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Progressive Thrombosis of Brain Arteriovenous Malformations after Embolization with Isobutyl 2-Cyanoacrylate

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Embolization of brain arteriovenous malformations (AVMs) with isobutyl 2-cyanoacrylate (IBCA) is an alternative to surgical treatment when dealing with large AVMs with multiple arterial feeders. The deposition of IBCA in the nidus of the AVM may produce an active and progressive thrombosis that may lead to complete occlusion of the nidus and/or to progressive thrombosis of the draining veins. Four clinical examples of progressive thrombosis after IBCA embolization are demonstrated, including two cases in which late follow-up angiography showed complete obliteration of a partly embolized AVM.

The techniques of transfemoral and/or intraoperative embolization of brain arteriovenous malformations (AVMs) with isobutyl 2-cyanoacrylate (IBCA) have been described [1-5]. The aim of this type of embolization should be complete occlusion of the nidus of the AVM. Proximal occlusion of arterial feeders without embolizing the AVM is not only insufficient but disadvantageous [6].

If, by using the transfemoral technique, the calibrated-leak balloon cannot be positioned close enough to the nidus of the AVM to deliver the IBCA, this procedure should be discontinued and an intraoperative embolization should be considered [3, 7]. This technique allows surgical isolation and catheterization of cortical arterial feeders as close as possible to the AVM. It may be performed under local anesthesia in the awake patient when the AVM involves vital areas of the brain [7].

In vitro studies (Manelfe C, unpublished data) have shown that IBCA in contact with plasma and/or blood produces an active and expanding clotting process. This phenomenon may play a role in the progressive postembolization thrombosis that we observed in four cases from a group of 57 cases of brain AVMs embolized with IBCA. This report discusses these four cases, two with eventual complete AVM obliteration and two with prominent progressive thrombosis of the venous outflow after embolization.

Case Reports

Case 1

A 34-year-old man was admitted with temporal lobe seizures. Current seizure medications were questionably effective and produced severe drowsiness. Cerebral angiography showed a large AVM in the right temporal lobe. The AVM was fed by a single anterior temporal branch of the right posterior cerebral artery (fig. 1A). Transfemoral embolization with IBCA was undertaken. With the patient under neuroleptic analgesia, a no. 5.8 Elecath introducer was positioned in the right internal carotid artery. A no. 17 latex calibrated-leak balloon (Ingenor, Paris) glued to a Silastic microcatheter (Cook, Bloomington, IN) was injected through the introducer and positioned in the arterial feeder of the AVM, 2 cm from the nidus. A preembolization angiogram was obtained to check the position of the balloon, to depict the filling of normal arterial branches, and to measure the arteriovenous transit

