Embolism from intracranial aneurysms

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Embolism from an aneurysm is one of the mechanisms involved in the pathogenesis of ischemic symptoms associated with intracranial aneurysms. Four cases are reported in which aneurysms of the internal carotid arteries and middle cerebral arteries were the source of emboli resulting in cerebral infarction. In the treatment of these aneurysms, it is best to clip the neck of the aneurysm with great care to avoid embolism due to extrusion of clot into the distal artery.

Key Words • cerebral embolism • aneurysm • infarction

An embolus from an aneurysm may narrow or obstruct a cerebral artery transiently or permanently and cause neurological symptoms of ischemic origin. Taptas and Katsiotis presented such a possibility in 1968. These authors suggested that a cause of hemiplegia after subarachnoid hemorrhage might be arterial embolism from the aneurysm. Only eight cases of cerebral embolism in intracranial aneurysms have been published since then. In this paper we report four new cases and discuss the method of treatment of these aneurysms.

Case Reports

Case 1

This 35-year-old woman was admitted to our service in February, 1977. Two months prior to admission she suffered an episode of weakness of the left extremities that cleared within 12 hours. Similar attacks recurred 4 weeks and 1 week before admission. General physical examination was unremarkable. A lumbar puncture performed on admission gave normal results. Right carotid angiography showed a large unruptured aneurysm at the junction of the internal carotid artery and the posterior communicating artery (Fig. 1).

A right frontotemporal craniotomy was performed, and a large aneurysm, measuring $2.5 \times 1.5 \times 1.5$ cm, was seen to extend into the right temporal lobe. The neck of the aneurysm was clipped, and the sac was excised. In the lumen of the aneurysm there was a large clot that was partially fresh and partially organized (Fig. 1).

The postoperative course was benign, and there has been no recurrence of hemiparetic attacks.

Case 2

This 46-year-old woman was admitted in April, 1978. Two days before admission she noticed a transient episode of right-sided weakness. Six hours prior to admission she suddenly developed right hemiparesis and speech disturbance which prompted hospitalization. On admission her blood pressure was 145/95 mm Hg, and the physical examination was unremarkable. The patient was drowsy and had a marked right hemiparesis, more pronounced in the upper extremity. She had hyperreflexia and a Babinski sign on the right. She made frequent errors on naming objects. Two days later a computerized tomography (CT) brain scan showed a large low-density area in the anterior part of the region fed by the left middle cerebral artery (Fig. 2). Left carotid angiography showed a large, partially opacified aneurysm at the trifurcation of the left middle cerebral artery, with occlusion of the main branches of the artery (Fig. 3).

A left frontotemporal craniotomy was performed and an aneurysm, $2 \times 1.5 \times 1.5$ cm in size, was seen. The neck was broad and hard, and, because of this, the proximal and distal artery of the aneurysm was clipped temporarily, and the aneurysm sac was incised. Large clots filled the lumen of the aneurysm and branches of the middle cerebral artery. Following clipping of the aneurysm neck, the superficial temporal artery was anastomosed to the cortical branches of the middle cerebral artery.
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Postoperative left carotid angiography showed that the aneurysm clip was placed correctly, and the anastomoses between the superficial temporal artery and the branches of the middle cerebral artery were patent (Fig. 3). Two months after the operation, the patient's speech disturbance had cleared and the right hemiparesis was improving.

Case 3

This 52-year-old man presented with a 3-month history of frequent episodes of right-sided blindness. For 1 month before admission these episodes increased in frequency and at times were accompanied by left hemiparesis which cleared in about 10 minutes. He was admitted to our service in June, 1978. General physical and neurological examinations were unremarkable. A CT brain scan showed no abnormal findings. Right carotid angiography demonstrated a fusiform aneurysm in the cavernous portion of the right internal carotid artery (Fig. 4). It was considered that his symptom was caused by microemboli from this aneurysm.

A right frontotemporal craniotomy was performed, and the aneurysm was trapped by the ligation of the internal carotid artery intracranially and in the neck. Anastomosis between the superficial temporal artery and cortical branches of the right middle cerebral artery was then performed.

The postoperative course was benign, and amaurotic episodes of the right eye did not recur. Postoperative right carotid angiography showed that the enlarged superficial temporal artery filled all branches of the right middle cerebral artery (Fig. 4).
Case 4

This 57-year-old man was admitted in August, 1979. Three days prior to admission he had suddenly developed a speech disturbance that prompted hospitalization. General physical examination was unremarkable. The patient made frequent errors on naming, reading, and verbal memory tests on admission. A CT brain scan showed a small low-density area in the left parietal region (Fig. 5). Left carotid angiography demonstrated a saccular aneurysm at the trifurcation of the left middle cerebral artery (Fig. 5).

