Endovascular occlusion of intracranial vessels for curative treatment of unclippable aneurysms: report of 16 cases

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Among 121 intracerebral aneurysms presenting at one institution between 1984 and 1989, 16 were treated by endovascular means. All 16 lesions were intradural and intracranial, and had failed either surgical or endovascular attempts at selective exclusion with parent vessel preservation. The lesions included four giant middle cerebral artery (MCA) aneurysms, one giant anterior communicating artery aneurysm, six giant posterior cerebral artery aneurysms, one posterior inferior cerebellar artery aneurysm, one giant mid-basilar artery aneurysm, two giant fusiform basilar artery aneurysms, and one dissecting vertebral artery aneurysm. One of the 16 patients failed an MCA test occlusion and was approached surgically after attempted endovascular selective occlusion. Treatment involved pretreatment evaluation of cerebral blood flow followed by a preliminary parent vessel test occlusion under neuroleptic analgesia with vigilant neurological monitoring. If the test occlusion was tolerated, it was immediately followed by permanent occlusion of the parent vessel with either detachable or nondetachable balloon or coils.

The follow-up period ranged from 1 to 8 years. Excellent outcomes were obtained in 12 cases with complete angiographic obliteration of the aneurysm and no new neurological deficits and/or improvement of the preembolization symptoms. Four patients died: two related to the procedure, one secondary to rupture of another untreated aneurysm, and the fourth from a postoperative MCA thrombosis after having failed endovascular test occlusion. The angiographic, clinical, and cerebral blood flow criteria for occlusion tolerance are discussed.

Key Words • endovascular treatment • aneurysm • balloon occlusion

Selective complete exclusion from the cerebral circulation, either by surgical or endovascular techniques, is the optimal treatment for intracranial aneurysms. However, some inaccessible giant saccular or fusiform aneurysms cannot be treated by such conventional means. Until recently, they have required either complicated intracranial revascularization techniques7,12,13 or carotid or vertebral parent artery occlusion.4,5,32 If a direct surgical approach is chosen, it usually involves surgical exploration of the aneurysm and placement of a tourniquet around the parent vessel which, after the patient has recovered from anesthesia, is later closed with angiographic and clinical control in the radiology department.4,20 The advent and subsequent rapid development of intravascular navigation techniques and microballoon technology1,3,5,6,9,11,19,23-26 have allowed us to develop a new procedure. Instead of surgical exploration in these cases, endovascular occlusion of the parent intradural blood vessel is accomplished in one step in the awake patient, with constant neurological monitoring and arteriographic confirmation of adequate collateral blood supply.

Clinical Material and Methods

Angiography and Embolization Techniques

Neuroleptic analgesia was used in awake patients for all angiographic procedures in this series, which permitted vigilant clinical observation. Four-vessel angiography was carried out via the femoral approach to confirm collateral circulation from the carotid arteries and contralateral vertebral artery. Direct puncture of either the axillary or common carotid artery with a No. 16 catheter allowed the introduction of a balloon system. A detachable latex valve balloon was mounted on the tip of a No. 1 French polyethylene (PE) microcath-
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eter, which was coaxially contained within a No. 2.5 French microcatheter,* itself coaxial within a No. 3.6 French guiding PE catheter.23,24 In cases where balloon detachment was thought to be hazardous, such as where traction caused pain or where there was judged to be a risk of balloon displacement during detachment, the inflated microballoon was not detached. Rather, it was left in place, inflated, and the proximal end of the No. 1 French microcatheter was buried under the skin at the puncture site.19,24

Balloons were inflated with 3 nonionic water-soluble contrast medium (Iopamidol 300) and 1 normal saline. The thin, perfectly coaxial catheters with negligible dead space are only slightly thrombogenic, thus no heparin was used. Other materials used were embolization microcoils for vessel occlusion.†

Pretreatment Cerebral Blood Flow Studies

Cerebral blood flow (CBF) was measured before and after injection of 1 gm acetzolamide (Theraplix) in the pre-embolization work-up of all patients. Injection of 70 mCi 133Xe in 10 cc of saline over 10 seconds was performed with simultaneous measurement of pulmonary radioactivity. Three axial single-photon emission computed tomography (SPECT) scans were obtained during the wash-out phase at the level of the corona radiata, thalamus and internal capsule, temporal lobes, and posterior fossa. Before 1987, a similar protocol using six Na I detectors for each hemisphere was performed. Local rates of CBF were calculated using a monocompartmental model.51

Test Occlusion Protocol and Balloon Placement

Middle Cerebral Artery Aneurysms. The treatment protocol depended upon the location of the aneurysm. In aneurysms involving the M1 (three cases) or M2 (one case) segments of the middle cerebral artery (MCA), test occlusion involved selective catheterization with the balloon-bearing No. 1 French microcatheter and proximal occlusion by balloon inflation. The patient’s neurological condition was carefully monitored by a trained clinician during the test occlusion. Ipsilateral carotid and vertebral arteriography was performed to assess collateral blood supply. Only when complete collateral retrograde anastomotic revascularization was demonstrated and perfect clinical tolerance of vessel occlusion was documented for 30 minutes was the balloon left in place.

