Two Cases of Anterior Choroidal Artery Territory Infarction

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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 frequently observed in the thalamus, midbrain, temporal lobe, and lateral geniculate body (LGB) territories of the AChA. The most common clinical finding is hemiparesis. Hemianesthesia may be severe at onset but is usually transient. Homonymous hemianopia, upper-quadrant anopia, or upper-and lower-quadrant sector anopia can be present. Occasionally these patients are reported to have transient abnormalities of higher cortical function. The most common stroke mechanism is known to be small-vessel occlusive disease, predominantly found in hypertensive and diabetic patients. Vasospasm due to ruptured aneurysm or intraoperative mechanical manipulation, and cardiac origin the AChA territory. The infarct lesion is usually recognized and diagnosed by computed tomography. The best treatment is still unknown.

Key Words: Anterior choroidal artery, hemiparesis, hemianesthesia, hemianopia, cortical function

Foix et al., in 1925, described the syndrome of an infarct in the territory of the anterior choroidal artery (AChA) as characterized by contralateral hemiplegia, hemianesthesia, and homonymous hemianopia, although 2 cases had been reported by Kolisko in 1891 (cited by Decroix et al. 1986). Observations of subsequent authors, some of which included pathologic studies, tended to confirm the findings described by Foix et al..

We report two cases of AChA territory infarction that are recognized by computed tomography as a stimulus to review the anatomy, clinical features, stroke mechanism, and prognosis of this infrequently reported vascular lesion.

REPORT OF CASES

Case 1

A 57-year-old hypertensive right handed woman was found in bed by her family on the day of Feb. 1st 1989, at 1AM in the morning, with her right limbs

weak and voice slurred. There was no history of diabetes or cardiovascular diseases other than many years of hypertension which was left untreated. Blood pressure was 160/120mmHg. She was drowsy and dysarthric, but language use, repetition, and comprehension were normal. There was severe right hemiplegia equally involving the face, arm, and leg, and right Babinski sign. Moderate right hemisensory loss of pain, touch, position, and vibration senses were noted. On confrontation, a right homonymous hemianopia was found, but when we tried to perform further objective visual field tests, she could not do the taske due to her lethargic mental status and poor cooperation. The initial brain CT scan was normal. The hemoglobin level was 13.0g/dL and hematocrit was 38.6%. Blood glucose level was 110mg/dL, triglyceride was 78mg/dL, and HDL-chol was 35mg/dL. Serum VDRL was nonreactive. Electrocardiography revealed no abnormalities. During the next few days, there was an improvement in her mental status, but other neurologic deficits remained. Higher cortial function tests at that time revealed a depressed mood with preserved attention span, and her ability for caculation was severely impaired. Verbal memory functions were also shown to be severely impaired, but visual memory functions in contrast were very well preserved. Her reading capability was very much decreased as well as speech fluency.

