Giant Serpentine Aneurysms: Radiographic Features and Endovascular Treatment

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PURPOSE: To describe the characteristic CT, MR, and angiographic features of giant serpentine aneurysms and discuss their endovascular treatment. **METHODS:** Thirteen patients with giant serpentine aneurysms were studied at our institution in the last 3 years. They all underwent CT and MR studies as well as cerebral angiography. More recently, some of the patients were studied with MR angiography. Seven patients had endovascular occlusion of the giant serpentine aneurysms, 3 with *N*-butyl cyanoacrylate, 2 with Guglielmi detachable coils, and 2 with detachable balloons. **RESULTS:** Giant serpentine aneurysms mimic cerebral neoplasms on CT and MR studies; they are often associated with mass effect and adjacent edema, and they enhance with contrast medium. The cerebral angiogram shows a residual irregular lumen of the partially clotted aneurysm, which continues into normal branches supplying the distal arterial territory. Six patients were treated successfully with an endovascular approach consisting of complete and permanent occlusion of the parent artery. **CONCLUSION:** Giant serpentine aneurysms form a subgroup of large intracranial aneurysms that have specific CT, MR, and angiographic features, which should be recognized before their treatment. The endovascular treatment of the aneurysm consists of permanent occlusion of the parent artery.

Index terms: Aneurysm, giant; Aneurysm, therapeutic blockade; Interventional neuroradiology

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Giant serpentine aneurysms form a subgroup of large intracranial aneurysms that have characteristic computed tomographic (CT), magnetic resonance (MR), and angiographic features, which should be recognized before their attempted treatment. If unrecognized, they can easily be mistaken on imaging studies as neoplasms, particularly because they often present with progressive neurologic deficits and are often associated with adjacent edema and mass effect on the CT and MR studies.

Cerebral angiography establishes the diagnosis of giant serpentine aneurysms and provides the anatomic detail necessary for their treatment. In the past, no surgical therapy could be offered to the patients. Now, an endovascular approach is possible. The feeding parent vessel can be sacrificed proximal to the giant serpentine aneurysms by a variety of methods, with vascular supply to the distal territory through either spontaneous collateral circulation or surgical anastomosis.

Methods and Materials

Thirteen patients with giant serpentine aneurysms were evaluated at our institution in the last 3 years. They ranged in age from 5 years to the sixth decade with a predilection to the male gender (9 of 13). They all underwent selective cerebral angiography as well as a CT scan and/or MR imaging.

They all received either iodinated radiographic contrast material and/or gadopentatate dimeglumine. The MR studies included proton-density and T1- and T2-weighted pulse sequences. More recently, MR angiographic studies were used in the evaluation of five of these patients with three-dimensional time-of-flight and/or 3-D phase-contrast sequences.

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Seven of the 13 patients underwent endovascular occlusion of the parent vessel with detachable balloons or *N*-butyl cyanoacrylate or Guglielmi detachable coils (Target Therapeutics, Fremont, Calif). The detachable balloons were detached as close to or immediately proximal to the residual patent vascular channel of the giant serpentine aneurysms. The balloons were filled with isotonic iodinated contrast medium. The patients underwent balloon test occlusions before the permanent occlusions, with electroencephalographic monitoring and transcranial Doppler. When *N*-butyl cyanoacrylate was used to occlude the parent vessel, the liquid adhesive was deposited at the origin of the residual channel of the giant serpentine aneurysms often using a "sandwich technique." In one of these patients, a selective Wada test was performed before injection of liquid adhesive.

Six patients (6 of 13) were treated conservatively, although 2 of these patients are awaiting possible endovascular treatment.

Results

All patients presented with progressive waxing and waning neurologic deficits with or without epilepsy. The neurologic deficits varied from speech disturbances, hemiparesis, and sensory symptoms in those cases in which the giant serpentine aneurysms arose from the middle cerebral artery, to ataxia and lower cranial nerve involvement in those in which the vertebrobasilar circulation was affected. Two patients with giant serpentine aneurysms of the supraclinoid carotid artery presented with chiasmatic compression and resulting visual field defects. The neurologic symptoms improved in all patients who were treated with corticosteroids.

