

# Anterior Choroidal Artery-Territory Infarction

## Report of Cases and Review

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● Occlusion of the anterior choroidal artery (AChA) can cause infarction in the posterior limb of the internal capsule. Infarction is less frequent in the thalamus, midbrain, temporal lobe, and lateral geniculate body territories of the AChA. The most common clinical sign is hemiparesis. Hemisensory loss is usually transient but may be severe at onset. Homonymous upper-quadrant anopia, hemianopia, or upper- and lower-quadrant sector anopsia can be present. A homonymous defect in the upper and lower visual fields sparing the horizontal meridian is probably diagnostic of a lesion in the lateral geniculate body in the territory of the AChA. The most common stroke mechanism is small-vessel occlusive disease, predominantly found in hypertensive and diabetic patients, but cardiac-origin embolism also can affect the AChA territory. Two of our patients had infarction after temporal lobe resection for epilepsy. Occasionally patients have associated disabilities of higher cortical function that are usually transient. The lesion should be recognizable by computed tomography.

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In 1925, Foix et al<sup>1</sup> described a patient at necropsy who had an infarct in the territory of the left anterior choroidal artery (AChA). This patient suddenly developed severe right hemiplegia, sensory loss

to all modalities in the right limbs, and a right hemianopia. There was no loss of consciousness, aphasia, or cortical function abnormality. Following this communication, there were few case reports of spontaneous AChA-territory infarction in the era before computed tomography (CT),<sup>2,7</sup> but the results of experience with ligation of the AChA for parkinsonism were recorded.<sup>8-11</sup> The nature of the occlusive arterial process was usually not clarified or emphasized. The clinical findings noted by Foix et al, such as hemiplegia, hemianesthesia, and hemianopia, were usually reaffirmed, but the syndrome proved variable and impossible clinically to identify accurately.

Computed tomography now allows recognition of AChA-territory infarction during life. Its advent has prompted several reports in the French<sup>12-15</sup> and English<sup>16</sup> literature of the resulting clinical syndrome and the localization of lesions on CT. We now report the cases of five patients with CT-documented AChA-territory infarction, one of whom had an unusual visual field defect that is probably diagnostic of infarction of the portion of the lateral geniculate body (LGB) supplied by the AChA. Two infarcts followed temporal lobe surgery for epilepsy. We have used these cases as a stimulus to review critically the anatomy, vascular abnormalities, and clinical features of this infrequently reported vascular lesion.

### REPORT OF CASES

**CASE 1.**—A 70-year-old hypertensive right-handed black woman, two weeks before hospital admission, noted dull aching pain in the left leg that lasted for three days. Four days before hospital admission,

a "left arm and leg problem" prevented use of these extremities. She could not walk alone and used her right arm to move the left. She thought her weakness had increased but was unable to describe the evolution of her symptoms. She denied sensory or visual symptoms.

At the time of hospital admission, neurologic findings included the following: lethargy, left visual neglect, constructional apraxia, diminished left corneal reflex, a slight left hemiparesis affecting the face and arm more than the leg, left Babinski's sign, and a moderate left hemisensory loss of pin, touch, and vibration senses. Position sense, stereognosis, and point localization were intact. The next day, the patient was more lethargic. On confrontation, a left homonymous hemianopia was found, although she had noticed no change in her vision. She now had a severe left-sided lower facial weakness, dysarthria, a flaccid immobile left arm, and an almost flaccid left leg with only a flicker of residual movement. The sensory deficit was now severe. She could not feel pin, deep pain, touch, or vibratory stimuli in her left limbs, but position sense was preserved.

During the next month, there was total resolution of her sensory loss and partial return of the visual field. The hemiparesis remained severe, with no arm movement, though she no longer neglected her left side. Laboratory tests revealed an old myocardial infarction by electrocardiogram and a normochromic normocytic anemia. Unenhanced CT showed a hypodense area in the right posterior limb of the internal capsule and LGB (Fig 1). The right AChA did not fill on cerebral angiography. Formal visual field examination initially (Fig 2, top) revealed a left homonymous hemianopia with sparing of a beaklike zone straddling the horizontal meridian. Later the left inferior quadrant had partially recovered, leaving an incongruous left-upper-quadrant defect (Fig 2, bottom). The horizontal border of the defect had gently sloping margins that indicated ongoing improvement.

