Middle Cerebral Artery Occlusion as a Cause of Isolated Subcortical Infarction

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SUMMARY We report two patients with large subcortical hemispheric infarctions, located in areas prone to the development of lacunes, who had occlusion of the middle cerebral artery demonstrated by arteriography. The cortical vessels were perfused by leptomeningeal collaterals. We suggest that large vessel arterial disease should be considered as a possible etiology of large subcortical infarctions and propose that the term lacune should not be used in cases in which neither the size nor the pathophysiologic mechanism of the lesion conform to C. M. Fisher's description.

Case Reports

Case 1

A 24 year old right-handed woman was admitted with progressive left-sided weakness of 12 hours duration. She did not have a history of hypertension, heart disease or diabetes mellitus. For a two year period she had used oral contraceptive pills but had stopped the drug three months before admission. One week before admission she had an episode of left-sided weakness, which completely resolved after several hours. For three days before admission she had continuous bifrontal, retro-orbital and occipital headaches, not relieved by aspirin. Her father had a stroke at age 47.

On admission, her blood pressure was 110/78 and her general examination was normal. She had a severe left hemiparesis which included the face. No visual field loss or sensory deficit were detected. Mental status and language function were normal. Extraocular movements and tongue and palate function were normal.

The computed tomography (CT) may be normal or show a small deep lesion. But recently, relatively large lesions of the basal ganglia, internal capsule and thalamus, in patients with unquestionable vascular disease have also been designated as "lacunar" infarctions, or distinguished by such terms as "super" lacunes or "giant" lacunes. It appears to us that these large infarctions may be consequent to pathologic processes different from those described by C. M. Fisher for lacunes and that the use of the now classical term may be questionable.

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For instance, preexisting hypertension is no longer an invariable finding. In addition, large vessel arterial stenosis and occlusion or cardiac sources of arterial embolism, are being increasingly described in patients with these larger infarctions.

To underline the importance of major vessel arterial occlusion as a cause of such alleged "lacunar" infarctions, we report two patients who sustained subcortical infarction involving the putamen, the anterior limb of the internal capsule and the caudate nucleus, as a result of middle cerebral artery occlusion at its stem.

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have a history of hypertension, diabetes mellitus or heart disease. Two days before admission he had several brief attacks of transient dysesthetic numbness of the right side of the body. On the evening before admission, he developed a mild headache and right-sided weakness and numbness, which included the face.

On admission, his blood pressure was 110/80 and his general examination was normal. He was alert but had a non-fluent aphasia with paraphasic errors, perseveration and dysarthria. Naming, repetition, aural comprehension, reading and writing were impaired. He had a right flaccid hemiplegia which involved the face and mild right hemihypesthesia. Visual fields were normal. The eyes were conjugate and in the midline at rest. Eye movements were full. Palate and tongue function were normal.

Chest roentgenogram and electrocardiogram were
normal. Urinalysis and hematologic, serologic and clotting studies were normal. An echocardiogram demonstrated left atrial enlargement but no clot was seen. CT showed an area of low attenuation in the left hemisphere which extended over four consecutive cuts (fig. 4). It involved the head of the caudate nucleus, anterior limb of the internal capsule and putamen. It occupied the area supplied by the lenticulostriate arteries. The cuts were 8 mm apart and Figure 4 is the second from the bottom cut in which the lesion was visualized. Arteriography demonstrated occlusion of the first portion of the left middle cerebral artery. The supraclinoid portion of the left internal carotid artery was narrow and the lenticulostriate arteries were not visualized (fig. 5A and 5B). The cortical branches of the left middle cerebral artery filled via leptomeningeal collaterals from the anterior and posterior cerebral arteries (fig. 6).

He was treated with antiplatelet aggregating agents. He made modest improvement during hospitalization, prominent paralysis and language deficits were present at the time of discharge.

Discussion

Both patients had non-hemorrhagic infarctions in a site which is a common location for lacunes. Nevertheless, the size of these infarctions as gleaned from CT, is not, in our view, compatible with the concept of lacune, as articulated by C.M. Fisher. Other authors

Figure 5A and 5B. Lateral and anterioposterior views of a left carotid arteriogram in Case 2 display complete occlusion of the first segment of the left middle cerebral artery.

Figure 6. Subsequent lateral view of the same arteriogram in Case 2 demonstrates filling of the cortical branches of the left middle cerebral artery from branches of the left anterior cerebral artery.
have commented similarly.11, 15 Our contention is that not only do such infarctions not conform to the classical concept but also that their underlying pathophysiology may be different from the one proposed for lacunes. While we do not know if thrombosis developed upon an existing localized lesion of the middle cerebral artery, it is interesting to note that both patients were young adults without hypertension or obvious reason for premature atherosclerosis. For instance, it is reasonable to assume the subcortical infarction in these cases is due to a different process than the small vessel arterial disease that results in lacunar infarction.