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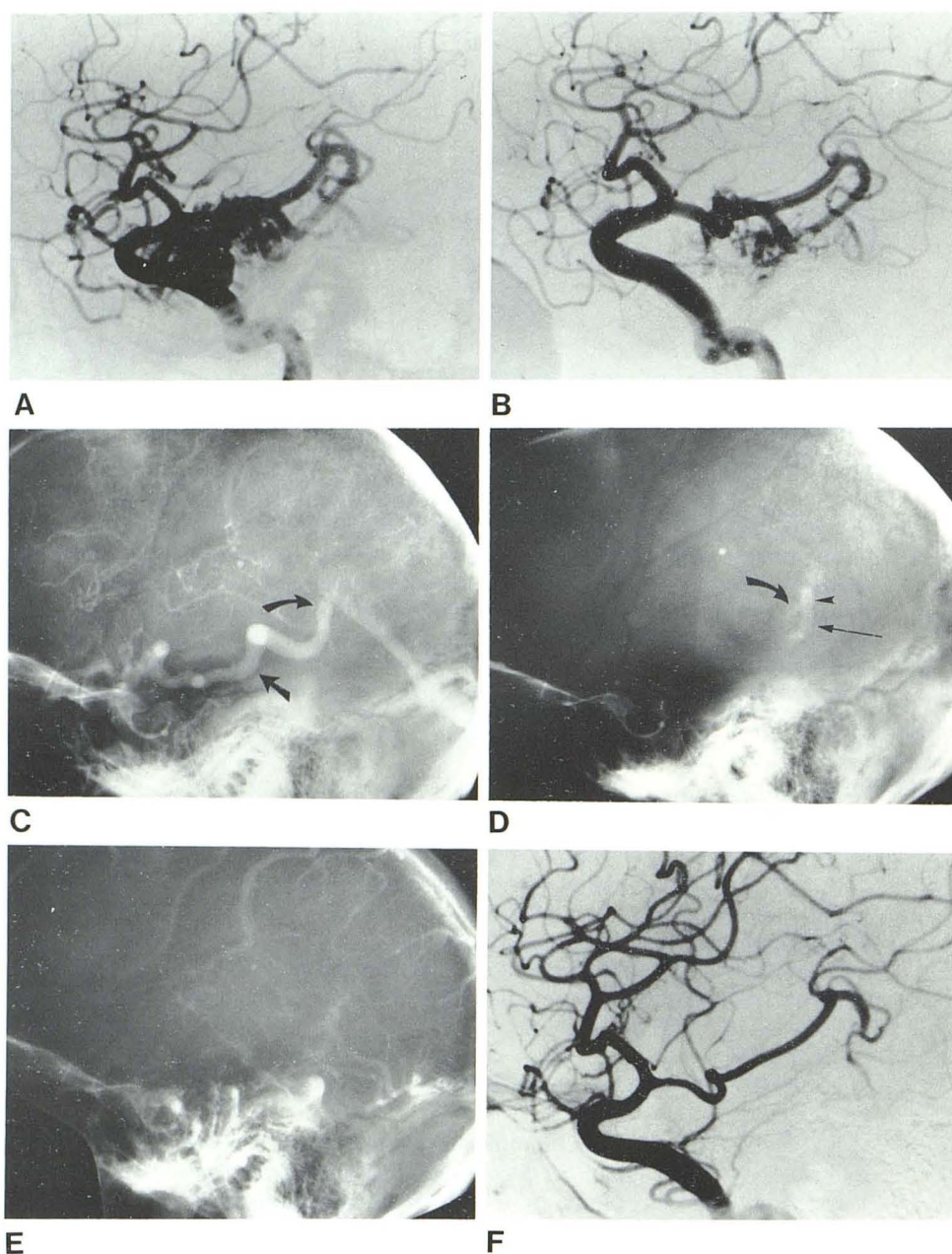


Fig. 1.—Temporal lobe AVM. Partial embolization of nidus with thrombosis of draining veins. Subsequent complete obliteration of AVM. **A**, Right internal carotid arteriogram, lateral view. Temporal AVM supplied by enlarged anterior temporal branch of right posterior cerebral artery. **B**, Immediately after embolization with IBCA. Obliteration of about two-thirds of AVM nidus. Patency of right anterior temporal feeder. **C**, Preembolization, venous phase. Enlarged basal vein of Rosenthal (*straight arrow*) draining into vein of Galen (*curved arrow*) and straight sinus. **D**, After embolization. Radiopaque cast of IBCA deposit at junction of base of vein of Rosenthal (*straight arrow*), internal cerebral vein (*curved arrow*), and vein of Galen (*arrowhead*). **E**, Immediately after embolization. Venous phase. Blood stagnation in AVM and proximal part of basal vein of Rosenthal. Nonfilling of distal aspect of basal vein of Rosenthal, vein of Galen, and internal cerebral vein. **F**, Right internal carotid arteriogram 1 week after embolization. Complete obliteration of AVM nidus and distal part of right anterior temporal artery. Preservation of blood supply to visual pathways. The patient remained neurologically intact.

time. The arteriovenous transit time was about 2.5 sec.

