Intracerebral Hemorrhage Caused by Transmural Dissection of the Anterior Cerebral Artery

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**Background and Purpose:** Spontaneous dissection of the intracranial carotid artery or its main branches is an unusual condition.

**Case Description:** A 72-year-old hypertensive woman after an intense nuchal rigidity showed a subarachnoid hemorrhage and an interhemispheric hematoma by computed tomography. The neuropathological study revealed a transmural dissection of the pericallosal artery.

**Conclusions:** The authors suggest that the dissection origin is an atheromatous plaque that bleeds, producing a dissection plane from the lumen to the adventitial artery. *(Stroke. 1993;24:1400-1402.)*

**KEY WORDS** • aneurysm, dissecting • dissection • subarachnoid hemorrhage

Spontaneous dissection of the intracranial carotid artery and its main branches is an unusual cause of ischemic neurological deficits. In exceptional cases, the dissection may produce a subarachnoid hemorrhage as in the patient described here, who had an anterior cerebral artery dissection within an atheromatous plaque.

**Case Report**

A 72-year-old woman, known to have hypertension, developed over several minutes an acute, severe headache and neck pain with loss of consciousness. On examination at the hospital her blood pressure was 170/110 mm Hg. She was by then alert and responded to commands promptly. She had intense nuchal rigidity and right hemiparesis. Cephalocaudal reflex testing showed bilateral flexion. The remainder of the general examination was normal. Computed tomography revealed a massive basal subarachnoid hemorrhage and interhemispheric hematoma. The blood extended from the genu to the splenium of the corpus callosum (Fig 1).

An angiographic study was entirely normal. The anterior cerebral arteries near the genu of the corpus callosum were of normal caliber, and no aneurysms were seen. Over the following 10 days the patient’s hemiparesis gradually improved. She was treated for 3 weeks with intravenous nimodipine, at a dose of 60 mg every 4 hours. The patient died suddenly 6 weeks later.

**Neuropathology**

A postmortem study of the brain showed a subarachnoid hemorrhage extending from the genu to the splenium of the corpus callosum and a marked atherosclerosis of the vessels of the circle of Willis. A large hematoma was found at the origin of the pericallosal artery; a hemispheric infarction in the distribution of the left anterior cerebral artery was also found. No aneurysms were seen, and the remaining cerebral vessels were normal (Fig 2). Microscopy showed a transmural dissection plane extending from the lumen through the internal elastic lamina and media to the adventitia. The adventitia surface and media of the anterior cerebral artery were infiltrated by polymorphonuclear leukocytes. There was atheromatous plaque in the arterial dissection, showing a recent hemorrhage. The origin of the dissection was identified in the distal segment where the hematoma communicated freely with the lumen through a large internal elastic tear (Fig 3). No aneurysms were found on dissection of the circle of Willis and its major branches or in a study of the affected vessels by sections at 3-mm intervals. The immediate cause of death was massive and recent gastric bleeding caused by an acute erosive gastritis.

**Discussion**

Spontaneous dissection of the intracranial carotid artery or its major branches is a rare condition, although it probably occurs at a greater frequency than the literature suggests. The clinical presentation is an explosive headache and a contralateral deficit. The patients frequently have a decreasing level of consciousness or coma. Ischemic infarct is the most frequent clinical occurrence produced by the dissection.1,2 The embolic mechanism is the main cause of the infarct in extracranial carotid dissection, but in intracranial dissection the hypoperfusion caused by obstruction is usually the cause.1,3,4

The mean age of patients with intracranial carotid dissection is between 20 and 30 years.3,5 There are various probable predisposing causes of the dissection, such as atherosclerosis,3 fibromuscular dysplasia, cystic...
medial necrosis, and moyamoya disease; in the absence of a primary vasculopathy, a vessel wall weakening due to a congenital gap defect in the internal elastic lamina has been described. There have been reports of chronic migraine and trauma, although they are of doubtful significance. The mechanisms responsible for extracranial and intracranial arterial dissections may be different because there are important basic anatomic variations in the structure of the intracranial and extracranial arterial walls. The intracranial arteries have a much thinner media, no external elastic lamina, and a thin adventitia or vasa vasorum. Dissection of the intracranial vessels usually involves subintimal rather than intramedial or subadventitial layers. There are very few reports in the literature of dissection of intracranial vessels leading to subarachnoid hemorrhage. The majority of such dissections involved the posterior circulation. Tanaka et al reported in a review that only 14% of the dissections causing subarachnoid hemorrhage were located in the anterior circulation. This preponderance strongly suggests that local anatomic factors could have a role in determining the transmural extent of the dissection. We agree with the opinion of Farrell et al regarding subarachnoid hemorrhage as the primary manifestation of intracranial dissection and the cleavage plane extending transmurally from the lumen.

FIG 1. Computed tomogram showing a massive interhemispheric hematoma and a subarachnoid hemorrhage.

FIG 2. Coronal section of the cerebral hemispheres showing a supracallosal hematoma and a hemispheric infarction in the distribution of the anterior cerebral artery.

FIG 3. Pericallosal artery showing an atheromatous plaque and an intraplaque hemorrhage dissecting the wall. There is a pericallosal hematoma in the reabsorption phase (Masson, original magnification ×40).
to the adventitia of the artery. This finding contrasts with previous reports in which the dissection plane in patients with subarachnoid hemorrhage was within the media or adventitia.  

The angiography study was normal, demonstrating the vessel reparation. The subsequent evolution of the transmural extent of the dissection is determined by several factors including systolic blood pressure, vessel location, and local anatomic factors. It is unlikely that hemorrhage could cause extrinsic compression and partial obliteration of a high-pressure intracranial vessel such as the anterior cerebral artery.  

We suggest that the dissection has its origin in an atheromatous plaque that bleeds, producing a transmural extension, with hypertension and age as other factors that lead to damage to the arterial wall. To our knowledge, the only other reported case of anterior cerebral artery dissection is that of a 26-year-old hypertensive woman, published by Gherardi and Lee.  

Our case may be the first report showing a complete dissection of the arterial wall from an atheromatous plaque and a subarachnoid hemorrhage. Although the natural history of the disease is far from being understood, it can be said that not all cases are fatal.  

Friedman and Drake first recognized the benign evolution of an intracranial dissection without a neurological deficit.

References

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Stroke. 1993;24:1400-1402
doi: 10.1161/01.STR.24.9.1400
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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