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Embolization of Arteriovenous Malformations of the Temporal Lobe via the Anterior Choroidal Artery

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The indications, technique, and results of embolization of arteriovenous malformations with liquid adhesive agents delivered through the anterior choroidal artery are reported. Arteriovenous malformations of the temporal lobe were found in four patients with intracerebral bleeding and two with intractable epilepsy. In five of the six, the dominant arterial feeder was the anterior choroidal artery. All patients underwent superselective catheterization of the anterior choroidal artery and embolization of the arteriovenous malformation. Complications related to the anterior choroidal artery embolization developed after embolization in one patient, after which we changed our technique of embolizing arteriovenous malformations via this artery. A thorough understanding of the functional anatomic structures supplied by each segment of the artery is important. Guidelines for safe catheterization and embolization are given.

Embolization of arteriovenous malformations fed predominantly by the anterior choroidal artery is difficult and dangerous. An understanding of the functional anatomy of this artery and proper technique can enable successful embolization of arteriovenous malformations via this route.

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Endovascular therapy is an accepted treatment for intracranial arteriovenous malformations (AVMs) [1-4]. In our institution the endpoint of AVM therapy for patients who have hemorrhaged is complete angiographic exclusion of the lesion from the cerebral circulation, thus preventing bleeding. This goal is achieved either by embolization alone or by preparing the patient for postembolization surgery or stereotaxic irradiation. In the large majority of AVMs treated, the working goal is reduction of the AVM nidus to a size that can be treated with stereotaxic irradiation (2 cm in most centers) [5]. These AVMs are located in deep cerebral structures or eloquent cerebral cortex. Temporal lobe AVMs frequently are nourished by the anterior choroidal artery (AChA), but in general this artery is not dilated, thus making selective catheterization of it difficult or impossible. Once the artery is catheterized, a complete and thorough understanding of the functional anatomy is essential prior to embolization.

Our functional/anatomic understanding of the complex anatomy of the AChA is based on the work of Theron and Newton [6] and Rhoton et al. [7]. The AChA nourishes many important anatomic structures. In general, it is the first branch of the internal carotid artery distal to the posterior communicating artery. Its course is divided into cisternal and intraventricular portions. In its cisternal course, the AChA runs along the optic tract, just superior to the upper margin of the uncus. All along its cisternal course it sends important branches to the mesencephalon, diencephalon, and cerebrum. It sends numerous perforating branches superiorly into the substance of the brain, into or through the optic tract. The most proximal branches extend into the medial portion of the globus pallidus. The more distal perforators extend into the substance of the posterior two thirds of the internal capsule and the tail of the caudate nucleus. The ventrolateral thalamic nucleus,

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TABLE 1: Summary of Findings in Patients Undergoing Embolization via the Anterior Choroidal Artery