The AVM was embolized with 0.9 ml of a mixture of 1 g of tantalum powder, 1 ml of IBCA, and 0.6 ml of iophendylate [8]. An immediate postembolization angiogram showed occlusion of about two-thirds of the nidus of the AVM with significant decrease in flow (fig. 1B); there was also evidence of deposit of IBCA occluding the vein of Galen (figs. 1C–1E). Right common carotid and left vertebral angiograms 1 week later showed complete obliteration of the nidus of the AVM and occlusion of the arterial feeder (fig. 1F). The change in caliber of the right posterior cerebral artery probably represented the effect of decreased flow through the AVM [9]. The patient remained neurologically unchanged throughout the hospital stay and was still seizure-free after 6 months. He continued on the same anticonvulsant medical therapy as before embolization.

Case 2

A 38-year-old man was admitted with episodes of right-sided headache accompanied by temporary bilateral visual loss. Angiography after a subarachnoid hemorrhage 6 months before revealed a right posterotemporal AVM fed by the right posterior cerebral artery. A large varix, part of the AVM's venous drainage, was observed inferior to the nidus of the AVM (fig. 2A). The transfemoral approach was selected. The arteriovenous transit time measured on the preembolization superselective angiogram was about 2 sec.

The AVM was embolized with 0.7 ml of a mixture of 1 g of tantalum powder, 1 ml of IBCA, and 0.5 ml of iophendylate [8]. Immediate postembolization angiography showed obliteration of about 50%–75% of the nidus of the AVM. The large varix was still