The CT, MR, and angiographic features of giant serpentine aneurysms are illustrated by the following representative examples (Figs 1–4). The precontrast CT scans often show a well-circumscribed heterogeneous hyperdense mass, either round or oval in shape, which appears extraaxial and is associated with adjacent edema, mass effect, and shift of the interhemispheric structures across the midline (Figs 1A and 2A). The degree of hyperdensity within the mass, which indicates the presence of blood clots, varies with the age and the relative chronicity of the clots. Thin peripheral rims of calcifications can surround the hyperdense mass, particularly in the older patients, indicating a slow, progressive organization. The progression of calcification process extends in a somewhat concentric and centripetal fashion to obliterate the entire mass of the giant serpentine aneurysms eventually.

The postcontrast CT scans (Figs 1B and 2B) show heterogeneous enhancement of the mass with the relatively hypodense regions of the giant serpentine aneurysms enhancing more intensely than the remaining hyperdense areas. The pattern of enhancement is similar to that seen in intracranial neoplasms. Additionally, careful examination of the mass on the postcontrast scans shows an eccentrically located enhancing tubular channel within the giant serpentine aneurysms, which represents the residual serpentine lumen of the aneurysm.

The T1-weighted MR scans (Fig 1C) show a corresponding heterogeneous hyperintense mass reflecting the presence of primarily extracellular methemoglobin and other less hyperintense blood products, all indicating the various chronological ages of the blood clots within the giant serpentine aneurysms. The postcontrast T1-weighted sequences (Fig 1D) show heterogeneous enhancement of the various clotted segments of the aneurysm. The persistent eccentric nonclotted lumen of the giant serpentine aneurysms does not enhance with contrast medium. The T2-weighted scans (Fig 1E) confirm the presence of the clots and show to a better advantage the associated hyperintense edema of the adjacent brain parenchyma and the peripheral rim of hemosiderin and/or calcium. In the heavily calcified giant serpentine aneurysms, the shortening of the T2 relaxation time related to the presence of calcium overwhelms the relative lengthening of the same parameter because of the extracellular methemoglobin and results in a hypointense appearance to the mass on the T2-weighted sequences. The selective cerebral angiograms (Figs 1F and G, 2C and D, 3A and B, and 4) show avascular mass effect related to the clotted segments of the giant serpentine aneurysms and a patent residual serpiginous and irregular lumen of the aneurysm traversing the region of avascular mass effect. By definition, the points of entrance and exit of the residual aneurysmal lumen are distinct and separated by a variable distance (Figs 1H and I and 3C and D). This distinguishes giant serpentine aneurysms from ordinary giant saccular aneurysms in which the neck constitutes the single point of entrance and exit of blood flow inside the aneurysmal lumen. The residual lumen of the giant serpentine aneurysms continues distally and gives rise to normal dis-

