Anterior Choroidal Artery Territory Infarction: A Small Vessel Disease

Askiel Bruno, MD, Neill R. Graff-Radford, MBBCh, MRCP, José Biller, MD, and Harold P. Adams Jr., MD

To investigate the cause(s) of infarction in the anterior choroidal artery territory, we studied 31 patients (18 men, 13 women) aged 19–82 (mean 58) years, with infarction in this territory documented by computed tomography. All patients were evaluated within 30 days after infarction and had carotid artery studies (arteriography in 17, duplex ultrasound in 14). Fifteen patients had echocardiography. Risk factors for atherosclerosis were chronic hypertension in 20 patients, smoking in 17, diabetes in 10, age >69 years in eight, and elevated serum cholesterol concentration in three. The percentage of patients with extracranial carotid artery stenosis (four of 31, 13%) and the severity of stenosis (mild in two, moderate in two) were similar to that reported in neurologically asymptomatic individuals. The percentage of patients with intracranial carotid artery stenosis (one of 17, 6%) or potential cardiac sources of emboli (two of 31, 6%) was also low. Our findings suggest that infarctions in the anterior choroidal artery territory usually result from small-vessel disease. Associated carotid artery stenosis and potential sources of cardiac emboli are rare and may be coincidental. (Stroke 1989;20:616–619)

The territory of the anterior choroidal artery (AChA) includes important motor and sensory structures,1–6 the destruction of which can produce severe neurologic impairment. AChA syndrome, consisting of hemiplegia, hemianesthesia, and homonymous hemianopsia, was first described by Foix et al.7 Hemiparesis is usually due to involvement of the posterior limb of the internal capsule, hemisensory loss to involvement of the ventral posterolateral nucleus of the thalamus or the thalamocortical fibers, and hemianopsia secondary to involvement of the lateral geniculate body or the geniculothalamic tract. Hemiparesis is the most constant deficit, dysarthria and hemisensory loss are less common, and hemianopsia is rare.8,9 An unusual form of homonymous hemianopsia, quadruple sectoranopsia with a spared thin strip of vision around the horizontal meridian in the blind field, has been noted in some patients with AChA territory infarction and has been suggested to be diagnostic of lateral geniculate body dysfunction.10,11 AChA territory infarction on the right side can produce spatial hemineglect, and infarction on the left side can cause a mild language disorder.8,9 Also, a syndrome of acute pseudobulbar mutism has been described in patients with bilateral AChA territory infarctions.12

The cause(s) of infarction in the AChA territory has (have) not been studied in a large group of patients. In previous reports, many patients did not have carotid artery studies or results were not given in detail. To investigate the cause(s) of infarction in this vascular territory, we studied the frequency and severity of ipsilateral carotid artery stenosis, potential sources of cardiac emboli, and the associated vascular risk factors in 31 patients with AChA territory infarction.

Subjects and Methods

Thirty-one patients with AChA territory infarction (18 men and 13 women), aged 19–82 (mean 58) years, were evaluated at the University of Iowa Hospital or Iowa City Veterans Administration Hospital within 30 days after infarction. Twenty-eight patients were white and three were black. Twelve patients were seen between August 1982 and June 1986, and eight of the 12 have been reported.9 The remaining 19 patients were identified consecutively and were evaluated prospectively between September 1986 and May 1988. AChA territory infarction was diagnosed by computed tomography (CT) when
A lesion was located primarily within this territory as outlined by a template of cerebral vascular territories (Figure 1). All infarctions involved the posterior limb of the internal capsule. For each patient, the assigned attending neurologist decided upon the diagnostic workup. All patients had a carotid artery study (arteriography in 17, duplex ultrasound in 14), complete blood count, prothrombin time, partial thromboplastin time, sedimentation rate, syphilitic serology, and fasting cholesterol concentration determinations. A fasting cholesterol level of >200 mg/100 ml was considered undesirable. Many patients, especially younger ones, also had antinuclear antibody, antithrombin III, protein C, and protein S determinations. Patients were classified as hypertensive if they had chronic elevated blood pressure (>140/90 mm Hg) requiring medical treatment.

Carotid stenosis was labeled as mild (16–49%), moderate (50–79%), or severe (80–100%) by the degree of luminal diameter reduction. Fifteen patients underwent echocardiography. Potential sources of cardiac emboli were diagnosed when there was atrial fibrillation, valvular heart disease, focal myocardial hypokinesis, or an intracardiac thrombus.

