Occurrence and Mechanisms of Occlusion of the Anterior Cerebral Artery

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SUMMARY Occlusion of the anterior cerebral artery is a rare condition. A review of CT scans from 413 patients with ischemic infarction confirmed this low relative incidence: only 3% of CT scans evidencing infarction involved the territory of the anterior cerebral artery.

Four major mechanisms of anterior cerebral artery occlusion have been identified in this series:
1. Emboli in unusual hemodynamic circumstances such as patients presumed to have increased flow through the anterior communicating artery because of unilateral internal carotid artery occlusion.
2. Propagation of thrombotic material from an occluded internal artery into the intracranial branches.
3. Spasm, emboli or propagating thrombosis associated with anterior communicating aneurysm.

Results

The distribution of infarctions seen on CT scan in 413 patients is listed in table 1. Eight of the 13 patients with CT evidence of infarction in the anterior cerebral artery territory had been subjected to angiography and presumed to be predominantly embolic. Indeed, this similarity provides a substantial contribution to the hypothesis that most intracranial arterial occlusions are of embolic origin and the pathogenesis of anterior cerebral artery occlusions have emerged from this review.

Material and Methods

The computed tomography (CT) scans and angiograms performed on patients with ischemic cerebrovascular disease were reviewed for the years 1979 to 1981. The correlation between the CT evidence of anterior cerebral artery (ACA) lesions and the cerebral angiographic evidence of ACA occlusive lesions was reviewed.

Clinical summaries of the patients in whom the cerebral angiograms detected an occlusion are described.

Case Reports

Case Report #1

A 54 year old presented with a history dating back nine months when he noted the sudden onset of decreased control of his left hand. During the subsequent two months he had several episodes consisting of numbness involving his left hand and fingers. Angiography demonstrated, at that time, stenosis of the supraclinoid portion of the internal carotid artery (ICA) on the right in addition to moderate stenosis of the carotid...
Table 1

<table>
<thead>
<tr>
<th>Distribution of Infarctions in the CT Material</th>
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<tbody>
<tr>
<td>ACA distribution</td>
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<tr>
<td>13 3.0%</td>
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<tr>
<td>MCA distribution</td>
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<tr>
<td>275 67.0%</td>
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<tr>
<td>total 39</td>
</tr>
<tr>
<td>parietal 144</td>
</tr>
<tr>
<td>frontal 57</td>
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<tr>
<td>parietal 87</td>
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<tr>
<td>bilateral 36</td>
</tr>
<tr>
<td>capsular 56</td>
</tr>
<tr>
<td>Small capsular and basal ganglia (&quot;lacunar&quot;)</td>
</tr>
<tr>
<td>56 8.4%</td>
</tr>
<tr>
<td>PCA distribution</td>
</tr>
<tr>
<td>59 14.1%</td>
</tr>
<tr>
<td>Cerebellar and brainstem</td>
</tr>
<tr>
<td>31 7.5%</td>
</tr>
<tr>
<td>Total 413 100.0%</td>
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The left ACA occlusion could reasonably be explained by an embolus which had originated from the left carotid bifurcation, and was directed into the ACA, presumably due to the increased flow along the proximal ACA due to the collateral cross-filling to the right hemisphere.

Case Report #2

A 45 year old hypertensive woman, a heavy smoker, presented with a one year history of a pounding noise in her right ear, and monthly episodes of left amaurosis fugax. Physical examination revealed an absence of a left common carotid artery pulse and a bruit over the right common carotid artery. There was no neurological deficit.

The CT scan was unremarkable. Cerebral angiography demonstrated occlusion of the left common carotid artery at its origin and a high grade stenosis of the right internal carotid artery. Collateral circulation to the left ICA territory was shown by the anterior communicating artery on the right carotid angiogram and by left ophthalmic artery, the external carotid artery being reconstituted from occipital branches shown on the left vertebral angiogram. Intracranially there was an occlusion of the right ACA distal to the horizontal segment with filling through leptomeningeal collaterals (fig. 2). The patient underwent right carotid endarterectomy with an uneventful postoperative course.

The ACA occlusion must have been the result of a similar pathophysiology as in case # 1: the right carotid bifurcation was an obvious source of emboli, and the increased flow through the anterior communicating pathway could direct these emboli into the ACA.

FIGURE 1. Left carotid angiogram. Early (A) and late (B) arterial phases: There is a cross flow through the anterior communicating artery to the right hemisphere (the right internal carotid artery is occluded); the left (!) anterior cerebral artery is occluded beyond the anterior communicating. It fills retrogradely through leptomeningeal collaterals (arrows) from the middle cerebral artery.
Case Report #3

A 65 year old male had his first transient episode of numbness of the left arm and leg seven years before the present assessment. Treatment for hypertension was initiated without further events until three months before admission. This was precipitated by ischemic left-sided arm, leg and face weakness with imperfect recovery. The CT scan revealed an infarction in the "border zone" between the right ACA and MCA.

