has been postulated that AChA infarcts are due to Small Vessel Disease (SVD) [17–19], although other studies related AChA infarcts to Large Vessel Disease (LVD) [20–22], cardioembolic, or other determined or undetermined causes [15,23–25]. Moreover, there are few studies of prognosis in AChA infarcts and most of them are case studies [18]. The main objective of our study was to study the vascular risk factors, aetiology and clinical evolution in patients with AChA infarct and compare them with unselected patients with ischemic lesion at other deep or cortical sites. Additionally, we evaluated whether the ischemic lesion size, dichotomized at 20 mm in patients with AChA infarct, allowed us to establish potential causes of stroke or provided prognostic data.

2. Patients and methods

From January 2003 to January 2007, 1669 consecutive patients with a diagnosis of acute ischemic stroke were prospectively evaluated at Hospital del Mar during the first 24 h after symptoms onset. We excluded 231 patients who presented transient ischemic stroke without radiological ischemic lesion and 88 patients without definitive localization of the ischemic lesion or lost during follow-up. The study included 1350 patients.

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3. Stroke localization

All radiological data were interpreted by trained radiologists (XP, AS), who were blind to patient data. All patients had a computed tomography (CT) study at hospital admission. New radiological studies [CT: ($n=456$); Magnetic Resonance Imaging (MRI) ($n=669$)] were performed during hospitalization to identify the ischemic lesion; when the infarct involved the AChA territory, the lesion was measured by MRI. Patients were classified in three groups: deep infarcts, including brainstem infarcts ($n=62$); hemispheric infarcts (cortical lesions in the territory of one of the large cerebral arteries, including the underlying deep territory of these arteries that might be involved [36,37]: MCA, $n=693$; ACA, $n=19$; PCA, $n=71$; cerebellum $n=78$); and AChA infarcts, including posterior limb of the internal capsule, optical tract, lateral geniculate body, medial temporal lobe, and medial part of the pallidum. In the AChA territory, the infarct was measured by the largest diameter in diffusion-weighted images (DWI) from the MRI study, except in one patient with a pacemaker. The AChA patients were divided into two groups according to the size of the infarct: large (diameter$\geq$20 mm) and small (diameter$<$20 mm) (Fig. 1). This cut-off point was chosen as described in previous reports [15,27]. The MRI study was performed without time-differences between patients with large and small infarct [6.47 days (SD: 2.27)]versus [6.10 days (SD: 1.88), $p=0.438$].

4. Vascular risk factors

Vascular risk factors were defined as follows: hypertension (patient's self-report of hypertension, use of antihypertensive drugs, or a systolic blood pressure >160 mm Hg and diastolic blood pressure >90 mm Hg, recorded at least two weeks after stroke onset); diabetes mellitus (fasting blood glucose level 120 mg/dl, patient's self-report of diabetes, or use of specific medication); hyperlipidaemia (cholesterol>200 mg/dl or triglycerides>150 mg/dl, patient's self-report of hyperlipidaemia, or use of specific medication); smoking habit (current smoker); ischemic heart disease (documented history of angor pectoris or myocardial infarct); peripheral arterial disease (documented history, intermittent claudication, or ankle-brachial index <0.90 in either leg); previous diagnosis of stroke; prior use of treatment with antithrombotic drugs (anticoagulant or antiplatelet).

5. Potential underlying stroke causes

All patients had an extra and intracranial arterial study. The arterial stenosis was established by concordance in at least two of the following neurovascular explorations: continuous Doppler, high-resolution echography techniques, MRI-angiography, CT-angiography or digital angiography (performed only in 86 patients). Cardiologic study included at least 24 h of cardiac monitoring or EKG recording and transthoracic or transoesophageal echocardiography. Presence of severe arterial stenosis (meaning $\geq$70% of the arterial diameter or extracranial occlusion) and major cardioembolic sources according to the previous validated criteria [26] were reported.

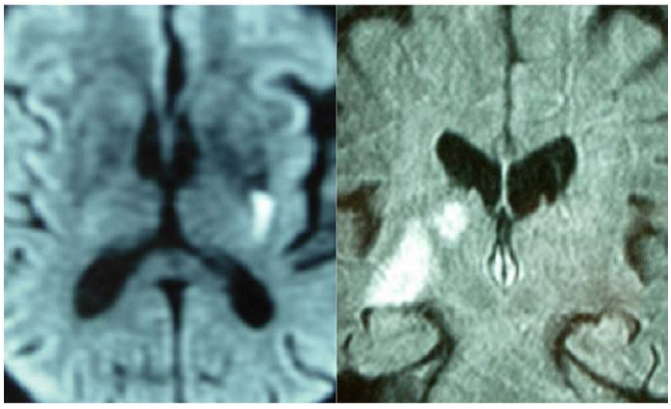

Fig. 1. Classification of anterior choroidal lesions according to the size of the infarct. Right: large (diameter$\geq$20 mm). Left: small (diameter$<$20 mm).