Case No.	Age at Presentation (years)	Sex	Initial Findings	Location	Size Before/After Emb. (cm)	Arterial Feeders	Venous Drainage	Grade Before/After Emb.	Emb. Procedure(s)	Complications	Other Treatment
1	8	F	L temporal hemorrhage, R homonymous hemianopia, mild dyphasia	L temporal; superior, middle, and parahippocampal gyri, including hippocampus	2.0 × 4.0/2.0 × 3.0 lat. 1.5 × 2.5/1.5 × 2.0 AP	(1) L AChA; (2) L lentostriates	Deep: subependymal veins to straight sinus	IV/III	(1) Failed to catheterize AChA; (2) 2 mo later: L carotid puncture with Pursil catheter into ostium of AChA and injection of 0.35 ml of 0.5/1.3 ml of IBCA/iophendylate	12 hr after Emb.: R facial and upper extremity plegia, dysphasia, memory deficit; 5-yr FU: R lower extremity hypertonia, upper extremity dystonia, memory deficit, mild dysphasia, R inferior quadrantanopia, dysgraphia	Stereotactic radiation 1½ yr after Emb.; partial re-sult
2	21	F	R temporal hemorrhage and IVH; total recovery except for inferior quadrantanopia	R post. medial and around temporal horn of lat. ventricle	3.5 × 1.0/2.5 × 1.0 lat. 3.0 × 1.0/2.0 × 1.0 AP	AChA and post. lat. choroidal artery	Deep: subependymal veins: basal vein to straight sinus	IV/III	R femoral approach: selective catheterization with Pursil propulsion chamber into AChA, laterally into lat. temporal branch; Emb. with 0.07 ml of 0.5/1 ml NBCA/iophendylate, single injection	None	1-yr FU: persistent closure of AChA component; unable to catheterize post. lat. choroidal artery; accepted radiation therapy
3	22	M	Medically refractory epilepsy	L superior and middle temporal gyrus	3.5 × 2.0/0.5 × 0.7 lat. 2.0 × 2.0/0.5 × 0.5 AP	(1) L AChA; (2) L anterior and middle temporal branches of MCA; (3) cortico-cortical anastomoses with PCA	Superficial and deep	III/I	(1) NBCA Emb. of three MCA feeders. (2) 2 mo later: L carotid approach: Pursil catheter to AChA, but would not advance; small nondetachable balloon at ostium was used to push Pursil further. When lat. temporal branch was reached, (3) Emb. was done with 0.1 ml of 0.5/1 ml of NBCA/iophendylate	None	Referred for stereotactic radiation
4	44	M	Large temporal and internal capsule hemorrhage; recovered to mild hemiplegia	R temporal lobe; superior, middle, and parahippocampal gyrus, including hippocampus	2.5 × 4.0/3.0 × 2.5 lat. 3.0 × 1.5/2.5 × 1.5 AP	R AChA; R MCA, insular branches	Deep: subependymal-basal vein-straight sinus; superficial: Labbé to lat. sinus and ant. temporal vein to cavernous sinus	IV/IV	R carotid approach: Pursil propulsion chamber, selective catheterization of AChA into lat. temporal branch; Emb. with 0.08 up to 0.12 ml of 0.5/0.8 ml of NBCA/iophendylate, five times. Unable to catheterize MCA branches	None	Referred for stereotactic radiation

	5	21	F	L temporal ICH, IVH, and SAH	L ant. temporal lobe, entire superior temporal gyrus	4.5 × 1.5/2.5 × 1.5 lat 1.5 × 1.5/1.5 × 1.5 AP	L AChA; L MCA	Two superficial: Labbé to lat. sinus and ant. temporal vein to cavernous sinus	III/II	L carotid approach: Pursil propulsion chamber catheterization of AChA distal to plexal point, laterally into lat. temporal branch; Emb. with single bolus of 0.07 ml of 0.5/1 ml of NBCA/lophendylate	None	Referred for stereotactic radiation
	6	19	F	Medically refractory epilepsy	L temporal lobe, entire superior and middle temporal gyrus	4.5 × 2.5/2.5 × 2.5 lat. 1.5 × 1.5/1.5 × 1.5 AP	L AChA; L MCA, temporal branches	Two superficial: Labbé to lat. sinus and ant. temporal vein to cavernous sinus; one deep: subependymal vein to internal cerebral vein	IV/III	Two MCA Emb. followed in 6 wk by L carotid approach; Pursil propulsion chamber catheterization of AChA distal to plexal point, laterally into lat. temporal branch, Emb. with single bolus of 0.15 ml of 0.5/1 ml of NBCA/lophendylate	None (after first Emb. Amytal test was positive and Emb. aborted)	Referred for stereotactic radiation

Note.—Emb. = embolization; L = left; R = right; lat. = lateral; AP = anteroposterior; post. = posterior; med. = medial; ant. = anterior; IVH = intraventricular hemorrhage; AChA = anterior choroidal artery; IBCA = isobutyl 2-cyanoacrylate; NBCA = *n*-butyl cyanoacrylate; FU = follow-up; MCA = middle cerebral artery; PCA = posterior cerebral artery; ICH = intracerebral hemorrhage; SAH = subarachnoid hemorrhage; yr = year(s); wk = week(s).

subthalamic nucleus, and tail of the caudate also receive a portion of their supply from these perforating branches. The cisternal segment also supplies the uncus, cerebral peduncle, chiasm, optic tract, and lateral geniculate body.

Just before reaching the lateral geniculate body, the AChA angles sharply laterally to enter the choroidal fissure at the plexal point. In all cases it sends branches to the choroid plexus of the temporal horn. The most lateral of the mobile intraventricular branches courses along the lateral aspect of the hippocampus. The medial plexal branch usually is a single large branch that courses along the attachment of the choroid plexus. The more distal portion runs in the stria terminalis, which is the groove separating the caudate nucleus and the thalamus. The size of this branch is inversely related to the collateral supply to the choroid plexus from the posterior lateral choroidal artery. In addition to its supply of the choroid plexus, the medial plexal branch may send small anastomotic branches to supply the roof of the temporal horn and the anterior wall of the atrium. Some of these branches extend deeply to participate in the vascularization of the internal capsule, the tail of the caudate nucleus, and the lateral portion of the thalamus. As the optic radiations pass over the temporal horn, they receive multiple small afferent vessels whose origin is from the medial plexal artery.