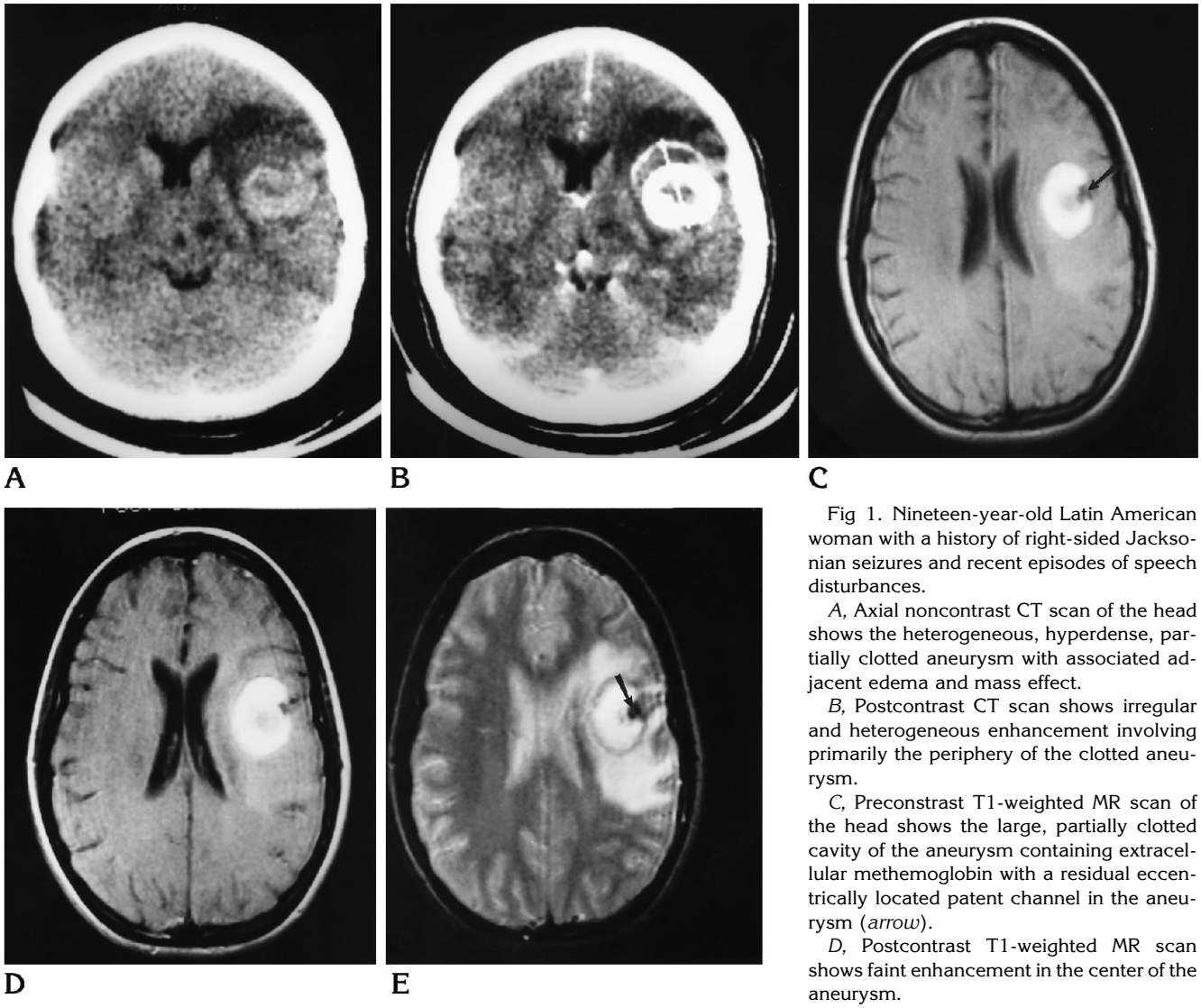


Fig 1. Nineteen-year-old Latin American woman with a history of right-sided Jacksonian seizures and recent episodes of speech disturbances.

A, Axial noncontrast CT scan of the head shows the heterogeneous, hyperdense, partially clotted aneurysm with associated adjacent edema and mass effect.

B, Postcontrast CT scan shows irregular and heterogeneous enhancement involving primarily the periphery of the clotted aneurysm.

C, Preconstrast T1-weighted MR scan of the head shows the large, partially clotted cavity of the aneurysm containing extracellular methemoglobin with a residual eccentrically located patent channel in the aneurysm (*arrow*).

D, Postcontrast T1-weighted MR scan shows faint enhancement in the center of the aneurysm.

E, T2-weighted MR scan shows the edema surrounding the aneurysm in the adjacent brain with the residual channel of the aneurysm manifested as a focal signal void (*arrow*). (*Figure continues.*)

tal arterial branches supplying territories that would have otherwise been supplied by the same parent vessel. The course of the residual lumen follows the normal direction of the parent vessel, even in cases in which the giant serpentine aneurysms involve the supraclinoid internal carotid artery or the vertebrobasilar junction.

In all of the seven patients we treated, the endovascular therapy consisted of intentional and permanent occlusion of the parent artery proximal to the origin of the giant serpentine aneurysms (Table). We presumed that leptomeningeal collateral circulation would revascularize in a retrograde fashion the distal territory of the parent vessel we intended to occlude. Two giant serpentine aneurysms arising from the P1 or P2 segment of the posterior cerebral artery were treated with permanent occlusion of the artery with Guglielmi detachable coils. In one of these patients a temporooccipital infarct developed because of lack of collateral circulation. In the second patient, the occlusion of the posterior cerebral artery was well tolerated without any sequelae.

