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Giant Intracranial Aneurysms: Evolution of Management in a Contemporary Surgical Series

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Abstract

BACKGROUND—Many significant microsurgical series of patients with giant aneurysms predate changes in practice during the endovascular era.

OBJECTIVE—A contemporary surgical experience is presented to examine changes in management relative to earlier reports, to establish the role of open microsurgery in the management strategy, and to quantify results for comparison with evolving endovascular therapies.

METHODS—During a 13-year period, 140 patients with 141 giant aneurysms were treated surgically. 100 aneurysms (71%) were located in the anterior circulation, and 41 aneurysms were located in the posterior circulation.

RESULTS—108 aneurysms (77%) were completely occluded, 14 aneurysms (10%) had minimal residual aneurysm, and 16 aneurysms (11%) were incompletely occluded with reversed or diminished flow. 3 patients with calcified aneurysms were coiled after unsuccessful clipping attempts. 18 patients died in the perioperative period (surgical mortality, 13%). Bypass-related complications resulted from bypass occlusion (7 patients), aneurysm hemorrhage due to incomplete aneurysm occlusion (4 patients), or aneurysm thrombosis with perforator or branch artery occlusion (4 patients). 13 patients were worse at late follow-up (permanent neurological morbidity, 9%; mean length of follow-up, 23±1.9 months). Overall, good outcomes (GOS 5 or 4) were observed in 114 patients (81%) and 109 patients (78%) were improved or unchanged after therapy.

CONCLUSION—A heavy reliance on bypass techniques plus indirect giant aneurysm occlusion distinguishes this contemporary surgical experience from earlier ones, and obviates the need for hypothermic circulatory arrest. Experienced neurosurgeons can achieve excellent results with surgery as the “first-line” management approach and endovascular techniques as adjuncts to surgery.

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Keywords

bypass; direct clipping; giant aneurysm; indirect aneurysm occlusion; microsurgery

INTRODUCTION

Giant intracranial aneurysms have always been, and remain, among the most difficult cerebrovascular lesions to treat. Surgical therapy has evolved with refinement of microsurgical technique, improvements in instrumentation, application of skull base surgical techniques, and application of anesthetic techniques like hypothermic circulatory arrest and cerebroprotection. Despite these advances, combined surgical morbidity and mortality have remained in the 20% - 30% range for many years, partly due to inherent treatment risks and partly due to merciless pathological anatomy, like wide aneurysm necks, complex arterial branches, intraluminal thrombus, atherosclerotic degeneration of arterial tissues, and adherent perforating arteries. Persistent morbidity with surgical therapy and steady advances in endovascular therapy have encouraged attempts at coiling of giant aneurysms [1], with or without adjunctive techniques like stents or balloon assistance. In addition to coiling techniques, flow diversion and endoluminal reconstruction with devices like Pipeline [2] have been utilized with promising early results, particularly with giant aneurysms located along petrocavernous and paraclinoid segments of the internal carotid artery (ICA), and along the basilar trunk.

Rational decision-making with giant aneurysms requires comparison of safety and efficacy of newer endovascular therapies with published surgical experiences. Many of the most significant series of surgically treated giant aneurysms are decades old and predate changes in surgical practices since the introduction of Guglielmi detachable coils in 1990. Endovascular alternatives have changed indications for open microsurgery and curtailed the use of extreme techniques like hypothermic circulatory arrest [3]. Fewer giant aneurysms come to surgical management due to improved radiological imaging and earlier diagnosis of large aneurysms, before they reach giant sizes. Consequently, surgical results from older publications do not reflect the current practice environment. In this report, we reviewed a contemporary surgical experience with giant aneurysms with the following objectives: to examine specific changes in surgical management relative to early reports; to better establish the role of open microsurgery in the management strategy; and to quantify surgical results for comparison with evolving endovascular therapies.

METHODS

Patients

This study was approved by the University of California, San Francisco Committee on Human Research and conducted in compliance with Health Insurance Portability and Accountability Act (HIPAA) regulations. Giant aneurysms were defined as having a diameter ≥ 25 mm. With thrombotic aneurysms whose intraluminal diameter on angiography was less than overall aneurysm diameter, axial computed tomography and/or magnetic resonance imaging were used to measure aneurysm size. Patients with giant intracranial aneurysms who were treated microsurgically were identified from the prospectively maintained Vascular Neurosurgery database. Operative reports, inpatient charts, angiographic studies, magnetic resonance imaging, computed tomographic imaging, and outpatient clinic data were analyzed retrospectively.

During a 13-year period from September 1997 to March 2010, 2455 aneurysms were treated microsurgically in 1913 patients by the senior author (MTL). Of these patients, 140 patients

had 141 giant aneurysms (5.7% of all aneurysms). There were 90 women (64%) and 50 men (36%), with a mean age of 54 years (range, 1 – 85 years).

Twenty-three patients presented with subarachnoid hemorrhage (16%). Hunt-Hess grades were: grade I, 5 patients; grade II, 5 patients; grade III, 6 patients; and grade IV, 7 patients. All 19 patients with cavernous ICA aneurysms presented with cranial neuropathies in the form of cavernous sinus syndrome. Six patients presented with recurrent aneurysms after previous endovascular coiling, and one patient presented with recurrent aneurysm after previous microsurgical clipping.