Multiple anastomoses exist along the various segments of the AChA. Proximally, the cisternal segment anastomoses with the posterior communicating artery; more distally it communicates with the peduncular segment of the posterior cerebral artery. The lateral geniculate body has a dual reciprocal blood supply, including the ambient segment of the posterior lateral choroidal artery. Intraventricularly, there are multiple anastomoses between the small lateral plexal arteries. As well, the medial plexal branch has free connections with the posterior branch of posterior lateral choroidal artery in the region of the atrium [6, 7]. These anastomotic connections probably account for the rarity of symptoms related to the AChA syndrome [8].

Embolization of this artery with polymerizing agents or particles is dangerous (Dowd CF et al., presented at the Symposium Neuroradiologicum XIV, London, June 1990). While the anastomotic channels may be helpful in reducing ischemic deficits in cases of unifocal embolic or thrombotic occlusion, embolization of the proximal cisternal segment of this artery, with its multiple end arteries that supply the deep nuclei, may have devastating results. Liquid adhesive was chosen for embolization to try to ensure the most definitive and permanent occlusion of the embolized territory possible.

Materials and Methods

The clinical features, anatomic location, angiographic features, AVM grade according to Spetzler and Martin [9], and treatment of each patient are recorded in Table 1. The patients had either intracerebral bleeding (four patients) or medically refractory epilepsy (two patients). The patients were 8–44 years old (mean, 24) at the time of presentation. All patients who had bled were treated endovascularly 2 months after initial presentation. Significant neurologic deficits in two patients were attributable to the initial hemorrhage.

The angiograms and MR images of each patient were graded

according to the proximity of the lesion to eloquent cortex, the size of the nidus, and the presence of deep venous drainage [9]. This grading system has been shown to be predictive of surgical outcome. All the AVMs were judged to be Spetzler and Martin grade III or IV lesions prior to embolization, which carries a 16–27% risk of surgical morbidity and mortality.

Endovascular treatment was performed under vigilant neuroleptic analgesia, allowing constant neurologic monitoring. Direct puncture of the common carotid artery was accomplished with a 16-gauge catheter. A 3.6-French catheter was passed through the puncture catheter and placed into the internal carotid artery. A 0.4-mm internal diameter/0.7-mm external diameter thermomalleable Pursil (Balt Inc., Paris, France) microcatheter with a small dilated distal segment was steam formed to achieve an acute distal curve and propelled into the AChA with the use of a simple propulsion chamber [10]. In case 1, the distal segment of the catheter was not dilated, and the catheter would not advance beyond the ostium; thus, embolization was performed in a very proximal location. In all the other cases, the flow-directed dilated catheter was advanced slowly until it passed the plexal point. Only when the tip of the catheter had passed into the lateral ventricle, through the choroidal fissure, was embolization undertaken. In case 3, we were unable to advance the catheter, so a nondetachable balloon mounted on a 0.2-mm internal diameter/0.3-mm external diameter catheter [11] was passed to the ostium of the AChA. By a series of inflations and deflations in this region, the microcatheter was pushed farther along the course of the AChA, reaching the lateral ventricular segment (Fig. 1). Superselective Amytal testing was used in all but case 1 [12]. Embolization was performed only if no transient neurologic deficit (e.g., motor, visual field, or sensory deficit) occurred after injection of 30 mg of Amytal.

Results

In one case, superselective Amytal testing was particularly useful in determining the amount of embolic material. Following catheterization of the medial plexal branch, 30 mg of sodium amobarbital was injected, which produced no deficit.

One embolization was accomplished with 0.15 ml of *n*-butyl cyanoacrylate, followed by repeat selective Amytal testing. The Amytal test caused an amygdaloid seizure, indicating reflux into eloquent branches. Embolization was not continued, and there were no neurologic sequelae.