Results

Infarctions occurred on the left side in 19 patients and on the right side in 12. Clinical information and results of evaluation are summarized in Table 1. All 31 patients had hemiparesis, 22 (71%) had dysarthria, 15 (48%) had hemisensory loss to touch and pinprick, and homonymous visual field defects developed in two (6%) patients (hemianopsia in one and a superior quadrantanopsia in another). All patients had at least one of the vascular risk factors listed in Table 1 or were >69 years old. Twenty (65%) patients had chronic hypertension. Vascular risk factors in the 11 nonhypertensive patients included smoking as the only risk factor in five, smoking and oral contraceptive use in three, diabetes and age >69 years in two, and age >69 years as the only risk factor in one.

The extracranial carotid artery was normal in 27 (87%) patients (15 on arteriography, 12 on duplex ultrasound). Mild stenosis was present in two patients and moderate stenosis in two; no patient had severe stenosis or occlusion. A potential source of cardiac emboli was found in only two (6%) patients (atrial fibrillation in both). One patient with atrial fibrillation also had mild carotid stenosis on the side of infarction.

The intracranial carotid artery was normal in 16 of 17 (94%) patients who had arteriography, and one (6%) had an irregular plaque with mild stenosis at the carotid siphon. The AChA was patent in 15 patients, and in two it was not visible. Nonvisibility of the AChA was not associated with proximal carotid artery stenosis or cardiac sources of emboli. We did not see an abrupt arterial cutoff, suggestive of embolization, in any patient.

Discussion

Neurologic deficits in our patients were similar to those reported in another study of 16 patients with AChA territory infarction. These deficits are not characteristic of lesions in the AChA territory. They can also result from infarctions in other vascular territories, such as those of the deep (lenticulostriate) or superficial branches of the middle cerebral arteries and the penetrating brainstem arteries. Absence of aphasia, depressed level of consciousness, or headache, abnormalities usually indicative of large cerebral infarctions, appears to be helpful in distinguishing involvement of the AChA territory from that of the superficial middle cerebral artery territory. None of our patients had aphasia, depressed level of consciousness, or headache. Similarly, the absence of brainstem signs and depressed level of consciousness is helpful in distinguishing AChA territory from brainstem infarction. To confirm infarction in the AChA territory, CT or magnetic resonance imaging is needed.

The percentage of our patients with extracranial carotid artery stenosis (four of 31, 13%) was low, and the severity of stenosis was mild to moderate (Table 1). Similar results of carotid artery studies were obtained in a large number of neurologically asymptomatic individuals. A similar percentage of patients with AChA territory infarction reported by Sterbini et al had a possible carotid embolic source (three of 27, 11%), but the severity of...
Table 1. Clinical Characteristics and Results of Evaluation in 31 Patients With Anterior Choroidal Artery Territory Infarction

