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CT of Ischemic Infarctions in the Territory of the Anterior Choroidal Artery: A Review of 28 Cases

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The purpose of the present study was to examine the incidence, causal factors, and anatomic localizations of infarction in the territory of the anterior choroidal artery. We studied 28 patients who had CT evidence of infarction in this territory. The affected structures taken into consideration were the posterior limb of the internal capsule, the retrolenticular portion of the internal capsule, the internal portion of the globus pallidus, and the lateral thalamus. Three conclusions are drawn: (1) that ischemic infarcts in the territory of the anterior choroidal artery are rare, representing 2.9% of all cerebral ischemic lesions in our material; (2) that the incidence of a possible embolic origin is significant; and (3) that the posterior two-thirds of the posterior limb of the internal capsule and the retrolenticular segment are the more frequently affected structures, and conversely, that the medial pallidus and the thalamus are less often involved.

The anterior choroidal artery usually arises from the internal carotid artery near the posterior communicating artery and the optic tract, although it is sometimes a branch of the middle cerebral or posterior communicating artery. However, variation in the origin of the artery does not produce any changes in its specific vascular territory. Two segments are differentiated by angiography: the basal cisternal segment and the intraventricular plexal segment [1]. The first, directed backward and laterally upward, ends in the temporal horn of the lateral ventricle; the latter is clearly in an upward and medial direction with a wide concave curve, ending near the choroidal plexus in the lower portion of the lateral ventricle (Fig. 1).

The anterior choroidal artery has numerous deep penetrating branches that supply a large somewhat variable territory of the telencephalon [2–6]: the posterior two-thirds of the posterior limb of the internal capsule, the genu and retrolenticular segment of the internal capsule with the optic and acoustic radiations, the medial portion of the globus pallidus, the lateral portion of the optic tract, the lateral half of the lateral geniculate body, the upper portion of the cerebral peduncle, the uncus, part of the amygdaloid nucleus, the anterior portions of the hippocampus, the anterior portions of the dentate fascia, the tail of the caudate nucleus, and the temporal portions of the choroid plexus (Fig. 2).

According to Percheron [7], the anterior choroidal artery cannot be considered an arterial source for thalamic vascularization, and the vascularization of this territory is only irregular and superficial. Anastomoses with branches of the posterior cerebral artery and posterior communicating artery often coexist.

The anterior choroidal artery syndrome was first described by Foix et al. in 1925 [8]. When complete, the syndrome includes hemiplegia, hemianesthesia, and homonymous lateral hemianopsia. Hemiplegia is caused by the interruption of the corticospinal fibers that cross the posterior half of the posterior limb of the internal capsule in the more caudal plane. The sensory deficit is due to the involvement of the thalamocortical radiations. The homonymous lateral hemianopsia may be caused by the lesion of the proximal geniculocalcarine tract and/or of the lateral geniculate body and/or of the optic tract. The neurologic picture may also be
associated with neuropsychological symptoms, including visual neglect, disorders of visuospatial strategy, constructional apraxia and anosognosia in the lesions of the minor hemisphere, and disorders of speech in dominant hemisphere lesions [9].

**Subjects and Methods**

This study includes 28 patients with CT evidence of infarction in the area of distribution of the anterior choroidal artery observed in the 2-year period between January 1983 and December 1984. Of these, 27 patients were examined by neurologists according to a protocol, and usual risk factors for cerebrovascular disease were taken into consideration.

Among the 28 patients in our study who had acceptable evidence of infarction in the territory of the anterior choroidal artery, 20 were hospitalized in a neurorehabilitation hospital and account for 2.9% of all 680 cerebral ischemic lesions found during the period 1983-1984.

The scans consisted of eight 10-mm thick sections, which extended from the base to the vertex of the skull. The internal capsule could be visualized in three slices. In some cases 5-mm thick slices were obtained. In selected cases the scan was repeated with contrast infusion.

