CLINICAL STUDY

# Small aneurysms as a cause of thromboembolic stroke

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**Abstract**

***Objective and importance:*** **A small percentage of patients with intracranial aneurysms present with embolic stroke distal to the site of the aneurysm. Thromboembolism typically occurs in large or giant aneurysms where reduction of flow within the aneurysm is thought to increase the possibility of clot formation. Only a few examples are available in the literature of patients with smaller aneurysms who develop embolic infarction distal to the lesion. We have experience with two such patients with an apparent common pathophysiology.**
***Clinical presentation:*** **Patient 1 with a distal left middle cerebral artery infarct was found to have an 18 mm carotid artery bifurcation aneurysm (patient age 49 years). Patient 2 had a 7 mm right middle cerebral artery aneurysm with a small distal embolus (patient age 65 years). At surgery both patients were found to have atherosclerotic disease involving the aneurysm base and parent vessel. In each instance, the aneurysm was opened during temporary vessel occlusion and microendarterectomy was performed. Occlusion of one of the major arterial branches exiting the aneurysm was also present with anterior cerebral artery occlusion in the case of ICA bifurcation lesion and MCA branch occlusion in the case of the MCA aneurysm. Both patients made a good recovery following surgery.**
***Conclusion:*** **In small aneurysms with atherosclerotic disease distal thromboembolism may occur. Surgical treatment with microendarterectomy is appropriate to prevent further emboli and potential for subarachnoid hemorrhage.** ***(Fig. 5, Ref. 16.)***
**Key words: aneurysm, cerebral embolus, stroke, atherosclerosis.**

Most intracranial aneurysms are discovered as a result of subarachnoid hemorrhage. Unruptured lesions may be found incidentally or may produce symptoms secondary to mass effect, either on cranial nerves or brain parenchyma, with or without seizures. Occasionally, large or giant aneurysms can produce an embolus with resultant stroke. It is felt that large or giant aneurysms which are partially thrombosed have the capacity to cause embolus. Thromboembolism caused by small-sized aneurysms is a rare event and only few cases are reported in the literature. We review this literature and present two patients who presented to Massachusetts General Hospital with cerebral infarction due to thromboembolism caused by small aneurysms. The surgical management of these two patients involved aneurysmorrhaphy with microendarterectomy of the parent vessel (surgeon CSO). These techniques are reviewed.

## Case reports

*Case 1*

This 65 year old female with a history of diabetes mellitus was in her usual state of health until two weeks prior to addmission when she suffered a small right parietal stroke with resultant mild left hemiparesis. She was recovering her strength until three days prior to addmission when she experienced several episodes of difficulty with fine motor movement of the left hand. An MRI/MRA study was performed which demonstrated a 7 mm partially thrombosed middle cerebral artery bifurcation aneurysm on the right side (Fig. 1a). An angiogram confirmed this lesion (Fig. 1b). In addition, there were areas of small subacute infarction in the right frontal and temporal region. Computerized tomography angiography (CTA) suggested an irregular border at the lumen of the neck of the aneurysm (Fig. 1c). She had normal duplex ultrasound evaluation of her carotid arteries and normal

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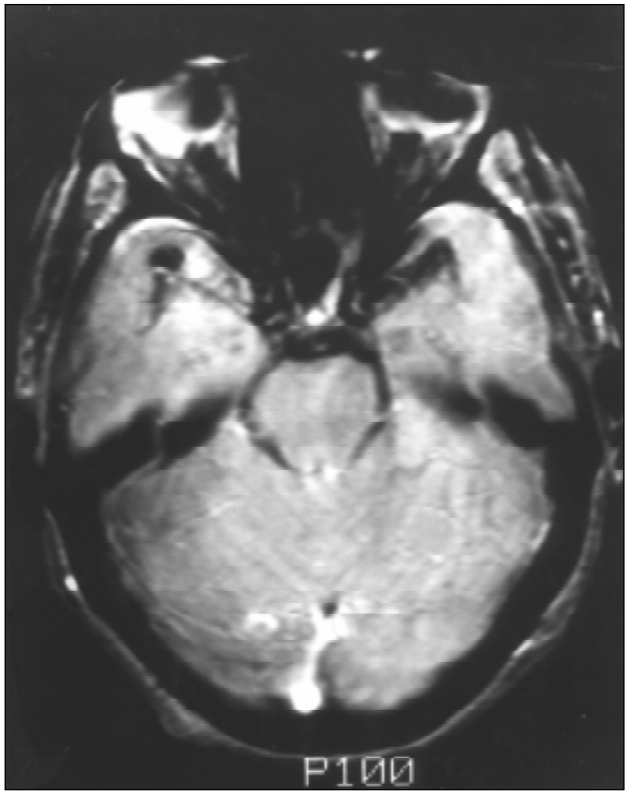