Two giant serpentine aneurysms arising from the supraclinoid internal carotid artery were treated with permanent occlusion of the parent vessel using detachable balloons and thrombogenic stainless steel coils. The permanent occlusion was preceded by a successful test occlusion of the same vessel under systemic

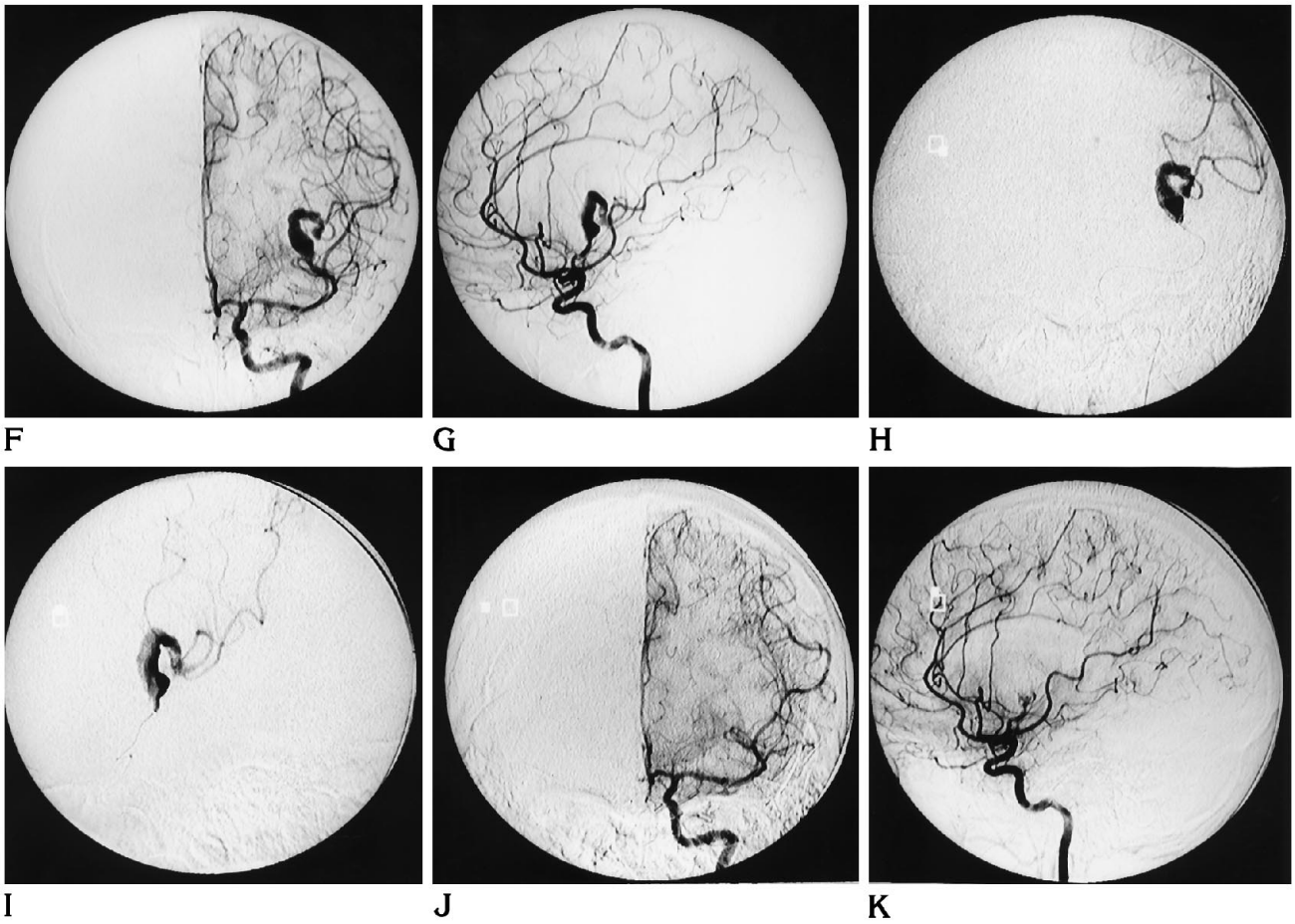
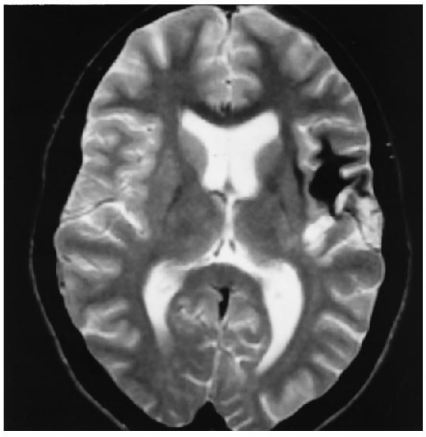


Fig 1, *cont'd. F* and *G,* Anteroposterior and lateral views of a selective left internal carotid arteriogram showing left centrosylvian mass effect and the irregular patent channel of the giant serpentine aneurysms after the expected course and position of a parietal branch of the left middle cerebral artery.