Reexamination of all postembolization angiograms revealed a reduction in all but one of the AVM lesions by one or two grades, according to the classification scheme of Spetzler and Martin [9]. The decrease in the size of the lesion was seen on the anteroposterior projection. In case 2, the only feeding arteries to the AVM were the AChA and the posterior lateral choroidal artery. Following embolization through the AChA, the collateral supply to the AVM through the posterior lateral choroidal artery remained unchanged (Fig. 2). The radiation therapy group considered the preembolization nidus too large for stereotaxic radiation. After embolization, all the nidi were thought to be small enough to be irradiated.

Complications occurred in the first case treated (case 1) (Fig. 3). In this 8-year-old girl, a large hematoma of the left temporal lobe caused right homonymous hemianopia and mild dysphasia. Therapy was attempted 2 months following hemorrhage when her visual field had recovered to a right inferior quadrantonopia, but we were unable to catheterize the AChA with a calibrated-leak balloon system. Two months later, we were able to propulse a microcatheter into the ostium of the AChA, but it would not advance further. It was decided to embolize from that position since there was rapid flow to the AVM, which in general provides adequate security to ensure occlusion of the nidus, and not pedicle. A single 0.35-ml mixture of 0.5 ml of isobutyl 2-cyanoacrylate (bucrylate, Ethicon, Inc., Somerville, NJ) and 1.3 ml of iophendylate (Ethiodol, Savage Labs., Melville, NY) was injected at this position. There was no immediate deficit, but transient hemiplegia, complete right homonymous hemianopia, and dysphasia developed 12 hr later [8]. At 5-year follow-up, there was partial

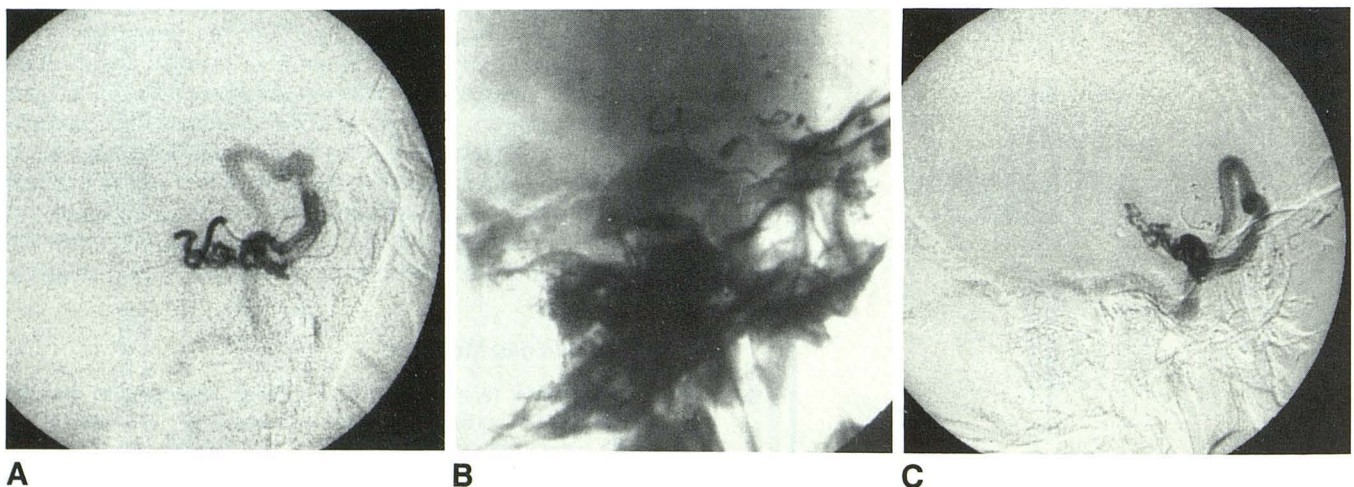


Fig. 1.—Correct catheter placement (case 3).

A, Superselective catheterization of anterior choroidal artery (AChA). Malpositioned catheter, in cisternal portion of AChA, was injected. Microcatheter would not advance farther with flow pressure alone.

B, After inflation and deflation of a nondetachable balloon near ostium of AChA, the microcatheter was advanced past plexal point, into lateral ventricle.