CT in these two cases shows large low density areas in the basal ganglia. The lesions involve the anterior limb of the internal capsule and adjacent portions of the head of the caudate and putamen. In the first patient, the right hemisphere lesion extends over three consecutive cuts (10 mm apart); while in the second patient, the left hemisphere lesion extends over four consecutive cuts (8 mm apart). In both patients, the infarctions are restricted to areas supplied by the lenticulostriate arteries. Preservation of the lower portion of the head of the caudate points toward normal perfusion through Heubner’s branch of the anterior cerebral artery. The lack of CT changes in the posterior limb of the internal capsules and medial aspect of the globus pallidus suggests that the anterior choroidal artery is not implicated.16 The size and site of the subcortical infarction prompts consideration of underlying middle cerebral artery disease.

The results of arteriography in these two cases support the concept that large vessel arterial disease can cause subcortical infarction in the territories where lacunes are commonly encountered. In both of our cases occlusion of the middle cerebral artery was the cause of the infarctions. This arteriographic finding has been described previously.9, 10 The arteriograms demonstrated retrograde filling of the cortical branches of the middle cerebral artery and that appears to have limited infarction to the area supplied by the lenticulostriate arteries for which no collateral circulation was available. C. M. Fisher described a case of a large subcortical infarction involving the putamen, anterior limb of the internal capsule and caudate nucleus as a result of occlusion of the origin of the lenticulostriate arteries brought about by an atherosclerotic plaque of the middle cerebral artery.7 The mechanism of infarction in our cases would be similar, but caused by a complete occlusion of the proximal middle cerebral artery.

Although the lesions in both cases were larger than the size of lesions traditionally described as “lacunar” infarctions, one of the patients had the classic syndrome of pure motor hemiparesis. Recently, Rascol et al14 described the hemispheric sites related to pure motor hemiparesis based on CT findings. The most common was a semilunar shaped infarction which involved the anterior limb of the internal capsule and adjacent portions of the putamen and caudate nucleus, a lesion similar to the one seen in our first patient.

The second patient had right hemiparesis and aphasia. CT showed an infarction of the head of the caudate nucleus, anterior limb of the internal capsule and putamen which is the mirror image of the abnormality seen in the first patient and was consistent with the type I infarction described by Rascol et al.14 Recently, aphasia has been described in association with deep non-hemorrhagic infarctions that involve the left basal ganglia and the anterior limb of the internal capsule.17, 18 There has been pathologic confirmation that basal ganglia infarction can cause aphasia in the absence of cortical lesions.19 Furthermore, although not inconsistent with true lacunar infarction, the presence of aphasia is unusual in association with lacunes.

The term lacune has become widely used to describe almost any subcortical infarction discovered on CT regardless of the size and site of the lesion and irrespective of clinical presentation. The definition of lacune used by the Harvard Cooperative Stroke Registry included specific clinical criteria as well as the requirement for a normal arteriogram, and for a CT that was either normal or demonstrated a small lesion.20 Using the term “lacune” as a blanket description for any subcortical hemispheric infarction, even if placed in the lacune prone territory, is confusing and it may also lead to erroneous etiologic diagnosis. That in turn might lead to an incomplete evaluation which could adversely affect treatment. We propose that such deep, large lesions found on CT should be given the descriptive term of “subcortical hemispheric infarctions” and that the term lacune should be restricted to those lesions that meet the clinical, CT and arteriographic criteria indicated above.

References

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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.

ISOLATED OCCLUSION of the anterior cerebral artery is rare. This lesion, being uncommon, has provided an opportunity for some elegant clinical and pathological reports. The reasons for this infrequency of anterior cerebral artery occlusions has not attracted much comment; studies have focused upon the vascular territories and variations of the anterior cerebral artery and on the symptomatology of its occlusion. No significant difference, pathologically, of involvement of the anterior and middle cerebral arteries by an atheromatous process has been suggested to explain the different incidence of these occlusions. On the other hand, a striking similarity was found by Gacs and co-workers between the distribution of occlusions in various cerebral arteries and the statistical regularity of the pathway of balloons used in superselective angiography. "Balloon emboli" occurred in the anterior cerebral artery in the same proportion to the middle cerebral artery as did spontaneous occlusions observed by 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.

A review of the clinical and radiological case material of University Hospital, London, Ontario, was carried out to confirm the validity of this distribution pattern. Additional facts concerning the occurrence 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.

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 occlusion of the anterior cerebral artery was found in 5. In another 3 patients, angiograms revealed an ACA occlusion without CT evidence of infarction in its territory.

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
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