H and *I,* Anteroposterior and lateral views of the selective angiogram of the parent vessel at the origin of the giant serpentine aneurysm.

J and *K,* Postembolization anteroposterior and lateral views of a left internal carotid arteriogram showing complete obliteration of the giant serpentine aneurysm with very faint retrograde opacification of the distal territory of the occluded parent vessel through leptomeningeal collaterals.

L, T2-weighted MR scan obtained 1 year after the embolization shows complete resolution of the mass effect and edema with significant shrinkage of the giant serpentine aneurysm, which is now replaced by either hemosiderin and/or calcium.



heparinization with electroencephalography and transcranial Doppler monitoring. The permanent occlusion was well tolerated without any neurologic deficit.

Two giant serpentine aneurysms arising from the middle cerebral artery were treated with permanent occlusion of the parent vessel using liquid adhesive. After the occlusion, one patient had transient dysphasia, which resolved with aggressive volume expansion and calcium channel blockers. The second patient became aphasic within 5 minutes of the occlusion and was treated with volume expansion, but demonstrated an infarct in the arterial territory with

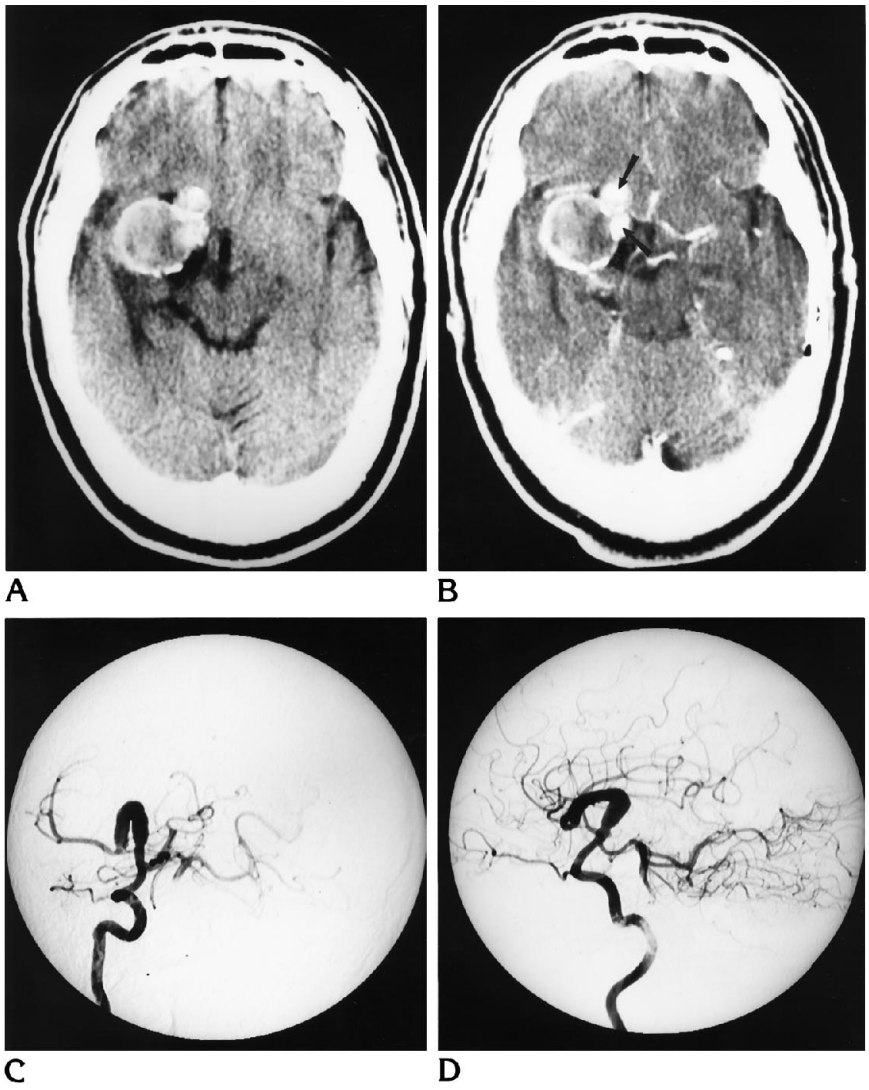


Fig 2. A 56-year-old black man with visual field defect and seizure disorder.