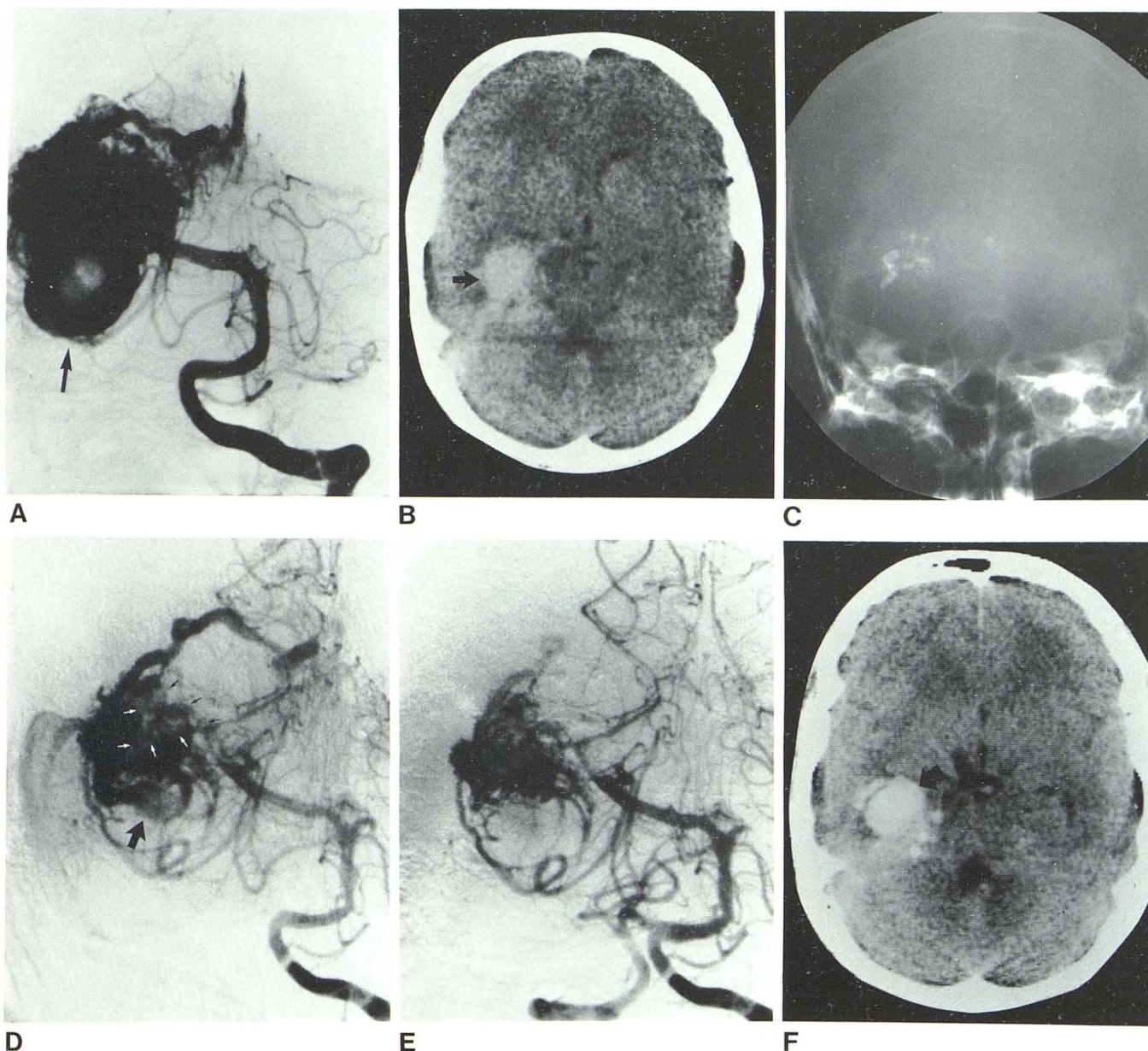


Fig. 2.—Temporal lobe AVM with venous varix. Delayed occlusion of varix. Incomplete obliteration of AVM. A, Left vertebral angiogram. Right mesiotemporal AVM with large venous varix (arrow). B, Preembolization noncontrast CT scan. Area of slightly increased density projects deeply into right temporal lobe (arrow). C, Postembolization IBCA deposit in the temporal AVM. D, Immediate postembolization angiogram. Obliteration of 60%–70%

of AVM nidus. IBCA has been deposited in superior medial aspect of AVM nidus (small arrows). Slow filling of partially thrombosed venous varix (large arrow). E, 1 week after embolization. Remaining nidus of AVM unchanged in size but complete thrombosis of large venous varix has occurred. F, Noncontrast CT scan 1 week after embolization shows increased density produced by thrombus filling venous varix (arrow).

visualized (fig. 2D). A repeat angiogram 1 week later showed further diminution of the AVM and nonfilling of the varix (fig. 2E). Comparison of the pre- (fig. 2B) and post- (fig. 2F) embolization noncontrast computed tomographic (CT) scans showed a homogeneous high density consistent with thrombosis of the large varix. The absence in this latter scan of CT artifacts produced by the deposit of tantalum powder-impregnated IBCA (fig. 2D) proves that the varix was not embolized accidentally.

At the time of discharge the patient had a superior homonymous quadrantanopia and Parinaud syndrome. Six months later only a residual left superior quadrantanopia remained. It may be postulated

that the postembolization neurologic deficit was produced by IBCA occlusion of normal arteries (visual field defect) and mass effect of the thrombosing varix on the corpora quadrigemina (Parinaud syndrome). Shrinkage of the thrombosed varix with time may explain the disappearance of the Parinaud syndrome.

Case 3

A 36-year-old man was admitted with fixed and dilated pupils due to an intraventricular hemorrhage. He was treated by left ventricular drainage, which was subsequently changed to a shunt.

