The Anterior Choroidal Artery Does Not Supply the Corona Radiata and Lateral Ventricular Wall

J.P. Mohr, MD; Wolfgang Steinke, MD; Serge G. Timsit, MD; Ralph L. Sacco, MD; and Thomas K. Tatemichi, MD

Background and Purpose: We sought first to characterize the clinical syndromes of patients found to have angiographic, computed tomographic, or magnetic resonance imaging scan indexes of anterior choroidal artery territory infarction and then to determine the frequency of involvement of the periventricular corona radiata in such patients.

Methods: Sixteen patients were selected based on angiographically, or surgically, documented occlusion of the anterior choroidal artery or based on infarcts whose minimal lesions included the anterior choroidal territory as defined by Kolisko and Beevor. We mapped the lesions using the templates of the Matsui and Hirano atlas and entered them into a computer using a program allowing overlapping diagrams of the cases.

Results: The anatomic distributions were fairly uniform, all involving the lower portion of the posterior limb of the internal capsule, the medial pallidum (75% of cases), cerebral peduncle in 44%, thalamus in 37%, and the medial temporal lobe in 38%. None extended outside these areas to include the upper corona radiata. The clinical picture corresponded to the well-established neurological syndrome featuring motor deficits with varying degrees of visual field and sensory impairments. Only two showed hyposthetic ataxic hemiparesis.

Conclusions: Our findings indicate that the syndrome of anterior choroidal artery infarction is fairly uniform; ataxic hemiparesis occurs infrequently; and lesions in the lateral ventricular wall and the corona radiata are not part of the territory supplied by the anterior choroidal artery. (Stroke 1991;22:1502–1507)

In 1891, Kolisko1 reported results of a pathological series using injection techniques to determine the territory of the anterior choroidal artery (AChA). According to his findings, the AChA supplies the posterior two thirds of the posterior limb of the internal capsule, the retrolenticular fibers posterior to the internal capsule, the medial aspect of the pallidum, the uncus, the posterior portion of the optic tract, the tail of the caudate nucleus, the lateral choroid plexus, and exceptionally superficial areas of the thalamus. Beevor2 added the anterior portion of the cerebral peduncle to the list of structures supplied by the AChA territory, and Abbie3 emphasized that the AChA also contributes to the supply of the lateral geniculate body. The results of these early studies were later confirmed by two other series also using injection techniques.4,5 The territory of AChA infarction in the few available autopsies was smaller compared with that found in the injection studies but consistently involved only the posterior limb of the internal capsule and the pallidum.1,6–9

Although Kolisko1 had already outlined the typical neurological syndrome in AChA infarctions, the first description is commonly attributed to Foix et al,10 who reported a patient with severe right hemiparesis, hypesthesia, and hemianopia due to an AChA infarct found at autopsy. Apart from studies in the 1950s investigating the therapeutic effect of surgical occlusion of the AChA on parkinsonism,7,11 the AChA did not receive much clinical attention until recently. Studies using modern imaging techniques not only confirmed the previously described neurological syndrome, but also reported a number of previously undescribed features such as neglect, language disorders, sectoranopia, and ataxic hemiparesis.9,12–15
The most recent series described 23 cases of hypo-
the
tic ataxic hemiparesis attributed to AChA infarc-
tions. This and a few other studies mapped the
AChA territory using the computed tomography
(CT) templates published by Damasio, which
appeared to include the periventricular corona radiata
in the territory of the AChA.

We used CT and magnetic resonance imaging
(MRI) scans available on 16 consecutively collected
personal cases to map the area of AChA infarction
on digitized axial tomographic templates. A special
computer program allowed the creation of overlay
diagrams that displayed variations and constant ter-
ritories in the AChA distribution.

Subjects and Methods

From 1985 to 1990 we collected 16 personal cases
(15 men, one woman, with median age 54 [range
18–73] years) with infarctions in the AChA territory.
Cases were accepted for inclusion if the AChA
occlusion was documented by angiography (eight
cases), was known to have occurred during surgical
procedures (two cases), or was found only by CT or
MRI scan (six cases) to involve at least some of the
territories included in the studies of Kolisko and
Beevor. In all cases, special effort was made to map
the lesion to determine if the corona radiata was also
affected. The films of the 38 other cases showing
involvement of the corona radiata found during this
period of time were also inspected to determine if
any of the territories included in the maps of Kolisko
and Beevor were also affected. All patients had a
neurological examination and a CT scan; six patients
had additional MRI studies. Fourteen patients had
vascular studies, including five patients with selective
angiography, six patients with extracranial and trans-
cranial Doppler tests, and three patients with both
ultrasound and angiography. Of the 16 strokes, three
occurred after embolization of an arteriovenous mal-
formation, and in two patients the AChA was clipped
during surgery (one for AChA aneurysm and one for
meningioma). AChA infarct was associated with an
internal carotid artery dissection in one and a cardiac
source of embolism in two (one patient with recent
myocardial infarction, one patient after cardiovas-
cular surgery). The cause of stroke was undetermined
in eight patients: three had hypertension, two had
diabetes, and one patient had both vascular risk
factors. None of the eight patients had a cardiac
history, and Doppler studies were normal in six. The
relatively large size of infarction in four of the eight
patients suggested AChA occlusion rather than small
penetrating branch disease.