**Fig. 1a. An MRI/MRA study demonstrated a 7 mm partially thrombosed middle cerebral artery bifurcation aneurysm on the right side.**

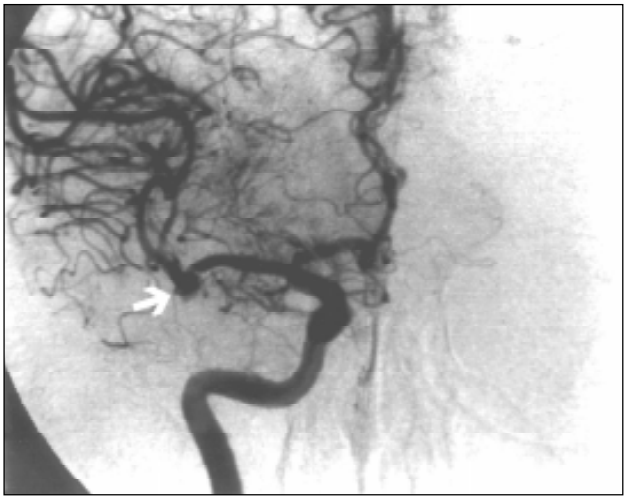

**Fig. 1b. An angiogram confirmed a middle cerebral artery bifurcation aneurysm on the right side.**

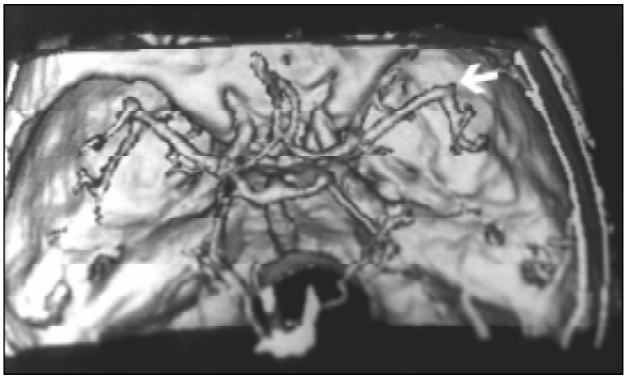

**Fig. 1c. Computerized tomography angiography (CTA) suggested an irregular border at the lumen of the neck of the aneurysm.**

complex cardiological examination including EKG, echocardiogram or 24-hour holter monitoring.

A standard frontotemporal craniotomy was used to approach the right middle cerebral artery. The sylvian fissure was split widely to give complete visualization of the middle cerebral artery trunk (M1 segment) and middle cerebral artery branches (M2 segment). The superior division of the middle cerebral artery appeared to be thrombosed and a loose piece of atherosclerotic plaque was visible through the wall of the M1 segment. The patient had been cooled to 32.5 °C for cerebral protection. 100 g of intravenous mannitol was given and we proceeded with temporary clip occlusion of the proximal M1 and distal M2 segments. Systolic blood pressure was elevated to 170 mmHg to maximize collateral blood supply to the area. The aneurysm was incised and a straight dissector was used to performe a microendarterectomy. Atherosclerotic plaque was removed from the aneurysm as well as from the M1 segment. Attempts at removing thrombus of plaque from the occluded superior division were unsuccessful. Using a microvascular doppler probe, it was possible to document excellent collateral flow in the M3 branches distal to the area of occlusion of the superior division. After removal of plaque from the aneurysm then the neck was clipped to reconstruct a smooth contour from the M1 to the inferior division of the M2 branch. A second 5 mm clip was placed on the stump of the thrombosed superior division of the M2 segment trunk.