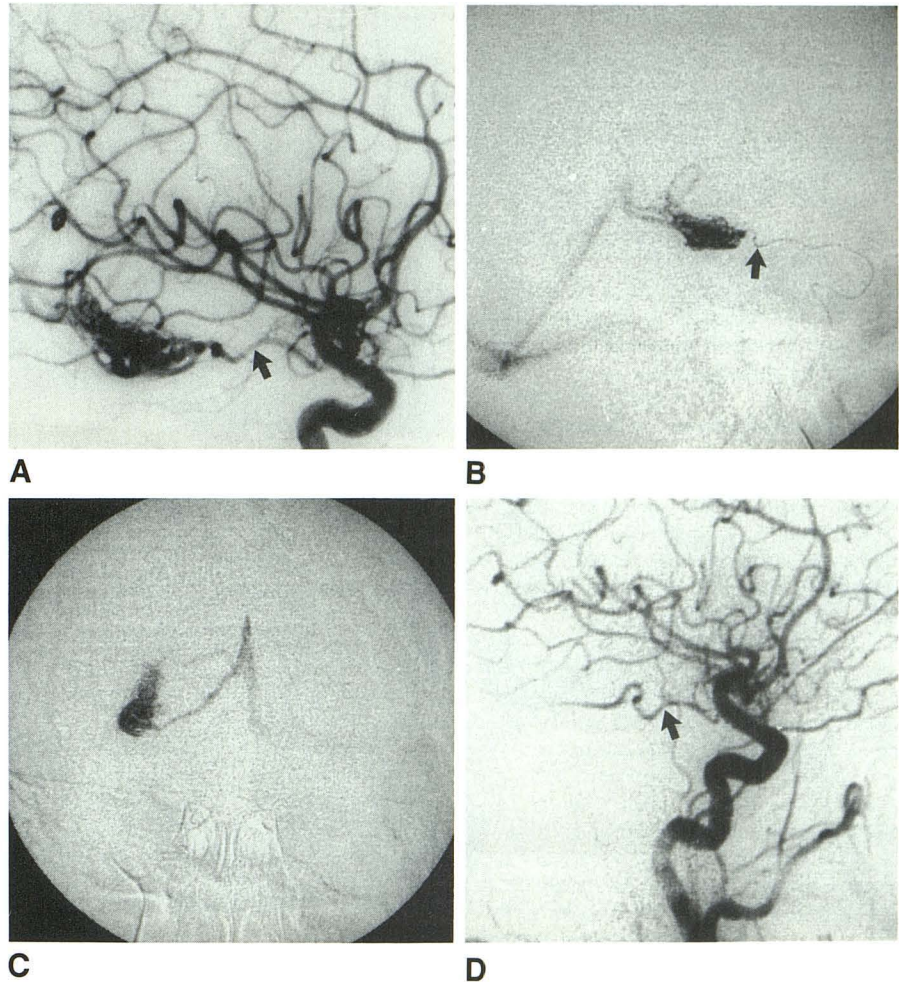
C, Injection of correctly positioned catheter shows opacification only of arteriovenous malformation fed by ventricular portion of AChA.

Fig. 2.—Case 2.

A, Right carotid angiogram, lateral projection. Preembolization angiogram shows temporal lobe arteriovenous malformation fed by dilated anterior choroidal artery (AChA) (arrow).

B and **C**, Superselective lateral (**B**) and anteroposterior (**C**) angiograms show placement of microcatheter in right AChA prior to embolization. Catheter tip has passed plexal point (arrow).

D, Right lateral carotid angiogram 1 year after embolization shows continued lack of filling of malformation from persistently dilated AChA (arrow). Filling of AVM through posterior lateral choroidal artery (not shown). The patient was referred for radiosurgery.



recovery and residual hemidystonia, right inferior quadrantanopia, and alexia [13].

Discussion

The AChA irrigates regions in both the traditional anterior and posterior circulations, supplying diverse structures such as the optic tract, lateral geniculate body, optic radiation, genu and posterior limb of the internal capsule, and portions of the medial temporal lobe and lenticular structures. As a result, surgical resection of lesions irrigated by this artery can lead to a variety of severe and unpredictable postoperative neurologic deficits. For this reason, we consider AVMs fed predominantly by the choroidal arteries to be unresectable, and thus have sought alternative methods of treatment.

When the ostium of the AChA is dilated, as it was in all our cases, we found it relatively easy to catheterize. With the simple catheter system described, including the small distal dilatation, the catheter usually could be advanced far enough beyond the plexal point to enable safe and successful embolization. In the one case where this was not true, we were able to alter the flow in the artery enough with a balloon situated at the ostium to cause the catheter to advance.