In treatment of the M1 segment aneurysm, a microcatheter was placed just proximal to the aneurysm and 50 mg of sodium Amytal (amobarbital) was injected; when this caused no deficit, the parent vessel was safely occluded with autologous blood clot. In one case of M1 aneurysm, the balloon was placed partially within the aneurysm occluding the parent vessel during the first treatment. Four years later, during retreatment, the test occlusion balloon was withdrawn and the MCA was occluded with two platinum minicoils.

Posterior Cerebral Artery Aneurysms. Six posterior cerebral artery (PCA) aneurysms were treated with a similar protocol. In four cases in which the aneurysms arose at the PCA origin, the balloon was inflated in the proximal P1 segment, and for two P2 aneurysms, it was inflated in the P2 segment. During test occlusion, angiographic injection of the ipsilateral carotid artery was performed to demonstrate the posterior communicating artery (P1 occlusion) and leptomeningeal collateral supply from the anterior cerebral artery and the MCA.

Anterior Communicating Artery Aneurysm. A nondetachable balloon was placed in the left A1 segment of the anterior cerebral artery of a patient with a giant partially thrombosed anterior communicating artery aneurysm, and was inflated for 30 minutes with good clinical tolerance. Continued filling of the aneurysm through the hypoplastic right A1 segment was apparent, and complete filling of the right A1 territory through leptomeningeal collaterals was demonstrated. The balloon was deflated. Because of continued filling from the right A1, the aneurysm was filled with platinum minicoils, and the left A1 segment was also occluded with a single minicoil.

Infratentorial Circulation Aneurysms. One patient with a distal intracranial dissecting fusiform vertebral artery aneurysm was treated with intracranial vertebral artery occlusion distal to the origin of the posterior inferior cerebellar artery (PICA).25 This approach was used because the segment of vertebral artery distal to the PICA and proximal to the aneurysm was not more than 5 mm, which allowed sufficient space to inflate the balloon without occluding the PICA.1 Two cases of giant fusiform aneurysm of the basilar artery were successfully treated: one by occlusion of the origin of the basilar artery, and the other by occlusion of the intracranial vertebral artery distal to the PICA and proximal to its origin. A single case of giant saccular aneurysm of the middle third of the basilar artery was treated by mid-basilar artery occlusion. In all three cases of basilar artery aneurysm, collateral blood supply was confirmed by injection of both carotid arteries which filled the superior portion of the basilar artery through the posterior communicating arteries. Also, injection of the ipsilateral and contralateral vertebral arteries demonstrated normal filling of both PCAs.

In one patient, the right PICA was occluded for treatment of two small aneurysms. In this case, premature permanent occlusion occurred during test occlusion (see illustrative Case 2).

* Pur-Sil microcatheter manufactured by Balt Co., Paris, France.
† Hilal embolization microcoil manufactured by Cook Inc., Bloomington, Indiana.
‡ Tomomatic 64 SPECT system manufactured by Medimatic, Copenhagen, Denmark.

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Illustrative Case Reports

Case 1: Occlusion of the Right PCA

This 49-year-old man suffered subarachnoid hemorrhage (SAH) in August, 1984. The sequelae of the SAH included a transient left hemiparesis and hemisensory deficit, moderate short-term memory problems, and severe headaches. A computerized tomography (CT) scan revealed a giant calcified, high-density, partially contrast-enhancing mass in the right retrosplenial cistern. Angiography confirmed the presence of a partially thrombosed right P2 aneurysm (Fig. 1a). The patient underwent an unsuccessful attempt at operative clipping in November, 1984. A repeat SAH occurred in March, 1985, with aggravation of his left hemisensory and memory deficit, and a transient right hemiparesis and left homonomous hemianopsia.