Cerebral angiography showed occlusion of the right internal carotid artery with collateral circulation from the opposite side through the anterior communicating artery. Occlusion of the left pericallosal artery was revealed as well. In addition, the angiogram demonstrated a left subclavian artery occlusion and an ulcerated stenosis of the left internal carotid in the neck.

The ACA occlusion in this case was associated again with a carotid occlusion of the opposite side, and with a collateral circulation through the anterior segment of the Circle of Willis, which apparently directed emboli to the rare ACA site of embolic occlusion.

Case Report #4

This 49 year old man, a heavy smoker for 35 years, suffered a sudden onset of numbness and clumsiness of right arm eighteen months prior to admission. Angiography revealed an occluded left carotid artery and bilateral vertebral artery stenosis. His right vertebral artery was operated on in another hospital. Before the present admission he had an episode of relatively slow onset of right arm and leg weakness and facial drooping with early resolution. Examination revealed a right carotid bruit and a Horner’s syndrome. A diminished verbal memory skill was indicated by neuropsychological testing.

CT scan suggested an infarction in the territory of both the left MCA and ACA arteries (fig. 3A and 3B) the latter involving the distribution of the artery of Heubner as well. Cerebral angiography demonstrated an occluded left internal carotid artery with a 1 cm smooth stump. The collateral circulation was developed partly from the opposite side, where an ulcerated plaque was present in the ICA, and partly by reconstitution of the internal carotid artery from external carotid artery branches. In addition there was an occlusion of the ipsilateral ACA (fig. 3C).

Presumably the increased flow through the first segment of the anterior cerebral because of the collateral circulation is the predisposing factor for the ACA occlusion.

Case Report #5

A 54 year old right handed male, a heavy smoker, hypertensive and diabetic with advanced peripheral vascular disease for ten years, had an episode of speech difficulty followed by left sided weakness and numbness. Angiography done in another hospital detected a right ICA occlusion and a left ICA stenosis. He underwent left carotid endarterectomy. The residual left sided hemiparesis did not change. He remained well for six months and then he began to experience episodic sensory disturbance in the left face followed by a brief period of loss of consciousness with transient worsening of the left hemiparesis. On examination a left upper motor neuron facial paresis and mild paresis of the left arm were revealed.
FIGURE 3. Case #4. CT scan (A & B) and right carotid angiogram (C): On the CT scan there is low density area involving the medial aspect of the frontal lobe on the left side (arrow) as well as the head of the caudate nucleus and adjacent part of the internal capsule (small arrows). This low density extends to the middle cerebral artery territory. On the angiogram (C) there is collateral flow to the left side of the right carotid angiogram (the left carotid artery is occluded). There is absence of most left anterior cerebral distribution, confirmed on the later phases.

Angiography demonstrated a right ICA occlusion, with the occlusion extending intracranially into both ACA and MCA on the right, since both filled only through the leptomeningeal collaterals from the posterior cerebral artery (fig. 4).

Case Report #6

This 38 year old man with a strong family history of cardiovascular disease suffered a right sided weakness and expressive aphasia five years before the present admission. The symptoms improved significantly but he continued to have transient episodes of right sided weakness with worsening of the speech disorder.

Examination revealed a mild expressive aphasia, slightly impaired memory and emotional instability.

The CT scan demonstrated circumscribed prominent atrophy of both medial frontal lobes and the left fronto-temporal area. The occipital region appeared to be hypervascularized on the contrast scan. A dynamic
FIGURE 4. Case #5. Left common carotid angiogram (A) and left vertebral angiogram (B); late phase: These demonstrate collateral flow to the right cerebral hemisphere because the right ICA is occluded. There are only leptomeningeal collaterals (arrows) to both the right middle and anterior cerebral arteries mainly from the posterior cerebral artery, indicating that the carotid thrombosis on the right had propagated into the anterior and middle cerebral arteries proximally.

scan revealed a prolonged transit time in the territory of both left ACA and MCA. The prominent vascular structure in the occipital region filled with the arteries, and was suspected to reflect collateral pathways. Angiography disclosed stenosis of both supraclinoid ICA segment and an occlusion of both ACA and left MCA. Apart from anastomoses between the posterior and anterior cerebral arteries, a rich network of moyamoya type vessels represented the collateral circulation.

The ACA occlusion in this patient appears to have developed on the basis of local arterial pathology; the family history suggested arteriosclerosis despite its uncommon localization.