6. Clinical and prognostic data

Initial clinical severity was measured with the National Institutes of Health Stroke Scale (NIHSS) at hospital admission. Clinical progression was defined as the worsening by 4 or more points on the NIHSS within the first 72 h from stroke onset. The outcome was established at three months, either by clinical examination in the hospital or by telephone interview ($n=186$). Moderate–severe disability (3 to 5 points in the modified Rankin Scale) was considered a poor outcome. We also documented recurrence and mortality in the follow-up period.

7. Statistical analysis

Univariate analysis was performed with the Chi^2 test for dichotomous variables. Continuous variables were tested by the *t*-test or the Mann–Whitney test if normality was difficult to assume. The adjusted odd ratios (OR) were obtained by a logistic regression model including the factors showing an association in univariate analysis ($p<0.1$). In the first part of the study, we compared the registered variables according to the topographic location of ischemic lesion: AChA infarct, hemispheric infarcts, and deep infarcts. In the second part, we performed a statistical analysis comparing patients with large and small AChA infarcts. Values of $p<0.05$ were considered significant. Results are presented using 95% confidence intervals (CI). Statistical analyses were performed with the SPSS package 13.0 for Windows.

8. Ethics

The data for the study were collected from our hospital's prospective clinical protocols, which comply with the local ethics guidelines.

9. Results

We analyzed 1350 patients with an acute ischemic stroke: mean age 74.1 (SD: 11.6), range 26 to 99; 625 males (51.7%) and 585 females (48.3%). Patients excluded by incomplete data or follow-up were older (mean 78.3 ± 8.2) but had no differences in initial stroke severity, poor outcome at hospital discharge (15/111) or vascular risk factors ($p<0.001$).

The study detected 112 patients (8.3%) with AChA infarct, 377 patients (27.9%) with deep infarct, and 861 patients (63.8%) with hemispheric infarcts. There were no differences in the diagnostic workup between groups and a similar proportion of echocardiographic studies: AchA (63.5%), deep (61.1%) and hemispheric (61.4%) infarcts.

The most common symptoms associated with AChA infarcts were contralateral motor weakness, $n=87$ (77.6%); sensory dysfunction, $n=85$ (75.8%); aphasia, $n=19$, all in the dominant hemisphere (16.9%); contralateral hemianopia, $n=16$ (14.2%); hemiparesis–ataxia, $n=14$ (12.5%); and confused state, $n=3$ (2.67%). The complete classical syndrome (hemiparesis, hemianaesthesia, and hemianopia) was registered in 12 (10.7%) patients. The vascular risk factors, aetiologic and prognostic data of patients with AChA infarct, and their comparison with patients with deep and hemispheric infarct are summarized in Tables 1a and 1b.

Table 1a
Differences between deep and AChA infarcts.

	Total	AChA	Deep infarcts	p	Adjusted OR (95% CI)
	n = 489 (%)	n = 112 (%)	n = 377 (%)		
Gender (male)	272 (55.6)	73 (65.2)	199 (52.8)	p = 0.020[a]	1.799 (1.10–2.94)[b]
Age — mean (SD)	71.5 ± 11.2	69.4 ± 11.3	72.3 ± 11.1	p = 0.019[a]	0.973 (0.95–0.99)[b]
NIHSS median (q1–q3)	3 (2–5)	4 (3–7)	3 (2–4)	p < 0.001[a]	1.121 (1.04–1.20)[b]
Diabetes mellitus	171 (35)	48 (42.9)	123 (32.6)	p = 0.046[a]	1.630 (1.01–2.65)[b]
Arterial hypertension	343 (70.1)	81 (72.3)	262 (69.5)	p = 0.566	
Hyperlipidaemia	195 (39.9)	48 (42.9)	147 (39)	p = 0.463	
Current smoking	117 (23.9)	28 (25)	89 (23.6)	p = 0.762	
Ischemic heart disease	60 (12.3)	12 (10.7)	48 (12.7)	p = 0.568	
Previous stroke	63 (12.9)	15 (13.4)	48 (12.7)	p = 0.855	
Peripheral arterial disease	51 (10.4)	10 (8.9)	41 (10.9)	p = 0.554	
Antithrombotic pre-treatment	162 (33.1)	26 (23.2)	136 (36.1)	p = 0.011[a]	0.489 (0.28–0.84)[b]
Major cardioembolic source	69(14.1)	19 (17)	50 (13.3)	p = 0.323	
Severe arterial stenosis	50 (10.2)	20 (17.9)	30 (8)	p = 0.002[a]	1.917 (0.98–3.76)[b]
Mortality	19 (3.9)	3 (2.7)	16 (4.2)	p = 0.452	
Poor outcome	71 (15.1)	28 (25)	43 (11.9)	p < 0.001[a]	2.283 (1.21–4.29)[b]
Progression	66 (13.5)	28 (25)	38 (10.1)	p < 0.001[a]	2.946 (1.49–5.80)[b]
Recurrence	35 (7.2)	8 (7.1)	27 (7.2)	p = 0.995	