The diagnosis of an ischemic lesion in the territory of the anterior choroidal artery was made according to the topographic criteria and clinical pictures mentioned above. The anatomic territories affected were carefully studied in spite of the difficulties in carrying out this task. Indeed, it is well known that although the inclination angle of the gantry remains constant (20°), standardization of the various layers is almost impossible. The differentiation of single territories supplied by the anterior choroidal artery was particularly difficult because of their limited volume and similar Hounsfield-unit values. According to the estimated time of the lesion the scans were divided into three groups: acute (within 10 days), subacute (between 11 and 30 days), and stable (more than 30 days).

To evaluate the extent of the lesion we considered only the anatomic structures that we believed to be the most detectable by CT in a horizontal plane [10]: the posterior limb and the retrolenticular portion of the internal capsule at midthalamic-pineal level, the internal capsule (posterior limb) at subthalamic level, the medial globus pallidus, and corresponding to plates 20° - 9 and 20° - 10 in the atlas of Matsui and Hirano [11]. Using their measurements, we divided the lesions into two groups: total infarctions and partial infarctions. Total infarctions were considered those that involved all the above-listed structures; partial infarctions were those that involved only a few of those structures. The latter group was subdivided into lacunar and nonlacunar infarctions; lacunar infarctions were considered only the smallest ones—less than 4 ml estimated volume, according to the criteria followed by Pullicino et al. [12]. The involvement of the thalamus and the coexistence with other ischemic lesions were also considered.

**Results**

Age, sex, and vascular risk factors are given in Table 1. One lesion was acute, 11 subacute, and 16 stable. The
hemispheres were equally affected: 14 lesions on the right, 14 on the left. Four patients had CT evidence of total infarction, seven patients showed ischemic lesions of the lacunar type, and the remaining 17 patients had partial nonlacunar infarctions (Figs. 3, 4, and 5).

The anatomic distribution of the lesions is summarized in Table 2. The single territories were involved as follows: the posterior limb of the internal capsule, including the anterior third (in addition to the four cases of total infarction) was entirely involved in four other cases; however, in one case a deep infarction in the territory of the middle cerebral artery was also present. Infarctions in the posterior two-thirds of the posterior limb of the internal capsule were seen in 10 cases. Lesions involving only the posterior third of the posterior limb were present in nine cases, and the middle third was only involved in one case. The retrolenticular portion of the internal capsule was involved in 16 cases. In this area the lesion was always associated with infarction in the posterior portion of the posterior limb: six cases with the entire posterior limb, eight cases with the posterior two-thirds, and two cases with the posterior one-third. The more caudal portion of the internal capsule (subthalamic level) was involved in 12 cases. This accounts for prominent caudal extension in the vertical plane, but hypodensity never extended upward to the corona radiata. The internal portion of the globus pallidus was involved in six cases, including the total infarctions. In five cases the lesion extended medially involving the most lateral portion of the thalamus. Fifteen patients had associated ischemic lesions, mostly located in the territory supplied by the middle cerebral artery.

**TABLE 1: Etiological Features in 28 Patients with Infarctions in the Anterior Choroidal Artery Territory**

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Mean</th>
<th>Range</th>
<th>Gender</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>63.1</td>
<td>34-81</td>
<td></td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Vascular risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>11</td>
<td>(40.7%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Possible embolic source</td>
<td>8</td>
<td>(29.6%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Possible carotid source</td>
<td>3</td>
<td>(11.1%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Possible cardiac source</td>
<td>5</td>
<td>(18.5%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>7</td>
<td>(25.9%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical transient ischemic attacks</td>
<td>2</td>
<td>(7.4%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>1</td>
<td>(3.7%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other etiology</td>
<td>1</td>
<td>(3.7%)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Data available for 27 patients.
* Infarction after intracranial ligation of posterior communicating aneurysm.