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Neurologic deficits</th>
<th>Vascular risk factors</th>
<th>Blood test abnormalities</th>
<th>ICA stenosis</th>
<th>Source of cardiac emboli</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/19/F</td>
<td>HP, HS</td>
<td>SMK, BCP</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>2/27/M</td>
<td>HP, DA, HS</td>
<td>SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>3/29/F</td>
<td>HP, DA</td>
<td>SMK, BCP</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>4/38/F</td>
<td>HP, DA, HS</td>
<td>HTN, SMK, DM</td>
<td>None</td>
<td>None (d)</td>
<td>No</td>
</tr>
<tr>
<td>5/41/F</td>
<td>HP, DA</td>
<td>SMK, BCP</td>
<td>None</td>
<td>Siphon 30% (a)</td>
<td>No*</td>
</tr>
<tr>
<td>6/41/M</td>
<td>HP, DA, HS</td>
<td>SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>7/47/M</td>
<td>HP, DA, HS</td>
<td>HTN, DM</td>
<td>None</td>
<td>None (d)</td>
<td>No</td>
</tr>
<tr>
<td>8/47/M</td>
<td>HP, DA, HS</td>
<td>SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>9/49/F</td>
<td>HP, DA, HS</td>
<td>SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>10/50/F</td>
<td>HP</td>
<td>HTN, DM</td>
<td>Fasting chol 380</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>11/52/M</td>
<td>HP, DA</td>
<td>HTN</td>
<td>None</td>
<td>None (d)</td>
<td>No</td>
</tr>
<tr>
<td>12/53/F</td>
<td>HP, DA, HS</td>
<td>HTN, SMK</td>
<td>Fasting chol 240</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>13/58/M</td>
<td>HP, HS</td>
<td>HTN</td>
<td>None</td>
<td>None (d)</td>
<td>No</td>
</tr>
<tr>
<td>14/58/M</td>
<td>HP, DA, HS</td>
<td>HTN, SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>15/61/F</td>
<td>HP, DA</td>
<td>HTN, SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>16/64/M</td>
<td>HP, HS</td>
<td>HTN, SMK, DM</td>
<td>None</td>
<td>None (d)</td>
<td>No</td>
</tr>
<tr>
<td>17/66/M</td>
<td>HP, DA, HS</td>
<td>HTN</td>
<td>Fasting chol 279</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>18/66/M</td>
<td>HP</td>
<td>HTN, DM</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>19/66/M</td>
<td>HP, DA</td>
<td>HTN, DM</td>
<td>PLT 1,650,000</td>
<td>Proximal 50% (a)</td>
<td>No*</td>
</tr>
<tr>
<td>20/67/M</td>
<td>HP, DA</td>
<td>HTN, DM</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>21/68/M</td>
<td>HP, DA, HS</td>
<td>HTN</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>22/68/M</td>
<td>HP</td>
<td>HTN, SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No*</td>
</tr>
<tr>
<td>23/69/M</td>
<td>HP</td>
<td>SMK</td>
<td>None</td>
<td>None (a)</td>
<td>No</td>
</tr>
<tr>
<td>24/71/M</td>
<td>HP, DA, HS</td>
<td>HTN, SMK</td>
<td>None</td>
<td>Proximal 30% (a)</td>
<td>No*</td>
</tr>
<tr>
<td>25/72/M</td>
<td>HP, DA, HV</td>
<td>HTN, SMK</td>
<td>None</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>26/75/F</td>
<td>HP, DA, HV</td>
<td>HTN, SMK, DM</td>
<td>None</td>
<td>None (d)</td>
<td>Atrial fibrillation*</td>
</tr>
<tr>
<td>27/76/M</td>
<td>HP</td>
<td>HTN</td>
<td>None</td>
<td>Proximal 50–79% (d)</td>
<td>No*</td>
</tr>
<tr>
<td>28/76/F</td>
<td>HP, HS</td>
<td>DM</td>
<td>None</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>29/77/F</td>
<td>HP, DA</td>
<td>HTN</td>
<td>None</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>30/77/M</td>
<td>HP, DA</td>
<td>None</td>
<td>None</td>
<td>None (d)</td>
<td>No*</td>
</tr>
<tr>
<td>31/82/F</td>
<td>HP, DA</td>
<td>DM</td>
<td>None</td>
<td>Proximal 16–49% (d)</td>
<td>Atrial fibrillation*</td>
</tr>
</tbody>
</table>

Pt, patient; ICA, internal carotid artery; F, female; HP, hemiparesis; HS, hemisensory loss; SMK, smoking; BCP, birth control pills; (a), arteriography; M, male; DA, dysarthria; HTN, hypertension; DM, diabetes mellitus; (d), duplex ultrasound; chol, cholesterol; PLT, platelet count; HV, homonymous visual loss.

*Echocardiogram not done.

AChA occlusion in our two patients with this abnormality. However, proximal AChA occlusion by itself usually does not result in infarction, as demonstrated by iatrogenic occlusions of this artery in an attempt to treat Parkinson's disease.18,19 The best explanation for this is that anastomoses between the AChA and branches of the posterior communicating and posterior cerebral arteries23 supply blood to the AChA when its stem is occluded. Therefore, infarction probably occurs when a branch of the AChA, or the AChA itself and the arteries supplying collateral blood flow to it, become occluded. Because of their small size, occlusions of the AChA branches could not be diagnosed with certainty on arteriography. It is important to emphasize that abnormalities suggestive of embolization were not seen in any of our 17 patients who had arteriography.

The percentage of our patients with potential cardiac sources of emboli was also low (two of 31,
6% (Table 1). Decroix et al \(^8\) found the same frequency of potential cardiac sources of emboli (one of 16, 6%) in their patients with AChA territory infarction. Sterbini et al \(^6\) found a possible cardiac source of emboli in five of 27 (19%) patients with AChA territory infarction, but the types of abnormalities were not stated. In two reports by Helgason et al, \(^11\), \(^12\) 11 patients had spontaneous AChA territory infarction, and a possible cardiac embolic event was diagnosed in two of the 11 (atrial fibrillation in one and ischemic cardiomyopathy in the other).