A, Axial noncontrast CT scan of the head shows the large partially clotted hyperdense giant serpentine aneurysm with a peripheral rim of calcification in its wall.

B, Postcontrast CT scan of the head shows the enhancement in the residual irregular channel of the giant serpentine aneurysm (*arrows*).

C and *D,* Oblique and lateral views of a selective right internal carotid arteriogram show the residual channel of the supraclinoid internal carotid artery, extending from the level of the origin of the posterior communicating artery to the level of the bifurcation of the internal carotid artery.

areas of petechial hemorrhage. After rehabilitation, the patient regained neurologic function.

One patient with a giant serpentine aneurysm of the right posterior inferior cerebellar artery was treated with permanent occlusion using a liquid adhesive agent. The patient improved clinically with resolution of intractable hiccups and improved gait.

Discussion

Giant serpentine aneurysms form a subcategory of intracranial giant aneurysms, which possess certain characteristic anatomic and radiographic features. The entity is comprehensively reviewed by Aletich et al (1), who detail previous cases in the literature in this issue of *AJNR*. By definition, a serpentine aneurysm is usually giant (>2.5 cm) and is almost completely clotted except for a residual irregular channel. The residual channel is eccentrically located within the clotted mass of the aneurysm and continues distally to resume a normal appearance beyond the aneurysm and to supply normal arterial branches. The residual channel often follows a wavy, sinusoidal course—thus the descriptive word *serpentine*. Unlike the saccular variety of aneurysms, giant serpentine aneurysms do not have discrete identifiable necks but consist rather of residual deformed arterial channels which follow the otherwise normal orientation and location of the parent vessel. The residual lumen is surrounded by a larger mass of organized blood clot. The parent vessel is usually a branch of the middle or posterior cerebral arteries, the distal vertebral artery at its