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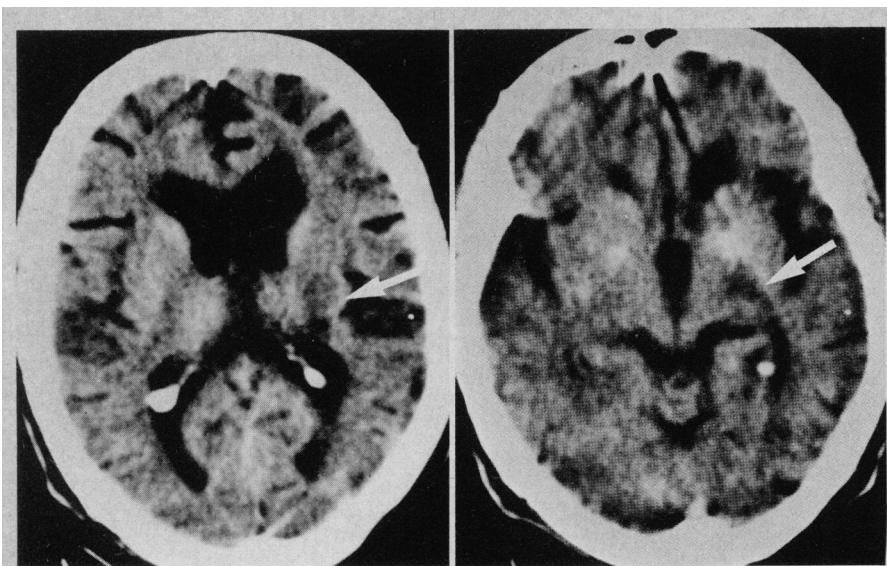


Fig 1.—Patient 1. Unenhanced computed tomographic scan showing lesion (arrows) in right posterior limb of internal capsule (left) and lateral geniculate body (right).

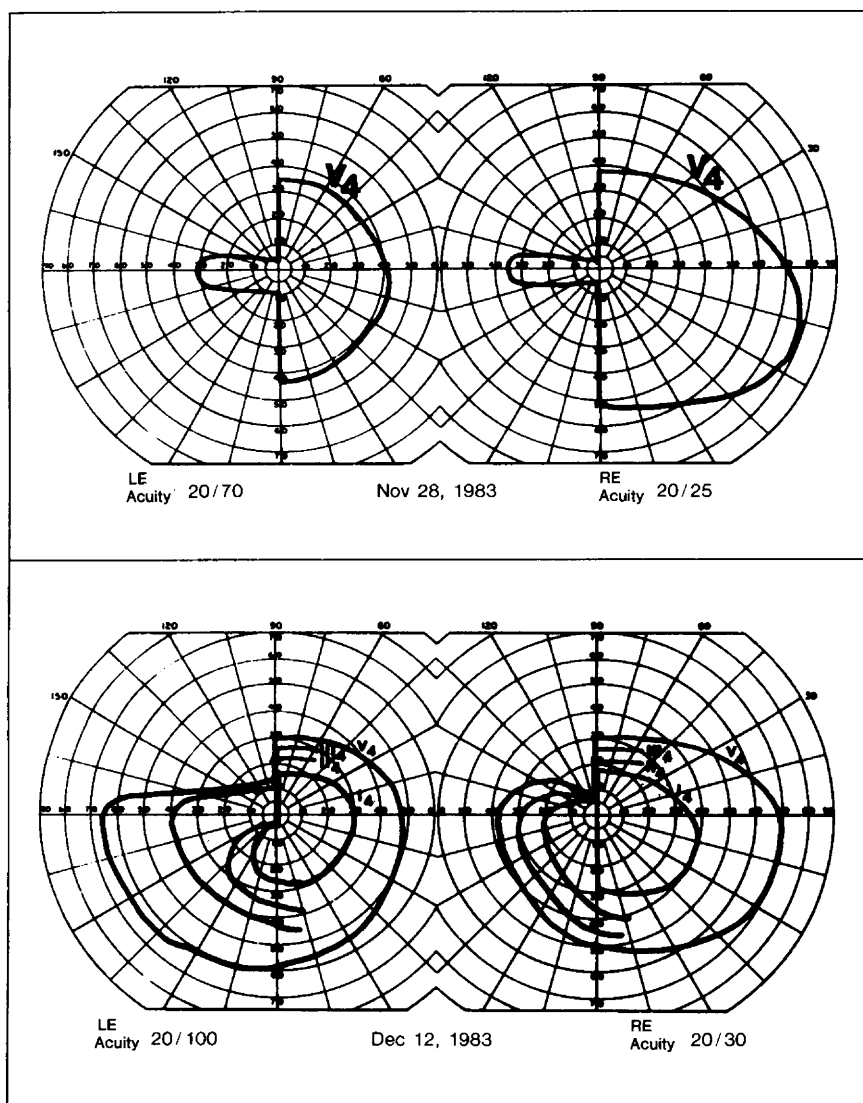


Fig 2.—Patient 1. Top, Initial visual field defect. Bottom, Later visual field defect.

**CASE 2.**—A 67-year-old white man had been in good health except for being overweight and addicted to cigarettes. After playing softball, he became inordinately sleepy. Although he arrived home in his car, he could not recall having crossed the Charles River (Boston). The next morning, he drove to work but could not find the entrance gate to work on the left side of the road. He realized that he could not see to his left. Later that day his right limbs tingled slightly.