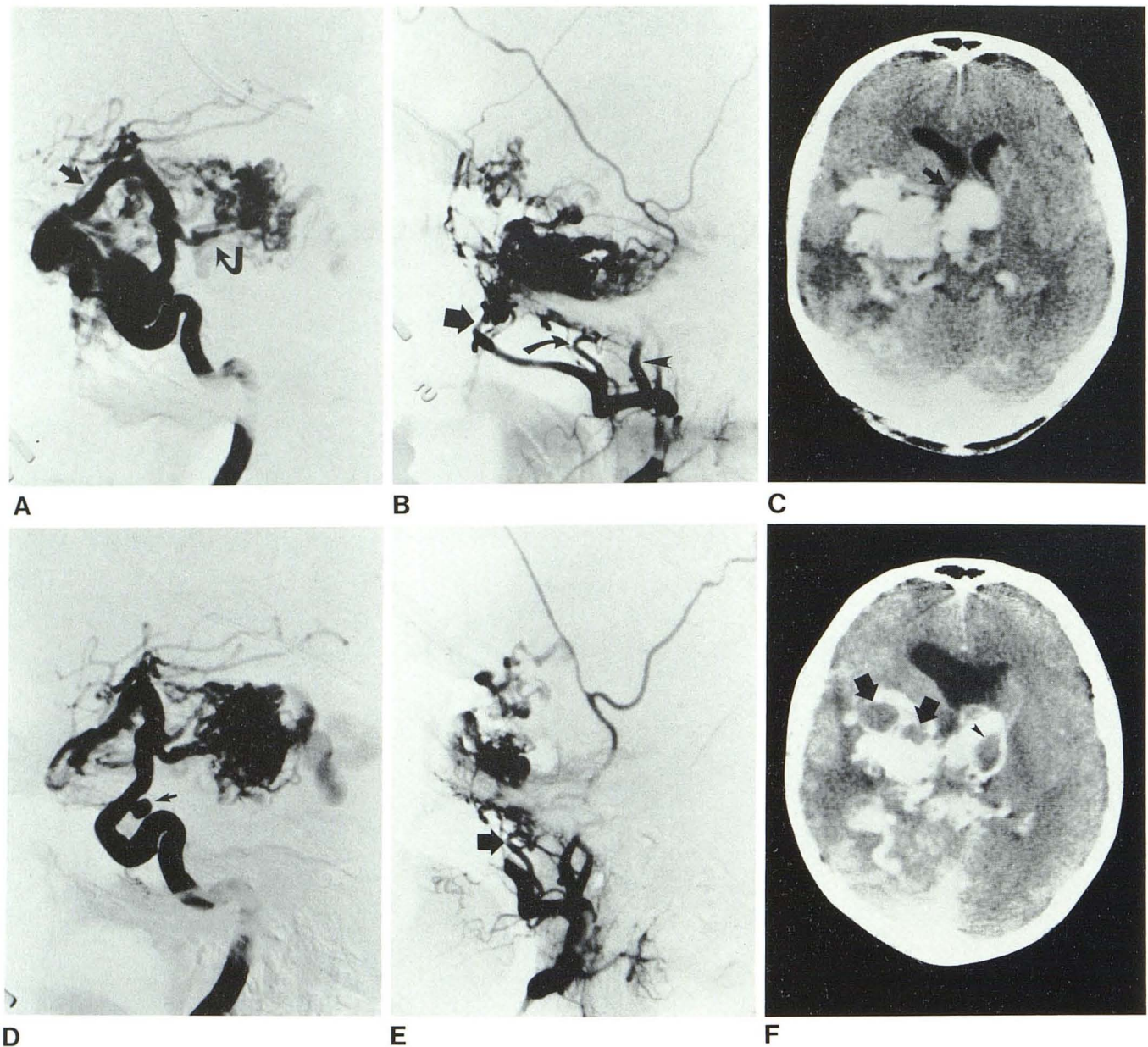


Fig. 3.—Middle fossa dural AVM. Postembolization thrombosis of venous varix causing hydrocephalus. **A**, Preembolization lateral view of right internal carotid angiogram. Anterior aspect of AVM supplied by right anterior temporal artery (*straight arrow*). Posterior part supplied mainly by anterior choroidal artery (*curved arrow*). **B**, Preembolization internal maxillary arteriogram. Middle fossa dural AVM supplied by internal maxillary (*straight arrow*), accessory meningeal (*curved arrow*), and middle meningeal (*arrowhead*) arteries. **C**, Preembolization enhanced CT scan. Large, deep venous varix compresses third ventricle (*arrow*). **D**, Postembolization carotid angiogram.