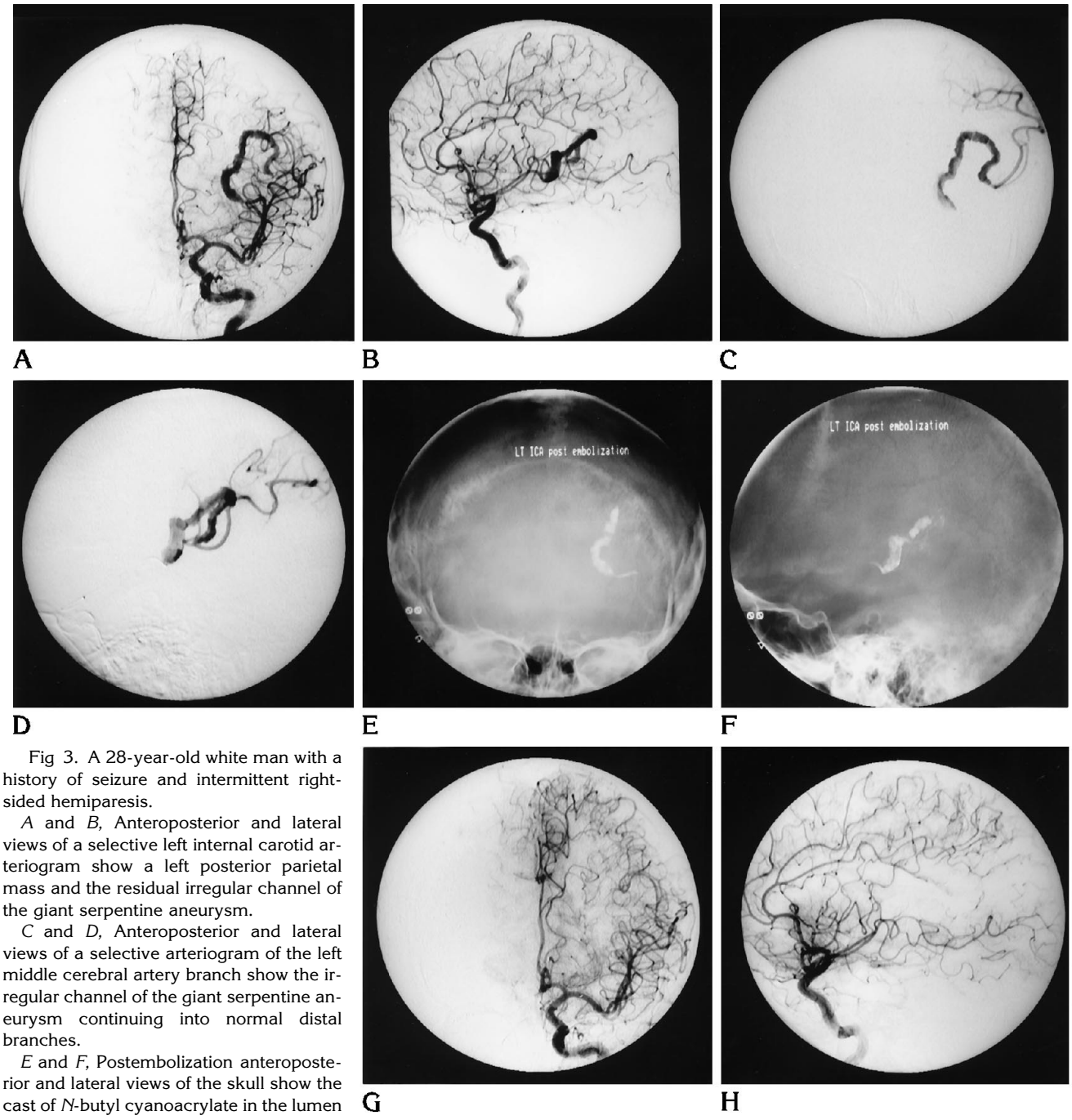


Fig 3. A 28-year-old white man with a history of seizure and intermittent right-sided hemiparesis.

A and *B,* Anteroposterior and lateral views of a selective left internal carotid arteriogram show a left posterior parietal mass and the residual irregular channel of the giant serpentine aneurysm.

C and *D,* Anteroposterior and lateral views of a selective arteriogram of the left middle cerebral artery branch show the irregular channel of the giant serpentine aneurysm continuing into normal distal branches.

E and *F,* Postembolization anteroposterior and lateral views of the skull show the cast of *N*-butyl cyanoacrylate in the lumen of the channel after the embolization.

G and *H,* Postembolization anteroposterior and lateral views of a left internal carotid arteriogram show complete obliteration of the giant serpentine aneurysms and minimal retrograde collateral circulation into the distal territory of the occluded left middle cerebral artery branch.

junction with the basilar artery, or the supraclinoid internal carotid artery. The anterior cerebral artery was not involved in our series. In one of our cases in which the giant serpentine aneurysms involved the supraclinoid internal carotid artery, the aneurysm incorporated the origin of the posterior communicating artery. Unlike saccular aneurysms, giant serpentine aneurysms do not occur at the apical medial gap found at arterial forkings (2).

Giant serpentine aneurysms usually present with seizures or a progressive neurologic deficits related to increasing intracranial mass effect and adjacent edema. Rarely, as in 1 of our

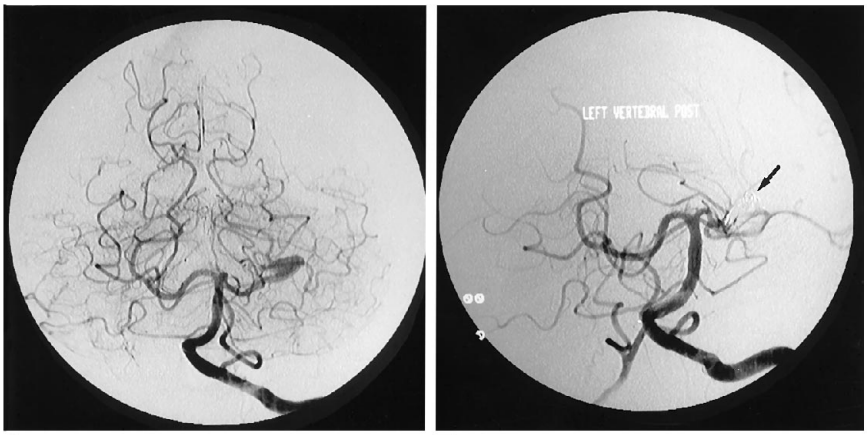


Fig 4. A 58-year-old man with progressive memory loss.

A, Preembolization vertebral angiogram demonstrates a serpentine aneurysm involving the left posterior cerebral artery.

B, Postembolization vertebral angiogram shows the Guglielmi detachable coils in the left posterior cerebral artery (*arrow*) at the neck of the aneurysm. Neither the aneurysm nor the posterior cerebral artery opacify with contrast medium.

