Giant thrombosed aneurysm associated with an arteriovenous malformation

Case report

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The authors describe a case of giant anterior cerebral artery aneurysm associated with an anatomically related arteriovenous malformation (AVM). The aneurysm was almost completely thrombosed and was resected along with the AVM.

KEY WORDS  •  giant aneurysm  •  arteriovenous malformation

The association of arteriovenous malformations (AVM's) with intracranial aneurysms has been reported repeatedly in the literature. However, the occurrence of AVM's with giant globoid intracranial aneurysms is very rare. We are reporting a giant globoid aneurysm arising from the left anterior cerebral artery (ACA) and a large frontal AVM originating from the right and left ACA's.

Case Report

This 59-year-old woman was admitted to The Johns Hopkins Hospital on May 5, 1976, because of seizures, progressive mental deterioration, and urinary incontinence. About 4 years before admission she lost consciousness on a golf course and had a grand mal seizure. She was taken to a nearby hospital where an angiogram revealed a frontal AVM, with no shift of the ACA's. About 2 years later she began to have difficulty with memory and control of her urine. In December, 1975, she began having periodic grand mal seizures that were treated by Dilantin (diphenylhydantoin sodium) 100 mg, four times daily. Occasionally her seizures had a focal onset in the left upper extremity. There was no history of intolerable headache, diabetes, or high blood pressure.

Examination. On admission the patient's blood pressure was 106/70 mm Hg, and vital signs were normal. The general physical examination was negative. She was conscious, alert, and mildly demented; recent and short-term memory was disturbed. Her speech was not disturbed. She had a mild left central facial weakness and left hemiparesis. Deep tendon reflexes were 2 to 3 plus with down-going toes. Tone was intact and sensation to all modalities preserved.

The skull films revealed a shell of calcification, 5 × 6 × 7 cm in size, in the right frontal region (Fig. 1). Computerized tomography (CT) revealed a rim of calcification measuring 5 to 6 cm in diameter in the right frontal lobe with enhancement anteriorly and medially, compatible with an AVM (Fig. 2). Four-vessel angiography disclosed a large
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AVM fed primarily from A1 on the right and, by way of the anterior communicating artery, from the left ACA (Fig. 3). There was no contrast visualization of the calcified mass. The ACA's were shifted to the left. The mass was diagnosed preoperatively as an old intracerebral hematoma from the AVM.

Operation. On May 24, 1976, a right frontal craniotomy was performed under general anesthesia. About 1.5 cm under the right
FIG. 4. Superior view of the aneurysm at craniotomy. A 1 × 1 cm window was incised in the dome (arrow) and the wall was dissected anteriorly to clip the major feeder and resect the aneurysm. Ant = anterior, Sup = superior, A = aneurysm, Inf = inferior, Post = posterior.

FIG 5. Upper: Lateral view of the postoperative right internal carotid angiogram with nonvisualization of the arteriovenous malformation (AVM) and right anterior cerebral artery at 3.5 (left) and 8 (right) seconds. Eight seconds after injection, there is retrograde filling of the pericallosal and callosomarginal arteries (arrowhead). Lower: Lateral view of postoperative left internal carotid angiogram showing nonfilling of the AVM and anterior cerebral arteries.
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frontal cortex a giant aneurysm with minimal pulsation was exposed. The aneurysm was more than 6 cm in diameter. No blood could be aspirated on insertion of a fine needle. Cautiously a small window measuring 1 × 1 × 1 cm was excised from the wall (Fig. 4). Immediately we encountered a grayish organized thrombus. The wall measured 3 to 4 mm in greatest thickness. Part of the thrombus was removed and after collapse of the wall, we could expose the major feeder which was from the left Aa. It was clipped and the aneurysm resected. Cross section of the aneurysm exposed a fresh clot measuring 1 × 1.5 × 1 cm at the distal apex of the major feeder and surrounded by organizing laminated thrombus. Immediately anterior and medial to the aneurysm was a large AVM. This was also resected after the feeders were clipped.

Postoperative Course. The postoperative course was complicated by akinetic mutism and confusion. On July 28, 1976, she had a ventriculoastral shunt performed because of moderate hydrocephalus demonstrated by CT scan. She gradually improved to the extent that 2 years after surgery she is free of seizures and has good sphincter control. She does not have any focal neurological deficit. She is able to participate in household activities regularly and golf when she has spare time. An angiogram performed immediately after surgery revealed nonfilling of the ACA’s (Fig. 5); later films, however, revealed that they filled retrogradely from the middle cerebral artery circulation. This could be caused by spasm, retrograde thrombosis of the remaining A1 segments, or inadvertent clipping of the ACA’s. The histopathological appearance of the surgical specimen was compatible with aneurysmal wall up to 5 mm in thickness, with mural calcification and organizing thrombus inside. A few fragments of brain around the aneurysmal wall showed astrocytosis.

Discussion

Giant intracranial aneurysms constitute about 5% of all intracranial aneurysms. An aneurysm with a diameter more than 1.5 or 2.5 cm is defined as a giant one. Reports of incidence of subarachnoid hemorrhage varies; it could be low or quite high. These aneurysms typically present with pressure phenomena, most frequently on the visual apparatus. They may cause headache, epilepsy, dementia, and hemiparesis. The association of berry aneurysms with AVM has been reported before; however, the presence of a giant aneurysm with AVM is extremely rare.

Giant aneurysms may be the result of relentless expansion of saccular aneurysms. There is evidence that they could be congenital. Shenkin, et al., believed that the concurrence of an aneurysm on the major feeder of an AVM is due to increased blood flow.

Different authors advocate different modalities of treatment. If these aneurysms are asymptomatic, they can be treated conservatively, hoping for spontaneous thrombosis. Carotid ligation may be performed, but sometimes it is not tolerated. These aneurysms may be resected if they cause pressure phenomena; however, this is not always possible, because the aneurysm may participate in cerebral circulation.

References


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