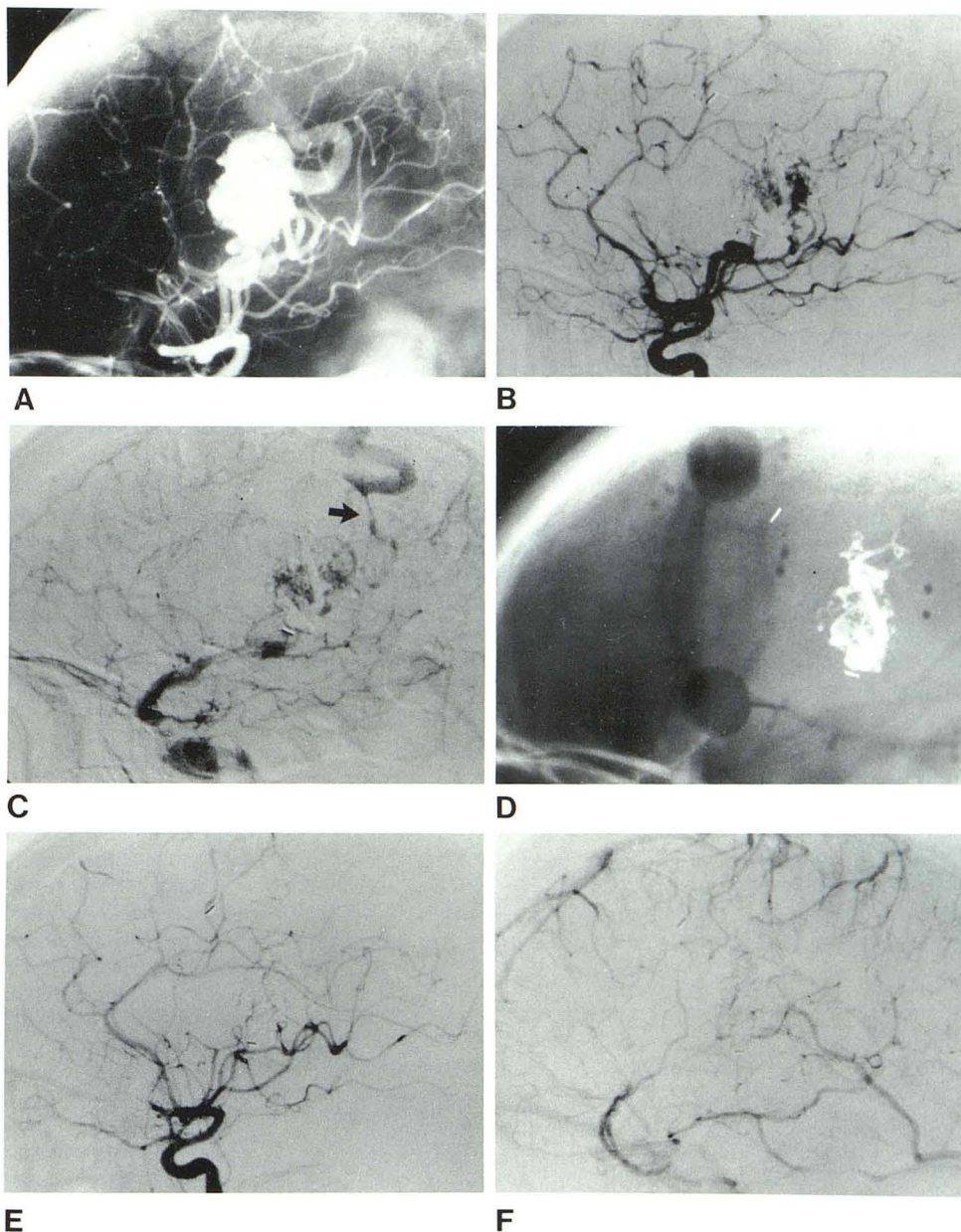
Obliteration of anterior part of AVM and distal aspect of right temporal artery. Shift of arterial flow to posterior aspect of nidus of AVM. Discovery of paraophthalmic aneurysm previously obscured by AVM nidus (*arrow*). **E**, Postembolization internal maxillary arteriogram. Residual AVM mainly supplied by accessory meningeal artery (*arrow*). **F**, Enhanced CT scan 2 weeks after embolization. Two large filling defects in nidus of AVM (*arrows*) are produced by progressive AVM thrombosis. Venous varix is larger and has a thrombus (*arrowhead*). Ventricles are dilated.