Fig. 1. Case 2. Left: Preoperative left carotid angiogram showing a large, partially opacified aneurysm at the trifurcation of the left middle cerebral artery, and occlusions of the main branches. Right: Postoperative left carotid angiogram showing that the aneurysm clip is well placed, and the anastomoses between the superficial temporal artery and the branches of the middle cerebral artery are patent.

Fig. 4. Case 3. Left: Right carotid angiogram showing a fusiform aneurysm in the cavernous portion of the internal carotid artery. Right: Right carotid angiogram performed 3 months after operation showing that the enlarged superficial temporal artery fills all branches of the middle cerebral artery.
A left frontotemporal craniotomy was performed, and a large atheromatous plaque was seen at the trifurcation of the middle cerebral artery, extending into the neck of the aneurysm. Because of this, it was thought that immediate clipping of the aneurysm neck might be hazardous. The proximal and distal segments of the middle cerebral artery were occluded temporarily, and the sac of the aneurysm was incised. Many small clots and an atheromatous plaque in the lumen of the aneurysm were evacuated, following which the neck of the aneurysm was easily clipped. The postoperative course was benign, and the speech disturbance cleared 4 months later.

Discussion

Cerebral aneurysms present varied clinical symptoms. They may rupture, causing subarachnoid hemorrhage (SAH), or present with focal signs due to localized pressure. Cerebral ischemia and infarction contribute to the morbidity and mortality of patients with intracranial aneurysms. The pathogenesis of infarction remains obscure. Based on autopsy studies, Crompton noted that cerebral infarction was found in 75% of 159 patients who died of ruptured aneurysm.

Since the report by Ecker and Riemenschneider, vasospasm has been accepted as a major etiological factor in producing cerebral infarction. Taptas and Katsiotis reported the case of a patient with SAH who developed ischemic symptoms 15 days after hemorrhage. An angiographic filling defect of the internal carotid artery was interpreted as a thrombus extending from an aneurysm. Subsequent reports of cerebral aneurysm with embolic infarction have appeared. Recently, Hoffman, et al., reported the case of a 60-year-old woman who had nine episodes of transient weakness, clumsiness, and hypesthesia of the right upper extremity over a 1-year period. Angiography revealed normal extracranial vessels and a left middle cerebral artery aneurysm. The aneurysm sac removed at operation contained an organized thrombus.

In our Case 1, there were three attacks of left hemiparesis before operation. These disappeared completely after operation, and histological examination of the excised aneurysm revealed various stages of thrombi, new and old, within the aneurysm cavity. We assumed that these thrombi resulted in symptomatic emboli. In Case 2, the aneurysm was cut open at operation, and a new thrombus was found filling the cavity, protruding into a distal artery. In Case 3, frequent right amaurosis fugax had occurred preoperatively, and transient ischemic attacks of the right hemisphere developed. These symptoms disappeared after operation. The symptoms of Case 3 could well be assumed to have been caused by microemboli from thrombi in the aneurysm. In Case 4, the aneurysm was opened at operation and many thrombi were found within the aneurysm cavity. In
this case, the cerebrospinal fluid on admission was clear and free of blood, and the CT scan showed no evidence of hemorrhage, although an area of infarction was noted in a region distal to the aneurysm. After operation for the aneurysm, the patient's condition improved, and it is reasonable to suspect that symptoms were caused by an embolism induced by thrombi within the aneurysm.

Angiographic evidence of cerebral arterial occlusion was confirmed only in Case 2. However, even absence of such demonstration does not negate the diagnosis of embolism, because clots in blood vessels can be lysed. 6

Antunes and Correll 3 have pointed out that excessive manipulation of aneurysms increases the risk of embolism. They recommended muslin gauze wrapping or coating with a plastic substance in cases with arteriosclerotic plaques on the side of a cerebral aneurysm or on the parent vessel wall. However, a patient reported by Mount and Antunes 9 died of encephalomalacia caused by embolism from a muscle-wrapped cerebral aneurysm, which suggested that coating or wrapping aneurysms may not be sufficient to prevent embolism. We believe that careful clipping of the aneurysm neck is the best treatment. If there appears to be a possibility of producing a fresh embolus by displacement of intra-aneurysmal clot in the course of neck clipping, one should consider temporary occlusion of blood flow proximally and distally, as was done in Cases 2 and 4. After opening the aneurysmal sac and removing clot as well as the arteriosclerotic plaque at the neck of the aneurysm, neck clipping can then be carried out with less risk. In patients such as Cases 2 and 3, where distal arterial occlusion was found or where a direct operation to eliminate the aneurysm was impossible and ligation of the internal carotid artery was necessary, a good result may be expected from anastomosis of the superficial temporal artery to the cortical branch of the middle cerebral artery.

References

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