A repeated brain CT scan showed a new lucency

Fig. 1. Brain CT scan showing hypodense area in the distribution of the left anterior choroidal artery (arrow). Both non-enhanced and enhanced scans of patient in case 1.
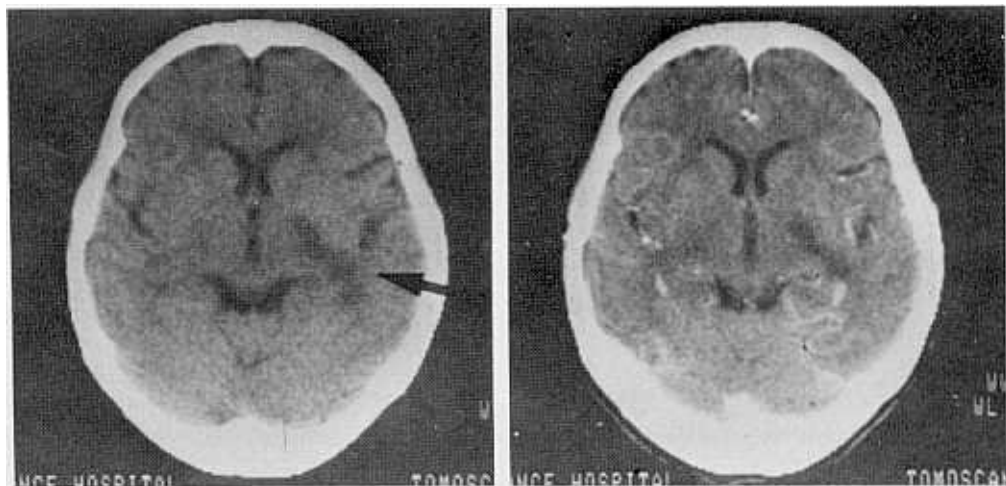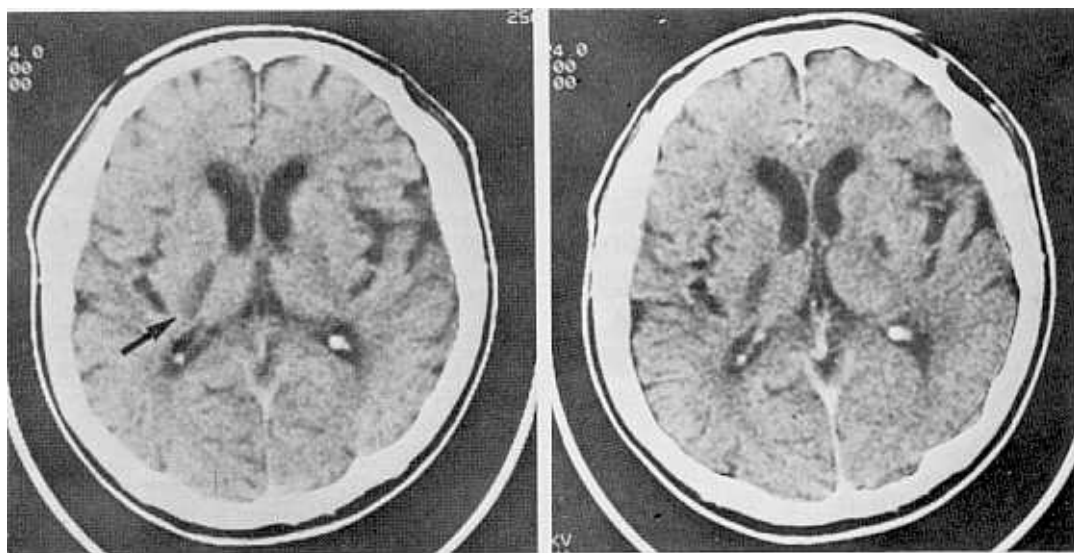

Fig. 2. Brain CT scan showing hypodense area in the distribution of the right anterior choroidal artery (arrow). Both non-enhanced and enhanced scans of patient in case 2.

in the posterior limb of the left internal capsule and optic radiation in the territory of the left AChA (Fig. 1).

Case 2

A 76-year-old right handed man, four weeks before admission, noted left side weakness which progressed to almost complete left hemiplegia within a matter of hours. He had a history of having a minor stroke 12 years ago, with symptoms of dysarthria and right hemifacial weakness which improved gradually. There was also a history of cardiac arrythmia for over 15 years without proper managment, but no history of diabetes or hypertension history were known.

At the time of hospital admission, neurological findings included the following: alert mental status, relatively intact mental functions, left hemifacial weakness, left hemifacial sensory changes on all modalities, almost complete left hemiplegia affecting both arm and leg, Babinski sign absent, and spared left sensory of pain, touch, and position senses except equivocally decreased vibration senses on the left lower extremity. There was a left homonymous hemianopia on confrontaton test. Blood pressure was 120/80, and heart sound was irregular but a murmur was not audible.

