Embolization from a Fusiform Middle Cerebral Artery Aneurysm

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SUMMARY A 34-year-old man had a transient ischemic attack and subsequently a completed stroke. Arteriography revealed a large fusiform aneurysm of the left middle cerebral artery with intraluminal thrombus. At surgery, the thrombus was seen within the lumen of the aneurysm. Absolute evidence for embolization is lacking as no examination for this could be done.

Embolization from intracranial aneurysms seems to occur exclusively in large or giant aneurysms. Turbulent flow and a “stagnant zone” probably promotes thrombus formation. The reasons for the relative rarity of subsequent embolization are discussed.

Because embolization from intracranial aneurysm is so uncommon and because aneurysms usually produce focal deficit by other mechanisms, 4 criteria are presented to determine whether embolization is likely.

CEREBRAL ANEURYSMS generally produce neurologic deficit by means of hemorrhage, vasospasm or mass effect. Embolization from cerebral aneurysms is rarely mentioned in reviews of transient ischemic attacks (TIA), non-atherosclerotic causes of stroke, and the non-hemorrhagic consequences of cerebral aneurysm.1-9

Case Report

A 34-year-old, white, right-handed man was admitted with aphasia and a right hemiparesis. He had been in good health until 10 days previously, when he experienced lightheadedness and right hemiparesis lasting 5 minutes. Six days later, he had a similar attack from which he did not fully recover. There were no associated headache or other neurological symptoms. There was no history of diabetes, hypertension, or other significant medical diseases. Family history was also unremarkable.

Examination revealed an alert, cooperative man with a moderate Broca’s aphasia. There was a mild, right central facial paressis and a right hemiparesis affecting the arm more than the leg. He had right-sided hyperreflexia with bilateral flexor plantar responses. The remainder of the neurologic examination was normal. There were no ocular or cervical bruits.

Routine blood studies and a coagulation profile were normal as were plain skull radiographs. An EEG revealed 1½ to 3 cps delta activity over the left hemisphere most marked in the temporal leads. Computed tomography (fig. 1) revealed an area of decreased density in the region of the left internal capsule. At the level of the suprasellar cistern on the left, there was an area of increased density which became enhanced with contrast injection. Arteriography revealed a fusiform aneurysm along the M-1 segment of the middle cerebral artery (fig. 2). There was a suggestion of thrombus within the aneurysm. The aneurysm measured 2 cm in length by 9 mm in height. A lumbar puncture was normal.

The aphasia and hemiparesis improved markedly and 11 days following admission, the patient underwent a left frontotemporal craniotomy. The aneurysm was dissected out using microsurgical technique and was much larger than it appeared on arteriography. Many vessels, clearly patent, were seen coming off the dome. Intraluminal thrombus was seen through the aneurysmal wall opposite the take-off of the small penetrating vessels. The aneurysm could not be clipped or trapped but was wrapped with muslin gauze.

In the immediate post-operative period, the aphasia and hemiparesis was slightly worse. However, he had no further attacks of TIA or stroke and was discharged from the hospital on aspirin 10 grains b.i.d.
and dipyridamol (Persantine) 50 mgs b.i.d. In the next 6 months, the patient continued to improve and had only a subtle Broca's aphasia and a mild right hemiparesis. He has had no further attacks of TIA or stroke.

Discussion

Because of the infrequency of aneurysmal embolization and the difficulty distinguishing it from other complications of subarachnoid hemorrhage (SAH), we suggest 4 criteria to determine whether embolization is a likely etiology: 1) clinical TIA or completed stroke, 2) arteriographic, surgical and/or autopsy verification of the aneurysm, 3) no other lesions which could produce TIA or stroke and 4) no clinical or radiographic evidence of recent subarachnoid hemorrhage (SAH) or vasospasm which could provide an alternative etiology.

We could find only 4 documented patients conforming to these criteria. Auld and Shafey reported 6 patients with TIA not related to atherosclerotic carotid disease. Two patients had giant middle cerebral artery aneurysm. One patient, a 52-year-old man, had frequent TIA of the right carotid system for 4 months prior to SAH. Arteriography and surgery
revealed a right middle cerebral artery aneurysm with intraluminal thrombus. Following microsurgical removal, TIA did not recur. A second patient had concurrent ulcerative plaque of the ipsilateral carotid artery and TIA ceased following endarterectomy. This patient would not, therefore, fulfill the above criteria.