Neurologic examination revealed him to be arousable only by painful stimuli. Angiography showed an AVM involving the right frontal and temporal lobes. Part of the nidus was supplied by the anterior temporal artery and part by an enlarged anterior choroidal artery (fig. 3A). Several serpiginous varicose veins drained into the superficial and deep venous systems. A selective right external carotid angiogram showed blood supply to the AVM from the right internal maxillary, accessory meningeal, and middle meningeal arteries (fig. 3B). Preembolization CT showed deep-seated large varices compressing the third ventricle and producing moderate hydrocephalus

(fig. 3C). The right anterior temporal artery was selectively catheterized with a no. 17 calibrated-leak balloon. The arteriovenous transit time measured on the preembolization superselective angiogram was about 1 sec.

The AVM was embolized with 0.6 ml of a mixture of 1 g of tantalum powder, 1 ml of IBCA, and 0.025 ml of iophendylate. The patient had slight improvement of his level of consciousness after this first embolization. Two days later the internal maxillary artery and middle meningeal artery were embolized with injections of 0.25 ml and 0.2 ml of IBCA, respectively. Postembolization angiography

Fig. 4.—Rolandic AVM. Partial thrombosis of draining vein after embolization. Subsequent complete occlusion of AVM. **A**, Preembolization left internal carotid angiogram shows left rolandic AVM supplied by suprasylvian left middle cerebral artery feeders. **B** and **C**, 1 week after IBCA embolization. Occlusion of one-half to two-thirds of AVM nidus. Irregular narrowing of proximal part of cortical draining vein produced by IBCA embolization (arrow). **D**, Skull shows location of radiopaque IBCA and rolandic AVM feeders and nidus. **E** and **F**, 6 months after embolization. Complete occlusion of AVM.



showed occlusion of the right temporal artery and the frontal part of the AVM (fig. 3D). Substantial decrease in blood supply from the external carotid artery was observed as well (fig. 3E). Contrast-enhanced CT scans then showed low densities within the AVM and a number of varices, implying progressive thrombosis (fig. 3F). The patient worsened after the second embolization with the development of increased obstructive hydrocephalus produced by enlargement of the partly thrombosed varix compressing the third ventricle, necessitating a second shunt on the right. Embolization of the remaining part of the AVM, which arose mainly from the anterior choroidal artery, was deferred. Unfortunately, the patient suffered another intracerebral hemorrhage about 4 weeks after the last embolization and died the next month.

Case 4

A 37-year-old woman was admitted with a history of grand mal seizures difficult to control medically. The patient was neurologically

intact. Cerebral angiography showed a left rolandic AVM supplied by two left middle cerebral artery feeders (fig. 4A). Serpiginous cortical veins drained into the superior sagittal sinus. Embolization with IBCA was carried out directly through a surgical craniotomy. The largest cortical arterial feeder was identified, isolated, and cannulated with a 3 French catheter (Cook). Preembolization angiography under fluoroscopic control and videorecording were performed to check the position of the catheter and to measure the arterial/venous transit time. The arteriovenous transit time was 1.5 sec. The AVM was embolized with 2.9 ml of a mixture of 1 g of tantalum powder, 1 ml of IBCA, and 0.2 ml of iophendylate. Intraoperative postembolization angiography showed occlusion of about two-thirds of the AVM. Twelve hours after embolization the patient became dysphasic and developed a right hemiplegia. CT showed an acute intracerebral hemorrhage anterior to the IBCA cast. Cerebral angiograms 1 week later showed obliteration of about two-thirds of the AVM and also IBCA deposition in a cortical draining vein (figs. 4B and 4C). No significant improvement was observed during that time.