When he was examined, his blood pressure was 128/70 mm Hg, but later rose to 180/110 mm Hg and remained high. There was a complete left homonymous hemianopia without macular sparing. He had left visual neglect. Motor strength was normal, but he held his left hand in a partially fisted posture as he walked. Pin sensation was less sharp on his left limbs, but he could localize objects well and correctly identified small objects placed in his left hand. Computed tomography showed a lucent region in the right optic tract near the temporal horn. Hemoglobin level was 14.2 g/dL (142 g/L), and hematocrit was 45% (0.45). Echocardiography and isotope angiograms were normal. A cerebral angiogram revealed minor irregularity at the origin of the right AChA but was otherwise normal. Vision gradually improved with sparing of the periphery of the left upper and lower quadrants. After two weeks, the patient could read and copy normally and no longer neglected the left visual space.

**CASE 3.**—A 77-year-old white man was found on the floor by his wife one morning. That day he seemed well, but the next morning he noted that his right limbs were weak and his voice was slurred. There was no history of cardiovascular disease. Blood pressure was 154/90 mm Hg. He was drowsy and dysarthric, but language use, repetition, and comprehension were normal. His head and eyes were deviated to the left. There was a severe right hemiplegia, complete except for a slight shrug of the right thigh. Primary sensory modality appreciation was normal and there was no extinction. There was a complete right homonymous hemianopia without paramedian sparing. Initial CT was normal, hemoglobin level was 16.5 g/dL (165 g/L), hematocrit was 49% (0.49), and platelet count was  $448\,000/\text{mm}^3$  ( $448 \times 10^9/\text{L}$ ). Blood glucose level ranged from 118 to 169 mg/dL (6.5 to 9.4 mmol/L).

Neurologic findings did not change, but the patient developed fever and pneumonia. Atrial fibrillation was noted, and repeated electrocardiograms and results of cardiac enzyme studies documented an acute myocardial infarct. Repeated CT showed a new lucency in the posterior limb of the left internal capsule and optic radiations in the territory of the left AChA.

**CASE 4.**—A 29-year-old black woman had had intractable complex partial seizures since childhood. She also carried the diagnosis of linear nevus sebaceum. A left temporal lobe resection in 1966 had not helped her condition. There was a left temporal and inferior parietal electroen-

cephalographic (EEG) spike focus. She underwent anterior temporal lobectomy and subpial resections of portions of the superior temporal gyrus, suprasylvian bank, and the supramarginal and angular gyri, eliminating spike discharges determined by EEG monitoring. Immediately postoperatively, a severe right hemiplegia was noted. No new visual field defect was detected, but she had had a right superior quadrantanopia since prior surgery. Sensory loss was not noted.

Follow-up examination showed marked but gradual improvement over the ensuing months, with only a mild right-sided hyperreflexia and distal hand weakness remaining. Postoperatively, CT showed a new lucency in the posterior limb of the internal capsule in the territory of the AChA not contiguous with the resection.

**CASE 5.**—A 29-year-old white woman had intractable complex partial seizures and underwent left temporal lobectomy because of a left temporal spike focus on EEG and preoperative magnetic resonance imaging changes in the left temporal lobe. Immediately after the operation, a severe right hemiplegia, right hemisensory loss, and complete right homonymous hemianopia without macular sparing were noted. She was transiently dysphasic with a mild anomia, some difficulty with comprehension, and preserved repetition. A dense homonymous hemianopia and right-sided hyperreflexia persisted through ten weeks of follow-up. Computed tomography showed a new lucency in the posterior limb of the internal capsule and lateral thalamus in the territory of the left AChA separate from the resection site (Fig 3).

#### COMMENT

The AChA is a small vessel (7 to 20 mm in diameter at its orifice)<sup>17</sup> that originates from the internal carotid artery 2 to 4 mm distal to the origin of the posterior communicating artery (PComMA).<sup>18</sup> It may originate from the proximal middle cerebral artery or the PComMA. The AChA courses posterolaterally as it lies at first inferior and lateral to the optic tract (Fig 4). It then courses to the medial side of the optic tract where the cerebral peduncle lies on its medial side and the mesial temporal lobe on its lateral side. The AChA terminates in the LGB and choroid plexus of the lateral ventricle, entering the plexus at the temporal horn and following it medially.