A brain CT scan showed multiple hypodense areas in the left basal ganglia which appeared to be the old lacunes, and in the posterior limb of the right internal capsule and in the territories of the right AChA, which is the more recent area of the infarction (Fig. 2). The hemoglobin level was 14.6g/dL and hematocrit was 43.5%. Blood glucose level was 100mg/dl, triglyceride level 123mg/dl, and HDL-chol level was 29mg/dl. Atrial fibrillation was noted on electrocardiography and an echocardiogram revealed a minimal degree of mitral regurgitation, but no evidence of thrombi or vegetation were detected. Serum VDRL was nonreactive.

DISCUSSION

The AChA is a small artery (0.7 to 2mm in diameter at its orifice) that originates from the internal carotid artery 2 to 4mm distal to the origin of the posterior communicating artery (Herman *et al.* 1966; Rhoton *et al.* 1979).

In its early, most anterior segment, the AChA supplies penetrating branches to the optic tract and to the medial segment of the globus pallidus. It then gives off branches to the uncus, piriform cortex, posteromedial half of the amygdala, and the anterior hippocampus and dentate gyrus. Medial branches penetrate the cerebral peduncle supplying its middle 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 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 lenticular fibers of the capsule, including the geniculocalcarine tract and some of the auditory radiation emanating from the medial geniculate body. The anterolateral half and hilum of LGB are supplied by the AChA, which then terminates in the choroidal plexus (Hegalson *et al.* 1986; Carpenter

et al. 1964). The most constantly supplied branches of the AChA are those to the optic tract, internal capsule posterior limb, cerebral peduncle, and choroidal plexus (Rhoton *et al.* 1979). As anastomosis exists between the AChA and the middle cerebral artery (MCA), posterior communicating artery, and posterior cerebral artery (PCA), an infarct of variable size and location may be produced when blood flow through the AChA itself or its penetrators is compromised (Hegalson, 1988).

Few authors have provided data about the vascular lesion or stroke mechanism in AChA infarction. Cardiogenic embolism, small artery occlusive disease, luetic arteritides, mechanical inteference after temporal lobectomy, aneurysm of the supraclinoid internal carotid artery adjacent to the AChA origin, embolus arising from carotid stenosis, compression of the AChA against the tentorium consequent to increased intracranial pressure, and intraoperative manipulation with subsequent direct vessel injury or vasospasm are the proposed mechanism in the reviewed literature (Decroix *et al.* 1986; Sterbini *et al.* 1987; Hegalson *et al.* 1986, 1987, 1988; Hegalson 1988; Fisher 1965, 1979; Jensen 1975; Cooper *et al.* 1955; Rand *et al.* 1956).

Our patient in case 1 had a long-standing history of hypertension without treatment, and the likely mechanism would be intrinsic small vessel disease. The patient in case 2 had a history of arrythmia, atrial fibrillation, and mitral regurgitation which suggests a possible cardiogenic source for embolism.

The onset of neurologic symptoms most often was sudden, occasionally preceded by prolonged headache. Both of our patients had sudden onset, almost complete hemiplegia, hemisensory changes, and homonymous hemianopia which are known to be the most common symptoms of the AChA territory infarction (Hegalson *et al.* 1986; Decroix *et al.* 1986). According to Derouesne *et al.* (1985), hemisensory symptoms are variable and sensory loss is usually incomplete and transient. Sometimes it may be an isolated finding. The AChA supplies sensory radiation within the posterior limb of the internal capsule and at the level of the ventral lateral nucleus of the thalamous. All modalities are usually affected, but sparing of proprioception is sometimes observed (Graff-Radford *et al.* 1985). Most often the sensory symptoms improve rapidly and seldom leave a severe residual sensory loss (Hegalson *et al.* 1986), as was the case in our second patient. The most consistent, and persistent symptom has been hemiparesis. The AChA supplies the corticobulbar and corticospinal tracts in both the posterior limb in the internal capsule and

the cerebral peduncle. In both of our patients, face, arm, and leg were involved as was the case in most of the patients in the reviewed literature (Decrotix et al. 1986; Healson et al. 1986; Steegmann et al. 1935; Ward et al. 1984). 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 (Hegalson et al. 1988).