Endovascular treatment was first attempted in July, 1985, with accidental placement of a detachable balloon in the right PCA, causing PICA occlusion and a transient 24-hour right cerebellar syndrome. In December, 1985, a small latex balloon on a No. 1 French microcatheter was passed via a left axillary approach into the basilar artery. Unsuccessful attempts were made to pass the balloon into the right PCA, until a second nondetachable balloon was navigated from the femoral approach into the left PCA and was inflated temporarily, causing the occcluding balloon to be flow-directed into the right PCA. Inflation of the balloon in the aneurysm itself was insufficient to exclude it from the circulation (Fig. 1b). The balloon was withdrawn into the proximal P2 segment and inflated for a 30-minute test occlusion, which was well tolerated (Fig. 1c). The position of the balloon in the proximal P2 segment just distal to the PCA bifurcation was thought to be critical and the risk of any inadvertent displacement with detachment maneuvers was avoided by leaving the catheter attached to the balloon. The proximal end of the catheter was occluded and buried under the skin in the axilla under sterile conditions. Slow filling of the aneurysm through right MCA collaterals was apparent during the test occlusion and in the 3-month follow-up angiogram (Fig. 1d). However, by 6 months postembolization no aneurysm filling was seen. At last follow-up review (40 months after treatment) in April, 1989, the patient had resumed his normal life, with only a mild nondebilitating memory deficit; the aneurysm remains excluded from the cerebral circulation (Fig. 1e).

Case 2: Occlusion of the Right PICA

This 67-year-old man with chronic obstructive lung disease suffered an SAH in September, 1989. A CT scan was normal, except for a finding of blood in the foramen magnum. Arteriography demonstrated two aneurysms of the right PICA: a large one at the origin and a small one located more distally along the same PICA (Fig. 2 left). Because of his lung disease, the patient was not considered a surgical candidate and was referred for endovascular therapy. In the following 4 weeks, the patient developed a wide-based gait and memory problem. A repeat CT scan showed mild ventriculomegaly and a small left subdural effusion. These CT and clinical

Fig. 1. Angiograms in Case 1 with a posterior cerebral artery (PCA) aneurysm. a: Left vertebral angiogram obtained prior to a failed operative attempt demonstrating an irregularly shaped aneurysm of the right P2 segment. There is no definable neck. b: Right vertebral angiogram during endovascular treatment. The nondetachable balloon has been placed inside the aneurysm lumen and inflated maximally without aneurysm exclusion. c: Right vertebral angiogram following balloon occlusion of the proximal right P2 segment of the right PCA. The catheter was left attached to the balloon and the proximal end was buried under the skin. d: Right retrograde brachial angiogram, late arterial phase, obtained 3 months after endovascular treatment, showing persistent P2-segment occlusion. There is continued filling of the aneurysm by leptomeningeal collaterals in the early venous phase of the injection. e: Left vertebral angiogram obtained 40 months after treatment confirming persistent P2 occlusion and aneurysm exclusion with no demonstrable angiographic change in the left vertebral artery carrying the nondetached catheter.
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findings resolved over a 2-week period with low-dose corticosteroid therapy. Endovascular therapy was performed in August, 1989, with the intention of achieving a right PICA occlusion, as the aneurysms were thought to be too small for selective treatment. Through a right axillary approach, a small nondetachable latex balloon was navigated into the right PICA origin. Balloon inflation and deflation caused spontaneous premature right PICA occlusion. After treatment, the patient developed a mild aggravation of his gait disturbance which resolved by his 2-month postembolization examination.

Follow-up findings at 6 months revealed return to normal intellectual functioning and a normal gait. Angiography at that time showed persistent occlusion of the right PICA, with continued exclusion of the aneurysms and complete collateral filling of the PICA territory by branches of the anterior inferior and superior cerebellar arteries (Fig. 2 right).

Summary of Results

Treatment Outcome

The follow-up period in these 16 patients ranged from 1 to 8 years (mean 3.5 years). Excellent clinical results were obtained in 12 patients, including complete angiographic exclusion of the aneurysm lumen with stable or improved neurological status after treatment. These patients either remained normal (if asymptomatic) or improved to a normal or nearly normal nondebilitated state. Some of these patients had transient mild neurological deterioration following treatment that had resolved completely at last follow-up examination. Endovascular treatment failed in one patient, who became hemiplegic 1 minute after test occlusion performed 3 days after SAH. The balloon was immediately deflated and the neurological condition returned to normal. After aneurysm clipping, MCA thrombosis occurred leading to the patient’s death.

Angiography showed recanalization of the parent vessel in four patients. One of these patients developed new aneurysms of the parent segment of the MCA, opposite the previously treated cured aneurysm, 4 years after treatment (see below). In eight of the 15 successfully treated patients there was no evidence of arterial dysplasia in the vessel carrying the nondetached microcatheters 1 to 8 years after treatment.