13 patients, they can cause subarachnoid hemorrhage. Administration of corticosteroids often results in amelioration of the neurologic deficit.

Tomasello et al (3) reported the progression over 5 years of a small fusiform aneurysm of a branch of the middle cerebral artery into a giant serpentine aneurysm, suggesting that the latter may have its origin in fusiform aneurysms that grow larger in time and undergo thrombosis and organization. Fodstad et al (4) reported formation of a giant serpentine aneurysm after carotid ligation for a giant aneurysm. Pathologically, surgically resected giant serpentine aneurysms show a thick fibrous wall surrounding old laminated clots with attempts at recanalization. The wall of the aneurysm contains neovessels, deposits of hemosiderin, and calcifications (5). Progression of calcification is often associated with a regression of mass effect and edema. In our series, the heavily calcified giant serpentine aneurysms showed relatively little mass effect and adjacent edema on the CT or MR studies when compared with the less calcified lesions. This suggests that giant serpentine aneurysms undergo repetitive episodes of localized eccentric hemorrhages, which become organized into mature clots and are incorporated into the original lumen of the parent artery. These repetitive

Thirteen patients with giant serpentine aneurysms, seven with parent artery occlusion

Patient	Location	Device for Parent Artery Occlusion	Pretherapy Test	Permanent Occlusion?	Complication
G.B.	Right PCA	Guglielmi detachable coil	None	Yes	None
D.P.	Left PCA	Guglielmi detachable coil	None	Yes	Left temporo-occipital infarct
B.G.	Right ICA	Detachable balloon	Successful test occlusion	Yes	None
P.H.	Left ICA	Detachable balloon	Successful test occlusion	Yes	None
E.D.	Left MCA	Liquid adhesive	None	Yes	Transient aphasia
R.D.	Left MCA	Liquid adhesive	Successful selective Wada test	Yes	Aphasia
R.B.	Right PICA	Liquid adhesive	None	Yes	None
V.B.	VBJ	None	···	···	···
D.L.	Left MCA	None	···	···	···
J.C.	Right ICA	None	···	···	···
G.R.	Right MCA	None	···	···	···
E.F.	Left PCA	None	···	···	···
M.E.	Left M1	None	···	···	···

Note.—PCA indicates posterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery; PICA, posterior inferior cerebellar artery; VBJ, vertebrobasilar junction; and M1, horizontal segment of MCA.

episodes of alternating hemorrhage and subsequent thrombosis lead to expansion and enlargement of the aneurysm and worsening mass effect. With time, the laminated clots of different ages become well organized and heavily calcified, limiting the expansion of the aneurysm and the associated edema in the adjacent brain.

The treatment of giant serpentine aneurysms should aim to arrest the growth of the aneurysm, to eliminate the mass effect, and to obliterate the abnormal vascular channel. The most effective way to accomplish these goals would be the direct and permanent occlusion of the parent artery at the origin of the aneurysm. This can be best achieved by endovascular means: selective catheterization of the parent artery and occlusion of the vessel with detachable balloons, *N*-butyl cyanoacrylate (Figs 3E and F), or Guglielmi detachable coils.

The endovascular occlusion of the parent artery is undertaken after careful functional testing of the distal territory to assess for any potential neurologic deficit that may ensue. Before the permanent occlusion of the parent artery (when feasible), a test occlusion angiogram is performed to study the presence and the anatomy of the leptomeningeal collateral circulation to the distal arterial territory. If temporary occlusion of the parent artery is not tolerated, a surgical revascularization procedure of the distal territory can be performed before permanent endovascular occlusion of the vessel or surgical resection of the aneurysm. This was not done in any of our cases.

After endovascular occlusion of the parent artery, the giant serpentine aneurysms progressively shrink in size and are replaced by a mixture of calcium and hemosiderin deposition (Fig 1L). Concomitantly, the adjacent edema and mass effect disappear, and the clinical symptoms subside.

Conclusion

Giant serpentine aneurysms are a specific pathologic entity that can affect the intracranial blood vessels. They have characteristic features on CT and MR studies that mimic neoplastic disease. Cerebral angiography is diagnostic and provides crucial information for the treatment of these lesions. When feasible, permanent endovascular occlusion of the parent vessel is the treatment of choice. Surgical resection is reserved for cases in which the endovascular treatment cannot be achieved.

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