In its early, most anterior segment, the AChA supplies penetrating branches to the optic tract and to the medial segments of the globus pallidus. As the artery moves posteriorly, it gives off branches bilaterally to uncus, piriform cortex, posteromedial half of the amygdala, and the anterior hippocampus and dentate gyrus. Medial branches penetrate the cerebral peduncle supplying its middle

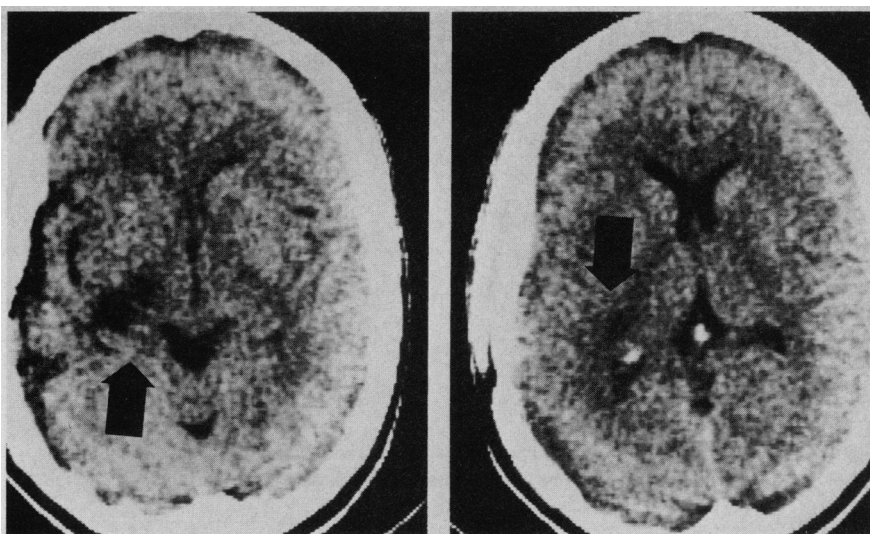


Fig 3.—Patient 5. Unenhanced computed tomographic scan showing lucency in posterior limb of internal capsule and lateral thalamus (arrows).

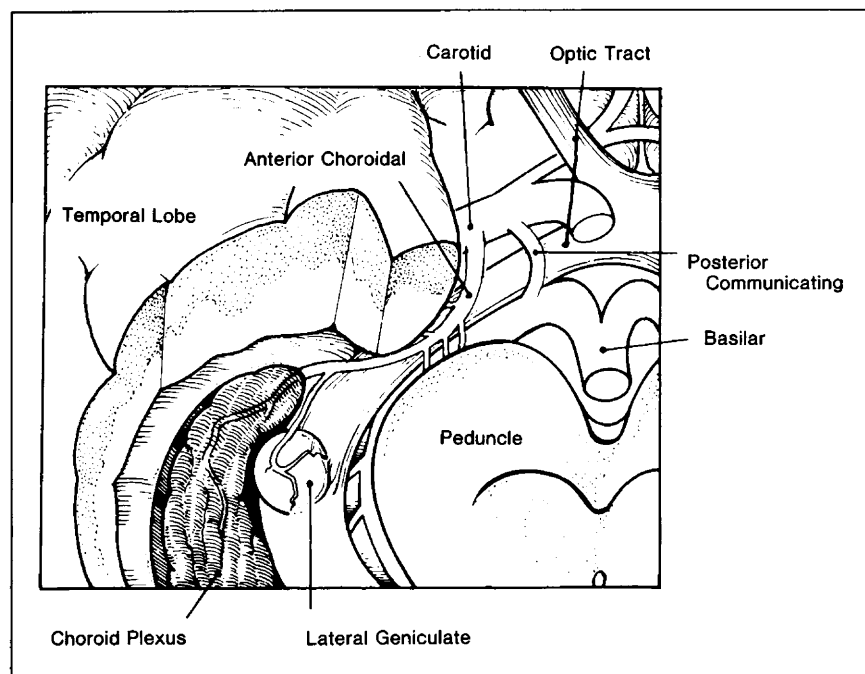


Fig 4.—Artist's depiction of vascular supply of anterior choroidal artery (patterned after Rhoton et al<sup>17</sup>).

third and extend variably to supply the substantia nigra, red nucleus, subthalamus, ventral anterior, ventral lateral, pulvinar, and reticular nuclei of the thalamus. The AChA also gives off penetrating branches at the level of the LGB that supply the posterior half of the posterior limb of the internal capsule, the tail of the caudate nucleus, and the retrolenticular fibers of the capsule, including the geniculocalcarine tract and some of the auditory radiations emanating from the medial geniculate body. The anterolateral half and hilum of the LGB are

supplied by the AChA, which then terminates in the choroid plexus. The most constant branches are those to the optic tract, cerebral peduncle, and choroid plexus<sup>17</sup>; other branches are more variable and anastomose with middle cerebral artery, PComMA, and posterior cerebral artery (PCA) branches. Anastomoses are usually rich but variable.<sup>17-21</sup> An intriguing and sometimes confusing feature of the AChA is that it irrigates regions in both the traditional anterior and posterior circulations.