The territory of infarction in the CT scan of each
case was mapped on digitized axial tomographic
templates derived from Matsui and Hirano’s atlas
using sections 8–13 at a 15° angle from the cantho-
meatal line. The data were entered into a MacIntosh
II computer via the commercially available graphic
software program IMAGE. The program provided
overlapping diagrams of the area of AChA infarction
in each of the six sections, with heavier shading
indicating more frequent involvement of the region.

Results

The distribution of the 16 AChA infarcts is dis-
played in the overlay diagrams (Figure 1). The poste-
rior limb of the internal capsule was consistently
involved in all cases, the medial portion of the palli-
dum in 75% of the cases, the cerebral peduncle and
the optic radiation in 44%, the lateral aspect of the
thalamus in 37% (superficially in five and more sub-
stantially in one case), and the medial temporal lobe
(hippocampus, uncus) in 37%. None of the infarcts
extended into the corona radiata. Coronal MRI views
of a large AChA infarction, which occurred during
embolization of a thalamic arteriovenous malforma-
tion, did not reveal extension into the brain adjacent
to the lateral ventricular wall (Figure 2).

All patients had motor deficits that were severe or
moderate in 69% and mild in 31%; a pure motor
syndrome occurred in 13%. Sensory deficits were less
frequent (56%) and usually mild or moderate; one
patient developed a Déjérine-Roussy syndrome. Two
patients with mild motor and sensory deficits dem-
onstrated ataxic symptoms. Visual field deficits were
found in 37% (five with homonymous hemianopia,
one with lower quadrantanopia, and one with supe-
rior sectoranopia). Six patients were dysarthric. One
patient with a left AChA infarct had initial motor
aphasia, which almost completely resolved within 1
month. One patient each had a transient contralat-
eral gaze preference, a ptosis and a Horner’s syn-
drome ipsilateral to the infarct, and transient
hemiballism.

Large lesions were regularly associated with severe
motor deficits, but small infarcts also caused hemi-
plegia in two patients (Figure 3). The presence and
degree of sensory abnormalities were associated with
neither the size of the infarct nor the severity of
weakness. Both patients with ataxic hemiparesis had
relatively small infarcts. Visual field defects were
seen only in large infarcts involving the optic radia-
tion; however, two patients whose CT scans sug-
gested involvement of the optic radiation had normal
visual fields. The MRI scan of one patient with
sectoranopia demonstrated involvement of the lat-
eral geniculate body.

Discussion

The territory of AChA infarction mapped in our
cohort is in good agreement with the AChA distribu-
tion as established by earlier workers who used autopsies or injection techniques in postmortem
specimens (Table 1). The posterior limb of the internal capsule (particularly the lower level), the
optic radiation, the medial aspect of the pallidum,
parts of the hippocampus and uncus, and superficial
areas of the thalamus appear to share a common
pattern of infarction, one that does not include the
corona radiata or the lateral ventricular wall. In only
one autopsy case did the AChA infarct extend to the
posterior lateral ventricular wall, sparing the tail of the caudate nucleus. However, involvement of the corona radiata was not mentioned. The autopsy case reported by Poppi was not included in Table 1 because this patient had an additional ipsilateral paramedian thalamic infarct. The pathological description of the case described by Abbie is too fragmentary to use for mapping.

Comparison of autopsies, pathological injection studies, and recent studies using neuroimaging demonstrates that the area involved in AChA infarction is usually smaller than the full territory inferred from experimental investigations (Table 1). The restricted infarct size may reflect the effects of collateral supply to the AChA territory via branches of the posterior cerebral, posterior communicating, and middle cerebral arteries. This collateral supply may explain the relatively small number of clinically obvious infarctions in patients who underwent surgical occlusion of the AChA as a treatment for parkinsonism. The unexpected occurrence of AChA infarcts and severe deficits as a result of embolization or surgical occlusion of the AChA in five of our patients probably reflects insufficient collateral blood flow in these cases.