Antunes and Correll reported 2 cases of internal carotid artery aneurysm with embolization. Their first patient was a 64-year-old woman with a right third nerve palsy. Cerebrospinal fluid was normal. Arteriography revealed that the right posterior cerebral artery originated from the internal carotid artery with an aneurysm at the junction. The aneurysm was wrapped with muscle. Three weeks later, she suddenly became comatose with a dense left hemiparesis. At autopsy, there was a partially organized clot within the lumen of the aneurysm, protruding from its posterior aspect. There were multiple areas of encephalomalacia in the distribution of anterior, middle, and posterior cerebral arteries. The second patient was a 53-year-old woman who presented with left hemiparesis. Five days later, she was found to have a complete third nerve palsy. Cerebrospinal fluid was normal. Arteriography and surgery demonstrated an aneurysm of the right internal carotid artery-posterior communicating artery junction. The aneurysm was clipped without further TIA or stroke.

Hoffman et al. reported a 60-year-old woman with 9 episodes of TIA involving the right arm. Arteriography revealed an aneurysm of the left middle cerebral artery trifurcation. There was no radiographic evidence of thrombosis of the sac. At surgery, the aneurysm was clipped and the sac excised. It contained thrombus, portions of which were still attached to the aneurysmal wall.

There are other patients reported in the literature which may represent aneurysmal embolization. Scott and Ballantine reported a patient with a known, unoperated, large left middle cerebral artery aneurysm who had aphasia and confusion which cleared in 2 days. There was no evidence of SAH. They suggested that the symptoms were "due to an embolus from the aneurysm or to a seizure with post-ictal phenomenon". Taptas and Katsiotis reported a 35-year-old man admitted with a SAH who 15 days later developed aphasia and right hemiparesis. Arteriography showed a filling defect in the left carotid which they said was more consistent with thrombus than vasospasm.

Thomas and Reagen studied 27 patients with non-hemorrhagic complications of intracranial aneurysm of the internal carotid artery. Two patients had TIA and one patient died of a massive cerebral infarct on the side of the aneurysm. No details were provided.

The occurrence of embolization from an aneurysm...
of the extracranial carotid artery has become more easily recognized. It is stated that over half of these patients present with TIA or stroke. 18

All of the above well-documented patients had large, if not technically giant, aneurysms (2.5 cm or more in greatest diameter). Schunk 16 has analyzed aneurysmal size and its relationship to thrombus formation. Both arteriographic and histologic evidence indicated that larger aneurysms have a greater tendency toward partial thrombosis. Two different experimental models of aneurysms 17, 18 suggest that the relationship of the size of the aneurysmal orifice to the volume of the sac may be the critical factor in thrombus formation. The presence of turbulent flow 19 and a "stagnant zone" may promote thrombus. Turbulence alone may be insufficient. Sato and Kamitani 20 reported an arteriographic study of a giant middle cerebral artery aneurysm. There was clear radiologic turbulence but at surgery no thrombus was present. It may be important that the neck of the aneurysm was broad and there was no "stagnant zone."

Clinically, most giant aneurysms contain significant thrombus. 21 Why then is aneurysmal embolization so rare in the central nervous system when it is relatively common elsewhere in the body? 21, 22, 23 One reason is that giant aneurysms represent only 3-4% of all intracranial aneurysms. 22 Large but not technically giant aneurysms are also uncommon. Secondly, there may be limited period(s) during the evolution of a large or giant aneurysm when embolization can occur. The "at-risk" period when the orifice size, volume, turbulence, and "stagnant zone" are optimal for thrombus formation and embolization may be short. The thrombus may then become organized into the aneurysmal sac and less likely to embolize. Thirdly, the intraluminal and extraluminal pressure exerted on the aneurysm is probably fairly constant. Aneurysms of either the extracranial carotid artery or the popliteal artery, 24 for example, would be subject to the local pressure generated by contraction of adjacent muscles. This might increase intraluminal and consequently intraluminal pressure and tend to dislodge unorganized or partially organized thrombus.

References
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