Angiography 6 months later showed complete obliteration of the AVM (figs. 4E and 4F). The patient had a slow though steady recovery; a mild expressive dysphasia and weakness of the right arm remained.

Discussion

Transfemoral, intraoperative, or combined techniques of embolization of brain AVMs with IBCA should be directed toward potential complete occlusion of the nidus of the AVM [3, 7]. Liquid IBCA may reach the core of the AVM before it hardens in contact with blood. The success of embolization depends on the proximity of the IBCA delivery (transfemoral calibrated-leak balloon or intraoperative catheter) to the nidus of the AVM and proper selection of the polymerization time of the IBCA [3, 7, 8]. This essential information is obtained from the arteriovenous transit time determined from the preembolization superselective angiogram [7].

The examples of postembolization thrombosis of AVMs described here may be explained on the basis of the phenomenon of rapid, active, and expanding thrombosis described by Manelfe (Manelfe C, unpublished data) when blood comes in contact with IBCA. It is very important that this phenomenon occurs in the core of the AVM. This supports the concept of performing the IBCA embolization as close as possible to the AVM [3, 7]. A proximal embolization increases the risk of occlusion of normal cortical arteries. On the other hand, the closer the delivery system to the nidus, the higher the risk of embolizing the venous drainage with potentially disastrous effects [7].

When embolization is incomplete but a significant part of the nidus has been occluded with substantial decrease in flow through the AVM, the process of active thrombosis produced by the deposition of IBCA may continue and the nidus may become completely obliterated. The process of active thrombosis may extend distally to involve the draining veins of the AVM. This occurred in case 3, where the thrombosis and enlargement of a deep varix produced obstructive hydrocephalus by compressing the third ventricle.

Cases 1 and 4 are examples of initial partial obliteration of the nidus of an AVM, further active thrombosis, and final complete occlusion of the AVM. Both cases also had accidental embolization of the venous drainage. It may be postulated that the combination of decreased arterial flow accompanied by sluggishness of the venous outflow facilitates the process of continuing thrombosis of the nidus. Despite the embolization of the venous outflow, considered a technical complication, neither case 1 nor 4 had any immediate clinical neurologic deterioration, though partial embolization of the venous drainage in case 4 possibly played a role in the production of the delayed hematoma 12 hr later. It is also possible that the hemorrhage destroyed part of the malformation. Cases 2 and 3 did not have angiographic evidence of accidental venous embolization but did show progressive thrombosis of both the nidus and the draining veins of the AVM. Presumably the ongoing thrombosis was secondary to stasis produced by the occlusion of a substantial part of the AVM.

This phenomenon of postembolization progressive thrombosis of a vascular lesion due to blood stagnation has also

been described by Latchaw and Gold [10] and Tadavarthy et al. [11] after embolization with polyvinyl alcohol (PVA). These authors also describe PVA as promoting progressive fibrosis by forming a scaffold for the ingrowth of connective tissue that incorporates the PVA, making it an integral part of the body. Vinters et al. [12] described a chronic inflammatory response in the vessel walls and adjacent brain parenchyma after embolization with IBCA. They postulate that it may be abnormal permeability of the vascular channels within the malformation that facilitates an inflammatory reaction to IBCA. This regional inflammatory reaction indeed may accelerate the process of thrombosis of the pathologic vessels within the AVM.

In summary, embolization of brain AVMs with IBCA may produce a progressive thrombosis as a process initiated by incomplete occlusion of the nidus of the AVM. The mechanisms may be direct, active thrombogenic effect of IBCA, substantial decrease in flow and pressure through the AVM, and reactive chronic inflammation involving occluded vessels and surrounding brain tissue produced by IBCA. This progressive thrombosis may extend into draining veins resulting from significant decrease in blood flow and/or stasis that develops in the AVM outflow when a substantial part of the nidus has been occluded.

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