p obtained in univariate analysis.
[a] Selected to regression analysis.
[b] Adjusted OR with statistical significance in logistic regression.

Of the whole series, severe arterial stenosis was detected in 266 (19.7%) patients: carotid stenosis, *n* = 137 (10.14%); carotid occlusion, *n* = 38 (2.81%); and intracranial stenosis or vertebral occlusion, *n* = 91 (6.74%). Cardiac embolic disease was detected in 499 (33.3%) patients: atrial fibrillation, *n* = 328 (24.29%); dilated cardiomyopathy, *n* = 67 (4.96%); valvular disease, *n* = 51 (3.77%); and other determined or undetermined causes, *n* = 53 (3.92%). The rate of infrequent cause of stroke was similar in the territories evaluated: AChA, 3 of 112 (2.67%); deep, 8 of 377 (2.12%); and hemispheric, 26 of 861 (3.01%). Comparing patients with AChA infarct and those with deep infarct, we found that AChA stroke patients were younger; less likely to be pre-treated with antithrombotic therapy; and more likely to be male, diabetic, and have severe arterial stenosis, higher initial severity and poor outcome. When comparing AChA infarcts with hemispheric infarcts, we again found that AChA stroke patients were younger, more often male and diabetic, and less likely to be pretreated with antithrombotic therapy than hemispheric stroke patients. AChA infarcts were less associated with embolic sources, and showed higher risk of progression but lower risk of recurrence or mortality than hemispheric infarcts (Tables 1a and 1b).

Large AChA infarcts were detected in 42 patients (3.1%) and small AChA infarcts in 70 patients (5.2%). There were no differences between large and small AChA strokes for sex or age (see Table 2). In those patients with a large AChA infarct, we found more presence of cardioembolic sources and severe carotid stenosis than in patients with small AChA infarcts (adjusted OR: 4.55 and 4.07, respectively). Due to the low number of deaths (3) we were unable to find any differences related to mortality.

10. Discussion

There is scarce information in the literature concerning the prevalence of AChA infarcts in patients with acute ischemic stroke. The main drawback is the heterogeneity of the series studied, with most being a selected series of patients. Other studies include infarcts not limited to the AChA territory, with AChA strokes accounting for 2.9% to 11% of patients with acute ischemic stroke [13,15,34,35]. None of these studies has been able to provide the prevalence of AChA strokes from the unselected total of ischemic strokes. In our study, we have included all patients attended in our centre with the diagnosis of AIS, including both anterior and posterior circulation. We obtained a rate of 8.3%, which is comparable with the previous literature. Several studies using CT and MRI have been published [28–34] but only in two previous reports [13,35] have DWI–MRI sequences been used, in a small number of patients, to identify AChA infarcts. Our study is the first to contribute a large number of patients with AChA-restricted infarcts diagnosed and measured by DWI–MRI. According to previous reports, presentation as the classic syndrome was infrequent [5–15]. AChA stroke syndrome is known to be very chameleon-like; the most common symptoms were motor impairment followed by sensory dysfunction, reflecting the direct capsular impact. One patient suffered severe pseudobulbar palsy secondary to acute bilateral AChA infarct, confirming other reported cases [35,39].

Previous aetiopathogenic studies are contradictory. Traditionally, infarcts in AChA territory have been considered as lacunar infarcts, because of the small artery diameter. Following this hypothesis, a previous report [17] found a low rate of embolic sources in a study of 31 patients. Nevertheless, only 15 of 31 patients underwent echocardiography. A later study [23] of 16 patients with AChA infarct determined by MRI suggested that large artery thromboembolism and cardiac embolism are the most common causes of AChA infarcts.