**Discussion**

This study confirms that ischemic infarctions in the territory of the anterior choroidal artery are rare. In this series of 28 cases, only 13 (46%) of the patients with infarction located in this territory had a single lesion without evidence of other ischemic lesions. Occlusion of the artery at its origin, involving the entire territory, was very rare: four cases (14%). On the
other hand, the most constantly affected site was the post-
erior portion of the posterior limb of the internal capsule sup-
plied by the deep penetrating arterioles.

It is interesting to note that in eight cases (one of these
cases was associated with a large infarct in the territory of
the middle cerebral artery), accounting for 28% of the 28
cases, the anterior third of the posterior limb of the internal
capsule was also involved. This finding supports the hypo-
thesis that the anterior choroidal artery may share in the vas-
cularization of this capsular segment, usually supplied by the
deep branches of the middle cerebral artery. A frequent finding
was the involvement of the retrolenticular portion (57%),
always associated with a lesion in the posterior third of the
posterior limb of the internal capsule.

The incidence of infarction involving the internal portion of
the globus pallidus was low (21%) and may suggest the pos-
sibility that the contribution of the anterior choroidal artery
in this area is not constant. The possible reasons for this low
incidence might be two. First, the lesion of the medial pallidus
is small and consequently undetected on CT scan. Second,
the medial pallidus may also be supplied by deep penetrating
branches of the lenticulostrate vessels. This view is sup-
ported by the anatomic study of Percheron and Escourroule
[13], who found in 96 cases of infarctions in the deep middle
cerebral territory four cases with involvement of the internal
portion of the globus pallidus. On the other hand, we have
observed numerous cases with CT evidence of striatocap-
sular infarctions involving the entire globus pallidus.

The limited involvement of the lateral thalamus confirms
also that the contribution of the anterior choroidal artery in
the thalamic blood supply is very poor and accounts for
individual variations.

Analyses of the concomitant vascular risk factors show the
high incidence of hypertension (40.7%), diabetes mellitus
(25.9%), and combined possible cardiac and carotid sources
of emboli (29.6%). The significant incidence of a possible
embolic source suggested that many of these cases had
embolic origins.

The present study was performed exclusively on CT and
substantiated by careful clinical examination. The vessel in-
volved was usually a penetrating artery, and its small diameter
did not permit visualization by angiography.

Pathologic confirmation is lacking; nevertheless, our data
seem to be reliable since they are based on well-defined
vascular topographic criteria and on retrospective clinical and
CT studies [14].

REFERENCES
2. Abbé AA. The clinical significance of the anterior choroidal artery. Brain
1933;56:233–246
Neurol Psychiatry 1954;71:714–722
4. Truex C, Carpenter M. Human neuroanatomy. Baltimore: Williams & Wil-
kins, 1969:71–72
5. Lazorthes G, Gouazé A, Salamon G. Vascularisation et circulation de
6. Rhoton AL, Fuji K, Frodd B. Microsurgical anatomy of the anterior choroidal
Rev Neurol (Paris) 1977;133:547–558
8. Foix Ch, Chavany JA, Hillemann P, Schiff-Wertheimer S. Oblitération de
l'artère choroidienne antérieure. Ramollissement cérébral, hémiplegie,
hémianesthésie et hémianopsie. Soc d'Ophthalmologie du 30 mai, 1925
de l'artère choroidienne antérieure. Etude neuropsychologique de 4 cas.
Rev Neurol (Paris) 1983;139:553–559
10. Damasio H. A computed tomographic guide to identification of cerebral
11. Matsui T, Hirano A. An Atlas of the human brain for computerized tomog-
12. Pullicino P, Nelson RF, Kendall BE, Marshall J. Small deep infarcts diag-
13. Percheron G, Escourroule R. Évaluation de la limite la plus médiale du
"territoire profond" de l'artère cérébrale moyenne sur 96 cas de ramollisse-
Syndrome de l'artère choroidienne antérieure. Etude clinique et tomoden-
sitométrique de 4 cas. Rev Neurol (Paris) 1983;139:547–552