Case Report #7

A 35 year old chemist experienced the sudden onset of slurred speech. The next day he vomited, had a focal seizure and became aphasic with right hemiplegia. Seizures recurred and he slipped into a coma. The CSF was normal eight days after onset. The CT scan at that time disclosed bilateral paramedial frontal lobe infarcts (fig. 5). Angiography at that time demonstrated bilateral ACA occlusions.

Convalescence from this severe deficit was complicated by pulmonary emboli. An exhaustive investigation for causes of thrombosis or emboli revealed no abnormalities. The angiography was repeated and was unchanged.

The hemiparesis and the aphasia resolved but the patient remained inactive with poor memory and emotional instability.

No definite cause for the bilateral anterior cerebral artery occlusions were found. Therefore it is not known whether an embolic event had occurred, an anterior communicating aneurysm (which subsequently thrombosed along with both anterior cerebral arteries) or some other primary local arteriopathy existed.

Case Report #8

This 67 year old woman collapsed unconscious one evening, a week before admission to hospital. She regained consciousness after 15 minutes, complained of an occipital headache and went to sleep. The next day her discomfort was gone but her gait was unsteady and her husband found her to be mentally dull. The symptoms did not change during the week prior to her admission.

On examination some involuntary movements were seen in the right hand and a slight weakness of the right leg was noted. Her memory was judged slightly impaired and her reactions slowed. The CSF was clear with 30 mgm protein per 100 cc and no cells.

On the CT scan a low density non-enhancing area was found in the left side of the corpus callosum and in the medial frontal region on the left side (fig. 6A). To differentiate between a low grade glioma and an ischemic lesion, the CT scan was repeated six days later and prominent contrast enhancement was found in the previously identified lesion confirming infarction. An area of enhancement was demonstrated in the distal right anterior cerebral artery territory as well (fig. 6B & 6C).

Considering the relative rarity of the anterior cerebral localization of infarction, anterior communicating aneurysm was suspected. The latter was revealed by the subsequent angiogram, as well as multiple areas of spasm of both anterior cerebral arteries (fig. 6D & 6E).
FIGURE 5. Case #7. Noncontrast CT scan (A) and contrast scan (B): Infarction in the anterior cerebral artery territory on both sides is demonstrated. Cortical parts of the infarctions enhance (B).

Discussion

The incidence of the anterior cerebral artery infarction in Table 1 (3%) was similar to that found in the previous angiographic material of Gacs et al. The conclusion seems to be justified that this occurrence rate expresses a general biophysical rule related to blood flow resulting from the basic anatomic difference between the main intracranial arteries.

Based on the previous study it appears that this difference lies in the characteristics of the hemodynamics determined by the anatomy of the arterial tree. Experience with handling balloon catheters confirms this impression. The catheterization of an enlarged anterior cerebral artery feeding an arteriovenous malformation often requires special manipulation and contralateral carotid compression.

The first four cases reported in this material may represent a similar mechanism. In three, occlusion of the anterior cerebral artery was associated with contralateral and in one case with ipsilateral carotid occlusion; i.e. lesions which had apparently caused a substantial change in the flow pattern. In addition three of the four cases showed a lesion of the carotid bifurcation which could be regarded as a source of emboli. It would be hard to neglect this correlation and the explanation which logically follows: the collateral flow through the anterior communicating artery must have facilitated emboli to take this otherwise unusual pathway. One more fact supporting this opinion is that a majority of intracranial arterial occlusions in Caucasian patients are embolic in origin.

The other important cause of intracranial occlusions — frequent and well known in the middle cerebral artery — is the distal propagation of carotid thrombosis. Such a propagation into the middle cerebral artery may involve often the origin of the anterior cerebral artery as well but clinical symptoms can be expected only in case of anatomical variations of the Circle of Willis or the involvement of the artery of Heubner. Sometimes the thrombosis may propagate beyond the horizontal segment of the ACA causing clinically and angiographically recognizable anterior cerebral artery occlusion as in Case #5.

Local thrombosis of the intracranial arteries is usually regarded as a rarity. One of the rare vascular lesions, the moy-a-moya syndrome and other vasculitides of the Circle of Willis may involve the anterior cerebral arteries and is represented in our series by case #6. The etiology of this bilateral progressive intracranial occlusive lesion is probably a vasculitis except in those cases where arteriosclerosis occurs occasionally in this location. In Oriental patients an intracranial...
al location of arteriosclerosis seems to be more common than the extracranial site.

The relatively rare incidence of anterior cerebral artery occlusions may be a useful guide to investigation in considering the differential diagnosis of an infarction in the anterior cerebral artery territory. This proved to be the case for example in the patient in this series in whom the infarctions were found to be due to vasospasm related to subarachnoid hemorrhage.17

*Editorial Note: In accordance with Stroke policy, this article was edited by Associate Editor Dr. J.P. Mohr.

References

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