Comparing patients with AChA, hemispheric, and deep infarcts, we found significant differences in vascular risk factors, potential causes of stroke, and outcome for patients with AChA infarcts. These patients were also younger and more likely to be male and diabetic than the

Table 1b
Differences between hemispheric and AChA infarcts.

	Total	AChA	Hemispheric infarcts	p	Adjusted OR (95% CI)
	n = 973 (%)	n = 112 (%)	n = 861 (%)		
Gender (male)	502 (51.6)	73 (65.2)	429 (49.8)	p = 0.002[a]	1.511 (0.95–2.40)[b]
Age — mean (SD)	74.6 ± 11.4	69.4 ± 11.3	75.4 ± 11.6	p < 0.001[a]	0.986 (0.97–0.99)[b]
NIHSS median (q1–q3)	6 (3–14)	4 (3–7)	6 (3–15)	p < 0.001[a]	0.939 (0.90–0.98)[b]
Diabetes mellitus	318 (32.7)	48 (42.9)	270 (31.4)	p = 0.015[a]	1.954 (1.24–3.08)[b]
Arterial hypertension	689 (70.8)	81 (72.3)	608 (70.6)	p = 0.709	
Hyperlipidaemia	380 (39.1)	48 (42.9)	332 (38.6)	p = 0.381	
Current smoking	200 (20.6)	28 (25)	172 (20)	p = 0.216	
Ischemic heart disease	162 (16.6)	12 (10.7)	150 (17.4)	p = 0.073[a]	0.716 (0.36–1.44)
Previous stroke	166 (17.1)	15 (13.4)	151 (17.5)	p = 0.273	
Peripheral arterial disease	100 (10.3)	10 (8.9)	90 (10.5)	p = 0.617	
Antithrombotic pre-treatment	395 (40.6)	26 (23.2)	369 (42.9)	p < 0.001[a]	0.613 (0.36–1.04)
Major cardioembolic source	399 (41)	19 (17)	380 (44.1)	p < 0.001[a]	0.345 (0.19–0.61)[b]
Severe arterial stenosis	236 (24.3)	20 (17.9)	216 (25.1)	p = 0.093[a]	0.311 (0.17–0.55)[b]
Mortality	203 (20.9)	3 (2.7)	200 (23.2)	p < 0.001[a]	0.162 (0.05–0.57)[b]
Poor outcome	202 (26.2)	28 (25)	174 (26.3)	p = 0.889	
Progression	163 (18.2)	28 (25)	135 (17.2)	p = 0.045[a]	3.351 (1.91–5.87)[b]
Recurrence	131 (13.5)	8 (7.1)	123 (14.3)	p = 0.037[a]	0.462 (0.20–0.99)[b]

p obtained in univariate analysis.
[a] Selected to regression analysis.
[b] Adjusted OR with statistical significance in logistic regression.

Table 2
Differences between large and small infarct in AChA territory.

	Large AChA	Small AChA	*p*, adjusted OR (95% CI)
	n = 42 (%)	*n* = 70 (%)	
Gender (male)	25 (59.5)	48 (68.6)	*p* = 0.331
Age — mean (SD)	69.8 ± 12.6	69.0 ± 10.1	*p* = 0.687
NIHSS median (q1–q3)	6 (2–8)	4 (2–5)	*p* < 0.001, OR = 1.12 (0.97–1.28)
Diabetes mellitus	16 (38.1)	32 (45.7)	*p* = 0.430
Arterial hypertension	33 (78.6)	48 (68.6)	*p* = 0.252
Hyperlipidaemia	13 (31)	35 (50)	*p* = 0.049, OR = 0.37 (0.13–0.99)[a]
Current smoking	9 (21.4)	19 (27.1)	*p* = 0.499
Heart ischemic disease	5 (11.9)	7 (10)	*p* = 0.752
Previous stroke	6 (14.3)	9 (12.9)	*p* = 0.830
Peripheral arterial disease	5 (11.9)	5 (7.1)	*p* = 0.392
Antithrombotic pre-treatment	9 (21.4)	17 (24.3)	*p* = 0.729
Major cardioembolic source	13 (31)	6 (8.6)	*p* = 0.002, OR = 4.55 (1.30–15.89)[a]
Arterial stenosis ≥ 70%	13 (31)	7 (10)	*p* = 0.005, OR = 4.07 (1.31–12.60)[a]
Mortality	2 (4.8)	1 (1.4)	*p* = 0.290
Poor outcome	17 (42.5)	11 (15.9)	*p* = 0.002, OR = 1.70 (0.53–5.43)
Progression	16 (38.1)	12 (17.1)	*p* = 0.013, OR = 2.15 (0.69–6.75)
Recurrence	4 (9.5)	4 (5.7)	*p* = 0.449

p obtained in univariate analysis.
[a] Adjusted OR with statistical significance in logistic regression.