Infarction of the posterior limb of

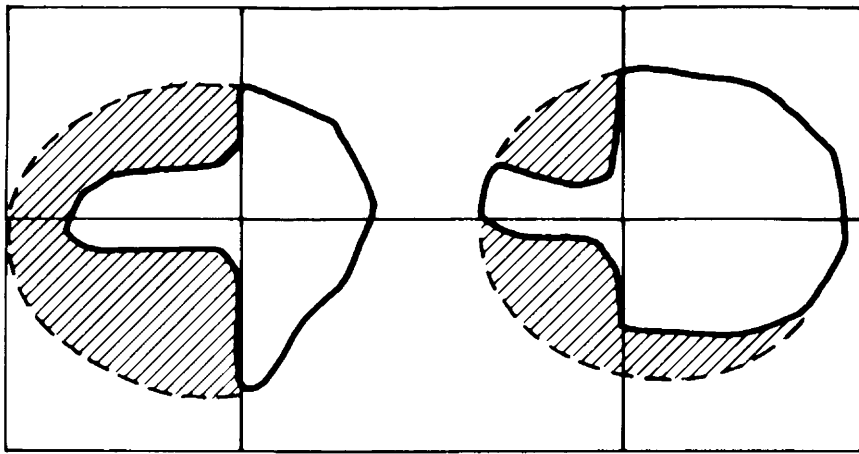


Fig 5.—Visual field defect of patient with anterior choroidal artery infarction described by Frisen.<sup>29</sup>

the internal capsule is found most consistently at necropsy.<sup>2,5,12</sup> Infarction of the optic tract, LGB, temporal lobe, thalamus, and midbrain is less common, possibly because of the plethora of rich collateral vessels. At times, the capsular infarcts are quite small and involve only penetrating branches of the AChA; these have usually been classified as lacunae.<sup>22</sup> Two reports concern patients with isolated, bilateral, AChA-territory, predominantly capsular infarcts.<sup>2,12</sup>

Few authors have provided data about the vascular lesion or stroke mechanism in AChA-territory infarction. Only 12 case reports could be identified that contained sufficient details to allow a presumptive diagnosis of stroke mechanism.<sup>2,5,7,12-16</sup> Four of these 12 patients probably had cardiogenic embolism (Ley,<sup>3</sup> Poppi,<sup>4</sup> and Masson et al<sup>14</sup> [cases 1 and 3]); one patient had an aneurysm of the supraclinoid internal carotid artery adjacent to the AChA origin.<sup>7</sup> The other seven patients had conditions predisposing them to small-artery occlusive disease and had no evidence of embolism or proximal large-artery occlusions: one young woman with syphilis had bilateral AChA infarcts,<sup>2</sup> three patients had hypertension,<sup>13,14</sup> one patient had diabetes,<sup>15</sup> and two patients had both hypertension and diabetes.<sup>12,16</sup> In our first two patients, the likely mechanism was intrinsic small-vessel disease. Both were hypertensive, and patient 2 had a high hematocrit and elevated blood glucose levels. Angiography revealed no proximal occlusive disease; patient 1 had occlusion of the AChA, while in patient 2 this vessel was small and irregularly narrowed. Patient 3 also had hypertension and elevated blood glucose levels, platelet count, and hemoglobin levels, indicating that he

may also have had intrinsic occlusive disease of the AChA. This patient, however, also had a possible cardiogenic embolic source (atrial fibrillation and recent myocardial infarction). As angiography was not done, the stroke mechanism is uncertain. Patients 4 and 5 developed strokes after temporal lobe resection, so that mechanical interference with the AChA or its branches was a likely mechanism.

Visual field changes are common after temporal lobectomy and are usually attributed directly to the resection.<sup>23</sup> Hemiplegia, hemisensory loss, and hemianopia have been noted after temporal lobe resection for epilepsy.<sup>24,25</sup> Penfield and colleagues<sup>25</sup> reported nine instances, all noted immediately postoperatively and attributed to capsular infarction due to intraoperative distortion of arteries within the anterior perforated substance, presumably the lenticulostriate middle cerebral artery branches. This was before CT, and there was no anatomic confirmation. Our cases 4 and 5 represent, to our knowledge, the first CT-studied cases of post-temporal lobectomy hemiplegia and document an AChA-territory lesion.

The onset of the neurologic symptoms has most often been sudden,<sup>1,4,7,12,14,15</sup> occasionally preceded by prolonged headache.<sup>7</sup> Our patients 1, 2, and 3 and the patient described by Ward et al<sup>16</sup> had a more gradual stepwise onset. The neurologic signs have been variable, but the most consistent and persistent abnormality has been hemiparesis. Curiously, ligation of the AChA seldom produced a serious lasting hemiplegia.<sup>8-11</sup> The AChA supplies the corticobulbar and corticospinal tracts in both the posterior limb of the internal capsule and the cerebral peduncle. Most often, as in our

patients, face, arm, and leg are involved. In a patient with bilateral AChA-territory infarcts, the resulting syndrome was an almost pure pseudobulbar palsy and asymmetric facial weakness without significant limb paresis.<sup>12</sup>