Complications

Deaths. Three patients died subsequent to their endovascular treatment. One patient died as a direct result of balloon occlusion of the parent vessel. This patient developed hemiplegia and oculomotor paralysis 2 hours after balloon occlusion of the mid-basilar artery, which transiently improved with heparinization, but culminated in coma and death 5 days later. A CT scan prior to death showed intrapontine hemorrhage.

Treatment resulted in the death of a patient with a giant fusiform aneurysm of the basilar artery. This 50-year-old man presented with a 6-month history of progressive left hemiplegia, palsies of the fifth through 11th cranial nerves requiring tracheotomy, and a left cerebellar syndrome. He also had a hypoplastic right vertebral artery terminating in the right PICA. Test occlusion of the vertebrobasilar junction from the left vertebral artery was well tolerated for 30 minutes, with adequate collateral supply through the posterior communicating artery. When traction was applied for balloon detachment, the balloon migrated proximally and during deflation of the balloon for repositioning, the patient became comatose, decerebrate, and died 3 weeks later.

The third patient died following microcoil occlusion of the left MCA for treatment of an aneurysm recurrence. He died from rupture of an untreated terminal carotid artery aneurysm while undergoing heparin therapy for a delayed transient neurological deficit secondary to MCA occlusion. This became evident on post-rupture arteriography, which revealed persistent MCA occlusion, but interval enlargement of the left carotid aneurysm.

Transient Neurological Deficits. Transient neurological deficits occurred in five of the 16 patients. Following MCA occlusion, one patient developed brachiofacial weakness contralateral to the vessel occlusion lasting several hours. During his first treatment in 1986, he was managed effectively with volume expansion and heparin, but with the same treatment in 1990, he developed an SAH from his untreated terminal carotid artery aneurysm and died (see above).

An accidental PICA occlusion (illustrative Case 1) resulted in a cerebellar syndrome lasting 12 hours with no radiological evidence of infarction. Additionally, a PICA occlusion for the two aneurysms on the same
artery resulted in a mild transient cerebellar syndrome (illustrative Case 2). One patient developed a transient decreased level of consciousness following coil embolization of the left P1 segment which resolved over 12 hours, and a mild cerebellar syndrome (thought to be secondary to a vertebral artery dissection) which resolved over 2 months. One of the patients treated by vertebral artery occlusion distal to the PICA developed a right PICA infarction the day after balloon occlusion. His symptoms had completely resolved 3 months after treatment but he had CT evidence of infarction at his 6-year follow-up examination.

Angiographic Complication. Angiography revealed migration into the aneurysm lumen of a P1-occluding detached balloon, which continued to swirl with the turbulent aneurysmal blood flow. This necessitated immediate P1 occlusion with a platinum minicoil. Following these emergency maneuvers, a vertebral artery dissection occurred with CT evidence of an ipsilateral right cerebellar infarction in the PICA territory. At the 12-month follow-up examination, the patient had resumed a normal life, with an unchanged visual field deficit.

Discussion

Endovascular treatment of vascular disease is conceptually very elegant. This form of treatment is less invasive than surgery, avoiding all cerebral manipulation. When uncomplicated, patients tolerate the treatment well, as it is carried out under neuroleptic analgesia, and the necessary duration of hospitalization may be very short. These procedures, performed as they are in the awake patient, allow constant clinical observation and arterial occlusion can be quickly reversed if the patient develops a deficit.

Surgical Complications

The surgical morbidity and mortality rates for direct treatment of large and giant aneurysms in difficult anatomical locations range from 16% to 38%.4,9,28,29 Surgical occlusion of intracranial parent vessels has been practiced for many years with varying results.6,14,17,20,27,31 The most reliable method of predicting the outcome of this intervention is surgical exploration with direct placement of a tourniquet proximal to the aneurysm, which is subsequently closed under radiological control in the angiography suite. There are several reported complications of tourniquet placement, including inadvertent vessel occlusion or dissection.4,30 Our reported results of endovascular parent vessel occlusion compare favorably with the above-cited surgical treatment results, and avoid both general anesthesia and operative intervention.

Case Selection and Outcome

The choice of patients for balloon occlusion of the intradural parent vessel is dependent on two criteria. If the aneurysm is clearly seen angiographically to be fusiform, it is treated primarily by balloon occlusion of the parent vessel. If the aneurysm is saccular, selective obliteration either by surgical or endovascular means is attempted prior to parent vessel occlusion.