other patient groups. Differences in age have already been described in a previous report [15], as has the association with diabetes [29]. Moreover, patients with AChA infarcts had a lower presence of severe arterial stenosis and cardioembolic sources than patients with hemispheric infarcts, but a higher presence of severe arterial stenosis than patients with deep infarcts. The most comprehensive previous work [15] studied the pathogenesis of AChA infarcts according to the infarct size, using CT scans to measure small (<20 mm) and large (≥20 mm) lesions. It also compared small AChA ($n = 77$) with small deep infarcts ($n = 83$), and large AChA infarcts ($n = 6$) with hemispheric infarcts ($n = 378$). No differences were found between large AChA infarcts and hemispheric infarcts, neither with respect to vascular risk factors nor to potential underlying stroke causes, which led to a conclusion that most such infarcts are caused by large artery thromboembolism or cardiac embolism. Moreover, in a finding that matches our results, the authors reported that small AChA infarcts were more associated with carotid stenosis but less associated with a cardioembolic source than were small deep infarcts in other territories.

Furthermore, our study found differences in prognosis between AChA infarcts and other ischemic infarcts. Patients with AChA infarcts had lower mortality and risk of recurrence than patients with hemispheric lesions, but higher risk of clinical progression during the first few days after infarct. Moreover, compared with deep infarcts, AChA infarcts had worse prognosis, showing an increased risk of dependent status at three months. However, the earlier study cited [15] found that small AChA infarcts had better prognosis in terms of 30-day case fatality and 1-year mortality than small infarcts in other territories. This was attributed to the younger age and fewer cardioembolic sources of AchA stroke patients, although multivariate analysis was not performed due to the small number of patients. Other studies of prognosis in AChA infarcts are mostly case reports [18].

Small infarcts may result from obstruction of perforating branches, whereas large infarcts could be caused by a complete AChA obstruction, perhaps resulting from large artery or cardiac embolism. However, in a previous angiographic study [31], the degree of AChA occlusion was not correlated with the extent of the infarct. This was attributed to variations between individuals in the distribution of the anastomosis with collateral circulation. In spite of this, it is reasonable to think that a complete occlusion may cause a larger infarct than the occlusion of a perforating branch.

In our study, we found an independent association between the infarct size, dichotomized at 20 mm, and the pathophysiological mechanism. Large AChA strokes were strongly associated with carotid stenosis and cardioembolic sources, compared with small AChA strokes. Moreover, we detected a significant impact on prognosis based on the size of ischemic lesion. Patients with large AChA infarcts showed greater severity and progression than patients with small AChA infarcts. The MRI–DWI study was performed within the first 10 days after stroke onset; therefore, larger size strokes with progressive clinical evolution might reflect the occlusion of the AChA and later collateral circulation failure. In agreement with this hypothesis, postoperative AChA infarcts have been reported as having a worse prognosis than spontaneous-onset infarcts, due to poor collateral circulation and anastomosis [38]. The previous study [15] did not compare prognosis between large and small AChA groups, probably because of the small number of large AChA infarcts ($n = 6$). In our series, large AChA infarcts showed a higher risk of poor prognosis than small AChA infarcts, even when adjusted for other risk factors.

Our study had several limitations. First, although the degree of arterial stenosis was reliable within the neurovascular explorations performed, only a few patients received arteriography. However, this methodology should not invalidate the reproducibility of the study. Second, echocardiography was not performed in all patients. Third, the MRI study was performed between 3 and 10 days after stroke onset. Therefore, we evaluated the definitive lesion but not the initial one. Nevertheless, using the dimensions of the DWI in the acute phase rather than at 1 or 3 months, we could identify small infarctions that are either not visible or not distinguishable from previous lesions on T2 or proton density MRI in conventional MRI [40].

11. Conclusion

Due to its location and size, the AChA constitutes a special territory where concurrent pathophysiological mechanisms may coexist. Moreover, AChA infarcts have different prognosis than hemispheric or deep ischemic strokes. The size of the ischemic lesion, dichotomized at 20 mm, could be a useful tool for studying the aetiology. In large infarcts, it is important to perform a comprehensive vascular study to rule out embolic sources.

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