Hemisensory symptoms are variable. The sensory loss is usually incomplete and temporary. It may be an isolated finding.<sup>26</sup> The AChA supplies the sensory radiations within the posterior limb of the internal capsule and at the level of the ventral lateral nucleus of the thalamus. All modalities are usually affected, but our first patient had sparing of proprioception, as did the second patient of Pertuiset et al<sup>7</sup> and all of the patients of Graff-Radford et al.<sup>27</sup> Abbie<sup>2</sup> noted the frequent presence of a "thalamic"-like syndrome of painful paresthesias in the patients with AChA-territory infarction. Formication,<sup>7</sup> a feeling of swelling of the limbs,<sup>7,26</sup> or pain in the arms or legs<sup>4</sup> can be an early symptom, as it was in our first patient. Patient 2 had only a minor subjective decrease in pin and temperature sensibility on the involved side, and patient 3 had no detectable sensory abnormality. Most often the sensory signs and symptoms improve rapidly and seldom leave a severe residual sensory loss, as was the case in all of our subjects.

Hemianopia is probably the most variable feature of AChA-territory infarction. Among 12 patients in whom the AChA was ligated to treat Parkinson's disease, one had a transient homonymous hemianopia, and only one patient had persistent visual field defects, an upper quadrantanopia.<sup>11</sup> The AChA supplies visual fibers at three separate loci: the optic tract, the origin of the geniculocalcarine tract in the retrolenticular portion of the internal capsule, and the LGB. Pertuiset et al<sup>7</sup> postulated that optic tract infarction should cause an incongruent hemianopia associated with failure of pupillary reaction when light is shown from the hemianopic side (Wernicke's hemianopic pupillary reaction seen in pregeniculate lesions). We could not find a description of such findings in any patient with known AChA-territory infarction. Ischemia of the geniculocalcarine tract could cause a congruent homonymous hemianopia, possibly sparing the macula with a normal pupillary response.<sup>7</sup> This visual field defect is the most common visual abnormality found in patients with AChA territory infarction but is often transient because of the rich collater-

Symptoms and Signs in Five Cases of Anterior Choroidal Artery (AChA) Infarction

Patient	Maximal Symptoms and Signs	Onset	Angiogram	Computed Tomography	Mechanism
1	Lethargy, L visual neglect, anosognosia, constructional apraxia, quadruple sectoranopia, dysarthria, sensory loss to pin, touch, vibration with spared proprioception, severe L hemiplegia	Gradual, stepwise	No filling of R AChA	Hypodense area in R posterior limb of internal capsule and lateral geniculate	Intracranial small-vessel disease related to hypertension
2	Lethargy, L visual neglect, L complete homonymous hemianopia, minor subjective decrease in pin, temperature sense, L hand in fist	Gradual, stepwise	Mild irregularity at origin of R AChA	Lucency in right optic tract near temporal horn	Intracranial small-vessel disease related to hypertension, increased blood viscosity, diabetes
3	Drowsy, head and eye deviation to L, dysarthria, complete R homonymous hemianopia, normal sensory findings, severe R hemiplegia	Gradual, stepwise	No angiogram	Lucency in posterior limb of L internal capsule and optic radiations	Intracranial vessel disease related to hypertension, diabetes, hyperviscosity, or cardiac embolism
4	No new visual field defect, normal sensory findings, severe R hemiplegia	...	No angiogram	New lucency in posterior limb of L internal capsule	Intraoperative manipulation near AChA
5	Anomia, verbal comprehension deficit, preserved repetition, R hemisensory loss, severe R hemiplegia	...	No angiogram	New lucency in posterior limb of L internal capsule and lateral thalamus	Intraoperative manipulation near AChA

al supply of the geniculocalcarine tract from PCA branches. The AChA supplies the hilum and anterolateral half of the LGB. McKenzie's patient, described by Abbie,<sup>3</sup> had bilateral AChA infarction with dimming, especially in her upper visual fields. Our first patient also had a persistent upper quadrantanopia.

Frisen and colleagues,<sup>28,29</sup> in a series of two articles, clarified the nature of visual field defects found in patients with occlusion of branches forming the terminal supply of the LGB. The lateral choroidal artery, a branch of the PCA, supplies the medial and posterior segments of the nucleus, whereas the AChA enters inferiorly and laterally and supplies the hilum and anterolateral portion of the nucleus. The upper quadrant of the visual field is represented anterolaterally in the LGB while the lower quadrant is more anteromedially represented. The LGB is organized in projection columns that are oriented vertically in the nucleus and represent sectors of the visual field parallel to the horizontal meridian.<sup>28</sup> The lateral choroidal artery and AChA send branches to the nucleus parallel with the projection columns. In 1978, Frisen et al<sup>28</sup> described two patients with isolated horizontal sectorial defects probably caused by occlusion of the branches of the lateral choroidal artery. In 1979, Frisen<sup>29</sup> described the converse situation, ie, sparing of a horizontal sector in a patient whose visual field defect was caused by occlusion of the AChA during removal of a partially intraventricular meningioma. The latter patient's visual defect (Fig 5) corresponds exactly to that of our first patient's initial visual defect and was

named a quadruple sector anopia by Frisen because of homonymous congruous defects in both upper and lower quadrants of each eye. This finding is probably diagnostic of AChA-territory infarction, as is late atrophy of the retinal nerve fiber layer corresponding to the involved sectors.<sup>29</sup>

A superior quadrantanopia with macular sparing, similar to our first patient's late fields, was attributed to lateral geniculate infarction in other cases.<sup>2,3,7</sup> Patients 2, 3, and 5 had complete homonymous hemianopia.