Our criteria for successful test occlusion permitting definitive vessel occlusion include a normal baseline CBF study prior to treatment; good clinical tolerance of occlusion as proved at the bedside in the angiography suite during test occlusion, and arteriographic demonstration of complete collateral blood supply. Three possible outcomes of parent vessel occlusion are recognized: 1) good clinical and angiographic tolerance; 2) intolerance of vessel occlusion with apparent neurological deficit within a few minutes, necessitating immediate balloon deflation; and 3) delayed neurological deficit becoming apparent some time (hours to days) after occlusion. The first and second type of outcome are most frequently observed in the anterior circulation. Prediction of the third requires more sophisticated monitoring, particularly in the posterior circulation (discussed below).

Nondetachable Balloons

For permanent occlusion, the balloon was subsequently detached or implanted together with the small catheter. The balloon was not detached when the patient complained of pain with traction on the catheter for balloon detachment. In eight patients the No. 1 French microcatheter was left in place attached to the inflated balloon. This subgroup of patients have had from 1 to 8 years of angiographic follow-up monitoring demonstrating that the artery containing the catheter has undergone no visible pathological changes secondary to the implanted catheter. The patients treated in this way had no difference in clinical or radiographic evolution compared with the other patients in the series and showed no evidence of thromboembolism. Animals treated with this nondetachable balloon system have exhibited no thrombotic, thromboembolic, or inflammatory complications; however, myointimal hypertrophy of unknown significance was found in small to medium-sized arteries carrying the implanted catheter.19

CBF Studies

Pretreatment CBF studies were very useful, as illustrated by our one immediate failure of MCA test occlusion (Fig. 3). The patient was treated 3 days post-SAH and his baseline CBF was asymmetrical. Test occlusion for 1 minute resulted in hemiplegia which reversed with balloon deflation. This result is contrasted to that in another case in which the baseline CBF study was symmetrical and the collateral blood supply rich; this patient tolerated the test occlusion well (Fig. 4). In another case, asymmetrical CBF which did not increase with acetazolamide (Diamox) in a patient with small posterior communicating arteries probably con-
Deficit. Rhagia, phalic occlusion suffered and occlusion that, patient monitoring for treatment of the aneurysm. There is no evidence of leptomeningeal collateral blood supply to the distal MCA branches, and the patient developed hemiplegia at 1 minute. The balloon was deflated and removed, with complete recovery.

The basilar artery occlusion test poses different problems. The existence of multiple perforating arteries along the length of the basilar artery makes placement of even the smallest balloon dangerous, as the balloon may mechanically occlude the perforators. This is in contrast to surgical placement of a basilar artery tourniquet that can be interposed between the microscopically visualized perforating vessels. While this eventuality should be detected during balloon test occlusion, we believe that this may explain a poor result. In cases of small posterior communicating arteries, occlusion of the basilar artery’s origin may result in borderline blood flow not causing an immediately apparent clinical deficit. Marginal blood flow in these perforators may lead to their occlusion. While small perforator occlusion in the MCA or anterior cerebral artery distribution may occur without significant permanent clinical manifestation, such ischemia may cause severe problems in the posterior circulation. Somatosensory and brain-stem evoked responses may be useful in detecting subclinical changes predictive of grave delayed clinical deterioration.

The only patient who was treated successfully with basilar artery occlusion was young, with excellent collateral circulation through the posterior communicating arteries. Since MCA occlusions were also well tolerated in young patients, age may play a significant role in the good outcome of parent artery occlusions in these vessel distributions.

In cases of test occlusion intolerance, extra- to intracranial arterial bypass should be considered prior to endovascular occlusion of the parent vessel. We cannot tell from our series whether parent vessel occlusion performed shortly after the occurrence of an acute SAH is safe. The only patient treated in the acute phase of
of SAH had asymmetrical CBF and did not tolerate test occlusion (Fig. 3).

Recanalization

In four patients the previously occluded parent vessel became recanalized. One of these patients, the only patient in our series in whom the MCA-occluding balloon was placed partially in the aneurysm, developed an aneurysm proximal to the occluded segment apparent 4 years after treatment. One patient had recanalization of the coil-occluded A1 segment but no evidence of recanalization of the coil-filled aneurysm at 3 months postocclusion.

Follow-up angiography at 3 and 6 months and then on a yearly basis in patients having undergone this treatment is performed for evaluation of long-term results. The incidence of vessel recanalization also raises the question of the need for permanent inflation of the occluding balloon with polymerizing agents which might avoid this development.8

Conclusions

Endovascular occlusion of the parent vessel in treatment of intracranial aneurysms is a useful technique for some otherwise untreatable lesions. Baseline CBF measurements with clinical and angiographic criteria appear to be adequate predictors of occlusion tolerance in supratentorial aneurysms. At this time these criteria seem to be insufficient to predict the outcome of basilar artery occlusion.

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