The low rates of carotid artery stenosis or potential cardiac sources of emboli in our patients suggest that small-vessel disease is the principal cause of AChA territory infarction. The most common pathologic lesions responsible for small-vessel disease of the brain are microatheroma and lipohyalinosis. \(^20\), \(^21\) Chronic hypertension is the single most important risk factor for small-vessel disease of the brain, and 20 (65%) of our 31 patients had chronic hypertension. The importance of other vascular risk factors in the pathogenesis of small-vessel disease of the brain remains to be elucidated. However, since microatheroma histologically resembles atherosclerotic lesions in large arteries, we listed the vascular risk factors for large-artery atherosclerosis as well. Additional documented causes of AChA stenosis include neurovascular syphilis \(^1\) and iatrogenic occlusion during temporal lobectomy \(^11\) and during ligation of saccular aneurysms of the supraclinoid internal carotid artery, \(^22\) the posterior communicating artery, \(^16\) and the AChA. \(^23\)

Based on our experience it appears that the principal cause of infarction in the AChA territory is small-vessel disease, with chronic hypertension being the single most important risk factor. The percentage of patients with carotid artery stenosis or potential cardiac sources of emboli is low, which suggests that these abnormalities may be coincidental. Evaluation of patients with AChA territory infarction should be individualized, but carotid arteriography and echocardiography usually will not demonstrate a causative abnormality. Therapies directed at the risk factors for small-vessel disease may reduce the incidence of ischemic events in the AChA territory.

Acknowledgments

The authors wish to thank Dr. Hanna Damasio for expert advice regarding Figure 1 and Verna Lee for help with manuscript preparation.

References

2. Abbie AA: The clinical significance of the anterior cho
doidal artery. Brain 1933;56:233–246
3. Alexander L: The vascular supply of the striopallidum. Res
4. Carpenter MB, Nabuck CR, Moss ML: The anterior cho-
doidal artery: Its origins, course, distribution, and variations.
Arch Neurol 1954;71:714–722
5. Herman LH, Fernandes C, Gurdjian ES: The anterior cho-
doidal artery: An anatomical study of its area of distri-
bution. Anat Rec 1966;154:95–102
6. Rhoton AL Jr, Fujii K, Fradd B: Microsurgical anatomy of
7. Foix C, Chavany H, Hillemand P, Schiff-Wertheimer M:
Oblitération de l’artère choroïdienne antérieure. Ramollis-
sement de son territoire cérébral. Hémiplegie, hémianesthésie,
hémianopsie. Bull Soc Ophthalom Fr 1925;221–223
8. Decroix JP, Gravelleau PH, Masson M, Cambier J: Infar-
cion in the territory of the anterior choroidal artery: A
clinical and computerized tomographic study of 16 cases.
Brain 1986;109:1071–1085
9. Graff-Radford N, Damasio H, Yamada T, Estlinger PJ,
Damasio AR: Non-hemorrhagic thalamic infarction. Brain
1985;108:485–516
10. Frisen L: Quadrapel parietooccipital and sectorial optic atro-
phy: A syndrome of the distal anterior choroidal artery. J
Neurol Neurosurg Psychiatry 1979;42:590–594
choroidal artery-territory infarction: Report of cases and
NA: Acute pseudobulbar mutism due to discrete bilateral
capsular infarction in the territory of the anterior cho-
doidal artery. Brain 1989;111:1572
13. Damasio H: A computed tomographic guide to the identifi-
cation of cerebral vascular territories. Arch Neurol 1983;
40:138–142
WB: Extracranial carotid atherosclerosis in a general popu-
and neuropathologic findings. Neurology 1956;6:390–401
16. Sterbini GLP, Agatiello LM, Stocchi A, Solivetti FM: CT of
ischemic infarctions in the territory of the anterior cho-
doidal artery. J Neurol Neurosurg Psychiatry 1985;48:
714–722
17. Osborn AG: Introduction to Cerebral Angiography. Phila-
18. Cooper IS: Surgical alleviation of parkinsonism: Effects of
occlusion of the anterior choroidal artery. J Am Geriatr
Soc 1979;24:691–717
19. Rand RW, Brown RJ, Stern WE: Surgical occlusion of
anterior choroidal arteries in parkinsonism: Clinical and
neuropathologic findings. Neurology 1956;6:390–401
Neuropathol (Berl) 1969;12:1–15
21. Fisher CM: Capsular infarcts: The underlying vascular
22. Takahashi S, Kawata Y, Uemura K: CT findings on anterior
23. Yasargil M, Yonas H, Gasser J: Anterior choroidal artery
aneurysms: Their anatomy and surgical significance. Surg

Key Words • cerebrovascular disorders • risk factors •
small-vessel disease
Anterior choroidal artery territory infarction: a small vessel disease.
A Bruno, N R Graff-Radford, J Biller and H P Adams, Jr

Stroke. 1989;20:616-619
doi: 10.1161/01.STR.20.5.616
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1989 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/20/5/616

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in
Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office.
Once the online version of the published article for which permission is being requested is located, click Request
Permissions in the middle column of the Web page under Services. Further information about this process is
available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/