The original patient of Foix et al<sup>1</sup> had no abnormalities of higher function. The absence of abnormalities of higher function despite hemiparesis, hemisensory loss, and hemianopia had traditionally been cited as diagnostic of a subcortical lesion and suggestive of AChA-territory infarction. In 1983, Cambier et al,<sup>13</sup> however, described four patients with CT-documented AChA-territory infarction with nondominant- or dominant-hemisphere syndromes, depending on the site of infarction. Visual neglect, constructional apraxia, anosognosia, and motor impersistence were found in three patients with right AChA-territory infarcts. These abnormalities tended to improve, as in our first two patients. The fourth patient had decreased language fluency, semantic paraphrastic errors, and speech perseveration. A single patient with CT-documented AChA-territory infarction had decreased speech fluency and paraphrastic errors despite preserved comprehension and repetition of spoken language.<sup>30</sup> The authors noted that the language deficit was similar to that described in patients with thalamic hemorrhage.<sup>30</sup> Graff-Rad-

ford et al<sup>27</sup> performed a detailed analysis of the neuropsychologic deficits in eight patients with CT-documented infarcts in the lateral thalamus and posterior limb of the internal capsule in the territory of the AChA. All had hemiparesis and two had abnormalities of the appropriate AChA corroborated angiographically. The patients with left thalamic lesions had dysarthria and slight language processing difficulties, eg, when making word associations or in comprehension of a written paragraph. Short-term verbal memory loss was also defective.<sup>27</sup> The patients with right thalamic lesions had defects in short-term visual memory.<sup>27</sup> To our knowledge, no patient with AChA-territory infarction and higher cortical function abnormalities has come to necropsy, so the extent of the lesion is uncertain. The cortical function signs could relate to a thalamic component or to a lesion of the temporal lobe or the temporal isthmus white matter.

Our patients 1 and 2 had striking but temporary left visual neglect, anosognosia, and constructional apraxia. Patient 5 had a transient aphasia. Patient 3 was drowsy, as was patient 1, and had rather persistent head and eye deviation to the side opposite the hemianopia. Conjugate eye deviation is a regular feature in patients with putaminal hemorrhage and large hemispheric infarcts but is not found in patients with pure motor strokes due to capsular infarction. Perhaps the far posterior localization or size of the capsular lesion might be a factor. No other patient in our series or previously described patient, to our knowledge, has had conjugate eye deviation.

Clinicopathologic correlation is least secure with reference to the mid-brain supply of the AChA. The patient of Poppi<sup>4</sup> had a plethora of abnormalities of eye movement but had necropsy evidence of paramedian lesions in the midbrain as well as infarction in the AChA territory, each likely caused by embolism of cardiac origin. The patient of Buge et al<sup>12</sup> had vertical gaze palsy likely of supranuclear type due to bilateral corticobulbar tract interruption and did not have a mid-brain lesion. A single patient

described by Viader et al<sup>15</sup> had diplopia and a loss of upward gaze in the left eye associated with hemiplegia and a hypodense lesion on CT in the internal capsule in the territory of the left AChA. It is not certain if there was a brain-stem component to the lesion.

On CT scan, the AChA supplies a discrete region in and lateral to the thalamus and just above the temporal horn and atrium of the lateral ventricles.<sup>14,31,32</sup> More widespread application of CT to patients who have had

strokes will undoubtedly lead to the recognition of more patients with AChA-territory infarction. Herein selected cases with abnormal CT findings were studied, and the AChA syndrome that emerges may not represent the full clinical spectrum of infarction in the territory of this vessel. (See the Table for a summary of our cases.) Careful study of these patients should help clarify the remaining clinicopathologic queries about AChA-territory infarction, especially the usual stroke mechanisms.