In one recent series of 23 infarcts attributed to AChA occlusion, the lesions extended into the paraventricular white matter and the corona radiata in 65% of cases. Infarctions in this region were included following what appeared to be the vascular territories as depicted in H. Damasio's CT templates (C. Helgason, personal communication). The templates were assembled by Damasio from the diverse information available at the time (H. Damasio, personal communication). The maps were intended to show the dominant patterns of vascularization, and the author noted that some areas were especially problematic. The author did not consider the templates to indicate an extension of the AChA territory into the centrum semiovale, but rather into the
FIGURE 2. T2-weighted coronal magnetic resonance imaging (sequence a–d in anterior–posterior direction). Right thalamic arteriovenous malformation fed by lenticulostriate, thalamoperforant, and choroidal arteries (angiographic finding) and anterior choroidal infarct (arrows) after embolization affecting posterior limb of internal capsule, retrolenticular fibers, lateral geniculate body, and upper portion of medial temporal lobe, but not corona radiata.

region of transition between the internal capsule and centrum (H. Damasio, personal communication). The matter of the exact areas of supply appears not to have been settled, as none of the references in the original article described the course of the AChA along the upper ventricular wall. The number of patients with ataxic hemiparesis attributed to AChA infarctions in the series of Helgason and Wilbur contrasts with the relatively infrequent occurrence of ataxic symptoms in all the other studies. Decroix et al reported limb ataxia in one of 16 patients, and Ghika et al saw ataxic hemiparesis in 13% of 23 patients with AChA infarctions, the low frequency in agreement with that found in our patients. Apart from lesions in the pons, midbrain, and thalamus, 33 patients with ataxic hemiparesis and description of the sensory examination have been reported since 1980, excluding the cases of Helgason and

FIGURE 3. Axial computed tomography scans of typical anterior choroidal artery infarct in patient with severe pure motor syndrome. Area of infarction involves hippocampal gyms (panel A), lowest level of internal capsule and medial aspect of pallidum (panel B), and posterior limb of internal capsule, sparing the thalamus and putamen (panel C).
Table 1. Anterior Choroidal Artery Territory in Autopsies, Anatomical Injection Studies, and Radiological Series

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<th>CP</th>
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PLIC, posterior limb internal capsule; CP, cerebral peduncle; PAL, pallidum; OT, optic tract; MTL, medial temporal lobe; LGB, lateral geniculate body; THAL, thalamus; CD, caudate nucleus; LVW/COR, lateral ventricular wall/corona radiata; +, area involved; (+), minimal involvement; -, no involvement or area not mentioned; CT, computed tomography; MRI, magnetic resonance imaging.

*Putamen.

Wilbur. Of the 33 infarcts, 16 involved the posterior limb of the internal capsule (one attributed to AChA infarction) and 17 the paraventricular corona radiata (none attributed to the AChA). Sensory deficits were reported in 38% of lesions in the posterior limb of the internal capsule and in 53% of corona radiata infarcts, reflecting the more frequent involvement of thalamocortical sensory pathways in corona radiata lesions. Inclusion of the corona radiata probably explains the disproportionately high frequency of hemispheric ataxic hemiparesis among patients whose infarction was attributed to the AChA territory.

Inclusion of the corona radiata may also influence the etiologic classification of AChA infarction. In our cases, a possible source of embolism was found in three of our 11 patients with spontaneous AChA infarctions (one carotid and two cardiac sources). The size of the lesion suggested embolic AChA occlusion rather than small-vessel disease in four of the eight patients with negative cardiac history and normal Doppler studies. Decroix et al, who did not include corona radiata lesions, found moderate carotid stenoses ipsilateral to the AChA infarct in 44% of patients with additional atrial fibrillation in one patient and another patient had nonstenotic carotid plaques. In a series of 28 AChA infarctions, Sterbini et al reported possible cardiac or carotid embolism in 30% of cases. In contrast, a recent study investigating the etiology of AChA infarcts selected those lesions mapped referring to Damasio's templates and also included the corona radiata. Twenty of their patients had hypertension and 10 had diabetes. A potential source of emboli from the heart or the carotid system was identified in only 19% of 31 cases. The authors concluded that AChA infarcts usually result from small-vessel disease. It seems possible that these studies, including that of corona radiata lesions, will show a disproportionate increase in the frequency of typical small-vessel disease.

The contribution of the AChA to the supply of the thalamus is a matter of discussion. In six of our cases, the AChA infarcts appeared to involve the lateral thalamus; however, in all but one, involvement was superficial. In one patient, whose lesion extended deeper into the posterolateral thalamus, a severe Dejerine-Roussy syndrome was present. Kolisko found supply to the superior external portion of the thalamus in some cases, and Abbie reported that the AChA distribution frequently included the superficial part of the ventrolateral thalamus. Helgason and Wilbur detected involvement of the posterolateral thalamus in seven of their 23 cases. In contrast, Beevor did not demonstrate any supply from the AChA to the thalamus. Some investigators who focused on the vascularization of the thalamus also denied a relevant contribution of the AChA. In summary, although superficial involvement of the thalamus in AChA infarctions may occur, extension deep into the lateral thalamus must be considered exceptional.
Based on brain mapping of AChA infarcts, our findings suggest that infarcts in the corona radiata and brain adjacent to the lateral ventricular wall are not in the territory of the AChA. We suggest that clinical studies of AChA infarction should be limited to those regions originally documented by injection of brain specimens and by autopsy. Such studies may better clarify the etiology and syndromes attributed to AChA occlusions.

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