The patient awoke from anesthesia without any new neurological deficit. Two ischemic events in the postoperative period are probably linked to the followup angiography. During this follow-up study the patient developed left-sided weakness of the arm and leg which persisted for approximately 20 minutes and then resolved. A follow-up CT scan demonstrated a new medial temporal area of infarction. Seven days after the angiogram she had another event with left hemiparesis and visual field deficit and was readmitted. She was placed on heparin and her symptoms resolved completely. Over a year of observation after surgery the patient had no further TIAs or areas of infarction, and has made a complete recovery.

*Case 2*

This 49 year old male with a history of hypertension and myocardial infarction was well until four months prior to addmission when he developed a left hemispheric stroke with aphasia, slight right hand weakness, and a facial drop. An MRI and DSA study were obtained at that time which demonstrated a left

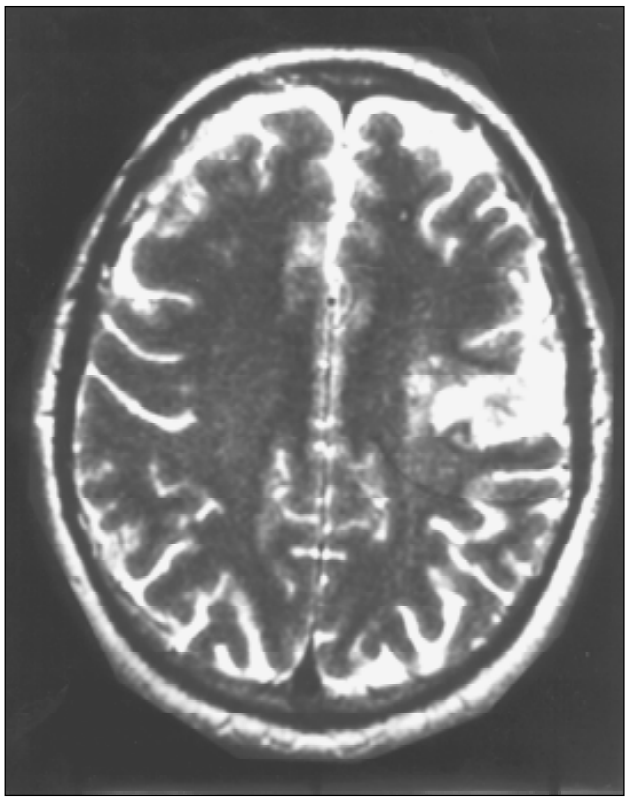

**Fig. 2a. An MRI study demonstrated a left frontoparietal area of infarction.**

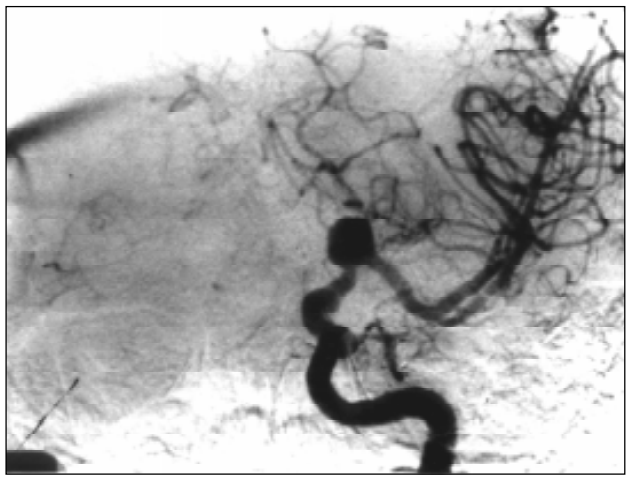

**Fig. 2b. DSA demonstrated an 18 mm partially thrombosed aneurysm of the left internal carotid artery bifurcation region. There was no filling of the A1 segment on the left side.**

frontoparietal area of infarction and an 18 mm partially thrombosed aneurysm of the left internal carotid artery bifurcation region. There was no filling of the A1 segment on the left side (Figs 2a and 2b).