#### References

1. Foix C, Chavany H, Hillemand P, et al: Obliteration de l'artère choroïdienne antérieure: Ramollissement cerebral hémiplegie, hémianesthésie et hémianopsie. *Soc Ophthalmol* 1925; 27:221-223.
2. Abbie A: The clinical significance of the anterior choroidal artery. *Brain* 1933;56:233-246.
3. Abbie A: The blood supply of the lateral geniculate body with a note on the morphology of the choroidal arteries. *J Anat* 1933;67:491-527.
4. Poppi U: Sindrome talamo capsulare per ramollimento nel territorio dell'arteria coroidea anteriore. *Riv Patol Nerv Ment* 1928;33:505-542.
5. Ley J: Contribution à l'étude de ramollissement cerebral, envisagée au point de vue de la pathogenie de l'ictus apoplectique. *J Neurol Psychiatr* 1932;32:785-970.
6. Steegman A, Roberts D: The syndrome of the anterior choroidal artery: Report of a case. *JAMA* 1935;104:1695-1697.
7. Pertuiset B, Aron D, Dilenge D, et al: Les syndromes de l'artère choroïdienne antérieure: Etude clinique and radiologique. *Rev Neurol* 1962;106:286-294.
8. Cooper I: Surgical occlusion of the anterior choroidal artery in parkinsonism. *Surg Gynecol Obstet* 1955;99:207-219.
9. DuBois-Poulson A, Guillaume J, Magis C: La ligature de l'artère choroïdienne antérieure. *Bull Mem Soc Fr Ophthalmol* 1956;69:450-465.
10. Rand R, Brown W, Stern E: Surgical occlusion of anterior choroidal arteries in parkinsonism: Clinical and neuropathological findings. *Neurology* 1956;6:390-401.
11. Morillo A, Cooper I: Occlusion of the anterior choroidal artery. *Am J Ophthalmol* 1955;40:796-801.
12. Buge A, Escourrolle R, Hauw J, et al: Syndrome pseudobulbaire aigu par infarctus bilatéral limité du territoire des artères choroïdiennes antérieures. *Rev Neurol* 1979;135:313-318.
13. Cambier J, Gravelleau P, DeCroix J, et al: Le syndrome de l'artère choroïdienne antérieure: Etude neuropsychologique de 4 cas. *Rev Neurol* 1983;139:553-559.
14. Masson M, DeCroix J, Henin D, et al: Syndrome de l'artère choroïdienne antérieure: Etude clinique et tomodensitométrique de 4 cas. *Rev Neurol* 1983;139:547-552.
15. Viader F, Masson M, Marion M, et al: Infarctus cerebral dans le territoire de l'artère choroïdienne antérieure avec troubles oculomoteur. *Rev Neurol* 1984;140:668-670.
16. Ward T, Bernat J, Goldstein A: Occlusion of the anterior choroidal artery. *J Neurol Neurosurg Psychiatry* 1984;47:1048-1049.
17. Rhoton A, Kiyotaka F, Fradd B: Microsurgical anatomy of the anterior choroidal artery. *Surg Neurol* 1979;12:171-187.
18. Herman L, Fernando O, Gurdjian E: The anterior choroidal artery: An anatomical study of its area of distribution. *Anat Rec* 1966;154:95-102.
19. Stephens R, Stilwell D: *Arteries and Veins of the Human Brain*. Springfield, Ill, Charles C Thomas Publishers, 1969.
20. Percheron G: Les artères du thalamus humain: Les artères choroïdiennes. *Rev Neurol* 1977;133:547-548.
21. Yasargil M, Yonas H, Gasser J: Anterior choroidal artery aneurysms: Their anatomy and surgical significance. *Surg Neurol* 1978;9:129-138.
22. Rascol A, Clanet M, Manelfe C, et al: Pure motor hemiplegia: CT study of 30 cases. *Stroke* 1982;13:11-17.
23. Falconer M, Wilson J: Visual field changes following anterior temporal lobectomy: Their insignificance in relation to 'Meyers loop' of the optic radiation. *Brain* 1958;81:1-14.
24. Jensen I: Temporal lobe surgery around the world: Results, complications and mortality. *Acta Neurol Scand* 1975;52:354-373.
25. Penfield W, Lende R, Rasmussen T: Manipulation hemiplegia: An untoward complication in the surgery of focal epilepsy. *J Neurosurg* 1961;18:760-776.
26. Derouesne C, Yelnik A, Castaigne P: Deficit sensitif isole par infarctus dans le territoire de l'artère choroïdienne antérieure. *Rev Neurol* 1985;141:311-314.
27. Graff-Radford N, Damasio H, Yamada T, et al: Non-hemorrhagic thalamic infarction. *Brain* 1985;108:485-516.
28. Frisen L, Holmegaard L, Rosencrantz M: Sectorial optic atrophy and homonymous, horizontal sectoranopia: A lateral choroidal artery syndrome. *J Neurol Neurosurg Psychiatry* 1978;41:374-380.
29. Frisen L: Quadruple sectoranopia and sectorial optic atrophy: A syndrome of the distal anterior choroidal artery. *J Neurol Neurosurg Psychiatry* 1979;42:590-594.
30. Hommel M, DuBois F, Pollak P, et al: Syndrome de l'artère choroïdienne antérieure gauche avec troubles du langage et apraxie constructive. *Rev Neurol* 1985;141:137-142.
31. Damasio H: A computed tomographic guide to the identification of cerebral vascular territories. *Arch Neurol* 1983;40:138-142.
32. Takahashi S, Kawata Y, Uemura K: CT findings in anterior choroidal artery occlusion. *Rinsho Hoshasen* 1980;25:575-581.