The importance of correct catheter positioning and changes in flow dynamics during the course of an embolization cannot be overemphasized. After our early disastrous result, which occurred at the beginning of our experience with the Pursil catheter system, we were reluctant to perform further catheterizations of this important artery. However, with an understanding of the functional neuroanatomy supplied by the AChA, more confidence in our catheterization technique, and superselective Amytal testing, we have had no further complications.

Discussion of complications of AChA embolization must include vascular perforations secondary to catheterization (Dowd et al., Symposium Neuroradiologicum XIV, London, June 1990). In our entire extensive experience with over 1000 superselective catheterizations using the Pursil catheter system without guidewires, we have had no vessel perforations.

The reduction in the Spetzler and Martin AVM grade [9] was sufficient to allow stereotaxic irradiation of all the AVMs. This rendered all these untreatable AVMs treatable and potentially curable. At this time, postradiation follow-up has been inadequate in all but the first patient treated, in whom partial obliteration of the AVM has been achieved.

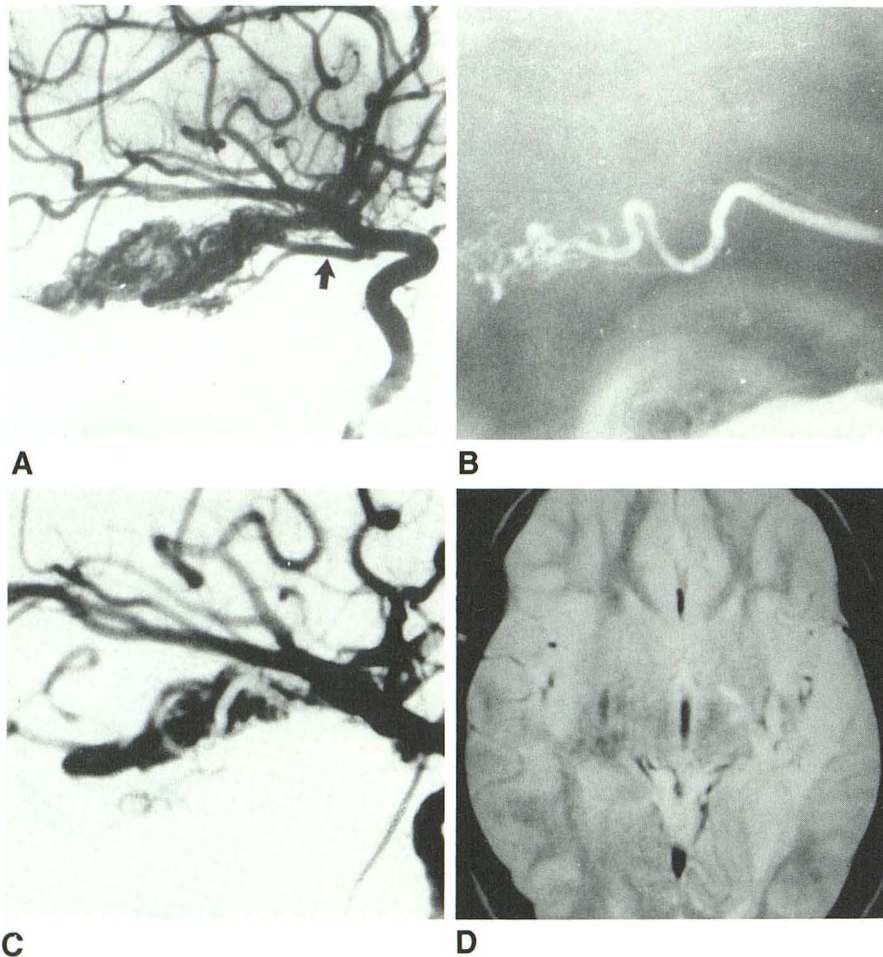


Fig. 3.—Complication (case 1).

A, Left carotid angiogram, lateral projection. Preembolization angiogram shows temporal lobe arteriovenous malformation fed by dilated anterior choroidal artery (AChA) (arrow).

B, Lateral skull film. Cast of isobutyl 2-cyanoacrylate is in entire AChA.

C, Postembolization left carotid angiogram shows reduction in nidus of arteriovenous malformation.

D, T2-weighted axial MR image, 2300/100 (TR/TE), 4 years after embolization. Increased signal intensity is seen in internal capsule.

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