The patient was taken to the operating room for aneurysm obliteration. At operation, an internal carotid artery bifurcation aneurysm was identified which was found to be partially thrombosed. The A1 segment at the base of the aneurysm appeared to be thrombosed at its origin, with no anterograde or retrograde filling through the vessel. Temporary vessel clips were placed on the middle cerebral artery and on the internal carotid artery in order to trap the aneurysm. An incission was made to open the aneurysmal dome, and microendarterectomy of the aneurysm neck and parent vessel was performed. Attempts were made to perform endarterectomy of the occluded anterior cerebral artery. Despite multiple attempts it was not possible to restore flow in the occluded A1 segment and therefore a clip was placed accross this vessel. Excellent flow in the middle cerebral artery was obtained and the neck of the aneurysm was subsequently clipped. The patient made an uneventful recovery with gradual improvement of his neurological deficits from his initial stroke. He has been followed for 1 1/2 years without subsequent sequelae.

**Discussion**

We present two cases of unruptured aneurysms which appear to play an important role in the pathophysiology of thromboembolism. Eller, in 1986, described a patients with an 18 mm middle cerebral artery aneurysm causing stroke, which he considered a small lesion (6). It is generally accepted that thromboembolism, if it appears from aneurysm, usually does so in patients with large or giant aneurysms (1, 3, 5, 8, 14). Some authors even conclude that this is an exclusive feature of large or giant aneurysms. In our two patients, the smaller of the two lesions was 7 mm, which is quite unusual to cause thromboembolism.

Thromboembolism is not the only way aneurysms may cause an ischemic stroke, however. There are rare instances of infectious aneurysms (4, 13) as well as aneurysms caused by atrial myxoma (2, 9). Embolus from these lesions may cause stroke. The fact that thromboembolism can occur in association with giant aneurysms has been an event reported by many authors. In this situation, low blood flow in the lesion appears to help in the formation of clot. With slow flow in the aneurysm, the incidence of thrombosis is increased, and therefore the potential for thromboembolism is higer in giant aneurysms. It is of some interest that the two aneurysm that we have observed were at the bifurcation of major cerebral vessels. In both situations, atherosclerotic changes in the wall of the aneurysm extended into one of the adjacent parent vessels. It may be that as part of the pathogenesis of these lesions, a partial dissection of the arterial wall played a role. In both situations, attempts at opening the adjacent occluded arterial segment were unsuccessful. Fortunately the collateral flow in both patients was sufficient to sustain adequate retrograde flow in the area of brain usually supplied by the occluded segment of vessel.

In the treatment of these complex atherosclerotic small aneurysms, opening of the aneurysm sac and microendarterectomy appeared to be of benefit in both our patients. In the patient with a small middle cerebral artery aneurysm, the atheromatous plaque had separated from the wall in one area, producing the potential for intraarterial thrombus. If the lesion had been clipped without

investigating the inside of the vessel, occlusion of the remaining M2 branch vessel may well have occured. In the patient 2 with an internal carotid artery bifurcation lesion, occlusion of what appeared to be the neck on the outside of the vessel would most certainly have produced compromise or complete occlusion of the junction between the internal carotid artery and middle cerebral artery vessels. Thus microendarterectomy appeared to be of benefit in both situations.

In patients who present with ischemic syndromes and small aneurysms, a search should be made for an extracranial source of such symptomatology, such as the carotid artery or thrombus from the heart. When such a search is negative and the only remaining potential etiology is that of an intracranial aneurysm, consideration should be given to a surgical exploration for microendarterectomy and aneurysm obliteration.

1) Thromboembolism may be the presenting symptom of small aneurysms, when an extracranial source is excluded.

2) Thrombosis of the aneurysm may extend to the parent vessel.

3) Microendarterectomy of the aneurysm and the parent vessel(s) with subsequent clipping of the aneurysm appears to be benefitial in preventing further ischemic events and a possible rupture of the aneurysm.

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