Hemianopia is probably the most variable feature of the AChA territoriy infarction. The AChA supplies visual fibers at three different loci: the optic tract, the origin of the geniculocalcarine tract in the retrolenticular portion of the internal capsule, and the LGB. Frisen and colleagues, in a series of two articles, clarified the nature of the visual field defect found in patients with occlusion of branches forming the terminal supply of the LGB. In 1978, they described 2 patients with isolated horizontal sectorial defects probably caused by occlusions of the branches of the lateral posterior choroidal artery. In 1973, Frisen described the converse situation, which is sparing of a horizontal sector in a patient whose visual field defect was caused by occlusion of the AChA during the operation and this visual defect 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. Inschemia of the geniculocalcarine tract could cause a congruent homonymous hemianopia, possibly sparing the macula with a normal pupillary response. This visual field defect is the most common visual abnormality found in patients with AChA infarction but is often transient because of the rich collateral supply of the geniculocalarine tract from PCA branches. In 1986, Hegalson and colleagues, in their report of 5 cases of AChA territory infarction said that they could not find any report of cases describing a visual field defect caused by an infarction in the region of the optic tract.

In the article by Foix et al. (1925), his original patients had no higher cortical function abnormalities (cited by Hegalson et al. 1986) and absence of higher cortical function abnormalities had traditionally been cited as diagnostic of a subcortical lesion and suggestive of AChA territory infarction. However in 1983, Cambier et al. described 4 cases of CT documented AChA infarction with dominant or nondominant hemisphere syndromes, presumed to be due to affection of the auditory or visual radiations as well as the thalamoparietal projections. Visual neglect, con-

structional apraxia, anosognosia, motor impersistence, and defects in short term visual memory were found in patients with right AChA territory infarcts. Decreased language fluency, semantic paraphrasic errors, speech perseveration, dysarthria, short term verbal memory loss and slight language processing difficulties, e.g., when making word associations or in comprehension of a written paragraph, were also observed.

The prognosis of AChA territory infarct would seem dismal when bilateral strokes are considered. In the series of Hegalsoon (1988), of those who survived, five of nine patients with pseudobular mutism remained severely impaired and locked in up to a year after stroke. In the same period, four patients died. Of those with unilateral infarcts, there is good prognosis with mild impairment present months from stroke onset. Overall, it appears that visual field deficits and sensory loss improved but most often hemiparesis recovered little, if at all.

The best therapy for AChA territory infarct is unknown at present but may turn out to be different from that for other arterial territory ischemia. The subgroup of AChA infarct patients with advanced vasculopathy may benefit from intervention at the neuronal level of ischemic damage with excitatory amino acid inhibition or calcium channel blockers (Hegalson et al. 1986). As hypertension appears to be intimately related chronically or acutely, long-term as well as acute control of arterial blood pressure with lowering of the latter to levels not compromising autoregulation in the acute phase of the stroke might prevent further worsening of ischemic damage (Dutka et al. 1987). The use of anticoagulants is of uncertain utility, but would seem either useless or dangerous in the face of advanced small vessel disease as well as imprudent in those with accelerated hypertension (Hegalson et al. 1986). Surgical intervention such as the carotid endarterectomy would appear to play a minor role (Bruno et al. 1987).

In the brain CT scan of a patient with anterior choroidal territory infarction, the AChA supplies a discrete region in and lateral to the thalamus and just above the temporal horn and atrium of the lateral ventricles (Damasio 1983; Bruno et al. 1987; Graff-Radford et al. 1985).

In conclusion, we believe these are the two cases of patients with AChA territory infarctions on the basis of CT confirmation of the predicted anatomic lesion and the clinical findings. One of these patients had an abnormality of higher cortical functions which is an interesting observation in such cases. Both of our cases presented with the classical triad of hemiple-

gia, hemi-sensory changes, and homonymous hemianopia.

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