Aneurysms

141 giant aneurysms were identified in 140 patients. 100 aneurysms (71%) were located in the anterior circulation, the most common sites being the middle cerebral artery (MCA, 23 aneurysms, 16.3%), cavernous ICA (19 aneurysms, 13.5%), and supraclinoid ICA (18 aneurysms, 12.8%) (Table 1). 41 aneurysms were located in the posterior circulation, the most common sites being the basilar bifurcation (14 aneurysms, 9.9%) and basilar trunk (9 aneurysms, 6.4%) (Table 1). Mean aneurysm diameter was 29 mm (range, 25 – 86 mm). 21 patients had additional aneurysms, 28 in total. One of these patients had two giant aneurysms.

Surgical Management

Decisions to treat microsurgically or endovascularly were made on an individual basis in conjunction with our multidisciplinary team, which included neurointerventional radiologists and neurovascular neurologists. Aneurysm location, morphology, presenting neurological condition, medical co-morbidities, predicted treatment risks, preferences of the treating team, and ultimately, preferences of the patient and family were considered in this decision. Exclusion criteria included Hunt-Hess grade V presentation, aneurysm calcification, location on the basilar trunk or vertebrobasilar junction, advanced age, significant anesthetic or surgical risks, and strong patient or family preferences against open surgery.

Giant aneurysms were exposed using standard site-appropriate surgical approaches, including 70 pterional craniotomies, 54 orbitozygomatic-pterional craniotomies, 13 far lateral-suboccipital craniotomies, 3 bifrontal craniotomies, and 1 torcular craniotomy. Direct aneurysm occlusion with conventional clipping of the neck was the primary treatment strategy, sometimes requiring temporary trapping, thrombectomy, and clip reconstruction. Indirect aneurysm occlusion was used as the alternative treatment strategy when direct neck clipping was not possible or considered too risky. Indirect aneurysm occlusion consisted of clipping the parent artery (proximal or distal occlusion), bypass with clipping the parent artery (proximal or distal occlusion, or trapping), or bypass with endovascular occlusion (proximal parent artery occlusion or aneurysm occlusion).

Balloon test occlusion (BTO) was used to select 26 patients for aneurysm management with a bypass, all of them with cavernous or supraclinoid ICA aneurysms. Ten patients failed the test with balloon inflation alone, and 16 patients failed with additional hypotensive challenge (lowering mean arterial pressure with nitroprusside drip by 20 mmHg, or 25% of mean arterial pressure, whichever was greater). Failed BTO was used as an indication for bypass. High-flow bypass was used in patients who failed BTO immediately, and low-flow bypass was used in patients who failed BTO after hypotensive challenge. The decision to perform a bypass with aneurysms in other locations was based on the aneurysm's unclippability and patients' angiographic anatomy (presence or absence of collateral circulation from the circle of Willis or leptomeningeal connections).

Adequacy of treatment and patency of parent vessels was analyzed intraoperatively using intraoperative angiography, indocyanine green (ICG) fluorescence videoangiography, and/or Doppler flow measurements.

Outcomes

Aneurysm occlusion was classified using postoperative angiography as: complete (no residual aneurysm), minimal residual aneurysm (small neck remnant or dog-ear), or incomplete (>5% of the original aneurysm lumen remaining). Bypass patency was also assessed angiographically. Aneurysm treatment failure was defined as post-treatment growth of residual aneurysm documented by angiography, or post-treatment aneurysm rupture.

Neurological outcomes were assessed using the Glasgow Outcome Score (GOS). A clinical nurse or clinician not directly involved in the care of these patients performed all outcome assessments preoperatively, early postoperatively (6 weeks), and at late follow-up. Preoperative neurologic condition was used as a reference point, and patient outcomes were expressed in terms of changes from this baseline (improved, unchanged, worse, or dead). Thus, asymptomatic patients who remain without neurologic deficits were classified as unchanged.

Statistical Analysis

Survival analyses for durability of surgical aneurysm repair were performed using the Kaplan-Meier method with group comparisons using the log-rank test. Binary variables were compared using Pearson's χ^2 test. Continuous variables were compared using an independent samples t-test. Continuous variables are presented as mean±standard error. Statistical tests were considered significant when $p<0.05$ after correcting for multiple comparisons using the Bonferroni method.

Given the potential confounding effect of fusiform morphology on the outcomes of posterior circulation aneurysms, we performed logistic regression to further assess for the impact of posterior circulation aneurysm location on the rate of ischemic injuries. Univariate analysis was used to identify covariates which might affect the rate of ischemic neurologic deficits in these patients. Binary and categorical variables were compared using Pearson's χ^2 test, or the χ^2 test for trend, respectively. Odds ratios on multivariate analysis reflect the risk of developing ischemic neurologic at the time of surgery. The goodness of fit of the regression model was confirmed by demonstrating a non-significant p-value on the Hosmer-Lemeshow test [4, 5].

RESULTS

Surgical Treatment

Overall, 141 giant aneurysms were treated in 140 patients (Table 2). Less than half (66 aneurysms, 46%) were treated directly with neck clipping and more than half (72 aneurysms, 51%) were treated indirectly with parent artery occlusion with or without bypass. Thrombectomy was performed with 34 aneurysms (24%) to facilitate direct clipping or to decompress brain or cranial nerves after direct clipping or trapping. Parent artery occlusion was performed surgically by clipping the proximal afferent artery (19 aneurysms), clipping the distal efferent artery or arteries (6 aneurysms), or complete trapping (24 aneurysms). Parent artery occlusion was performed endovascularly by coiling the proximal artery or aneurysm (23 aneurysms).

Bypass was part of the treatment with 54 aneurysms (38%). The external carotid artery-to-MCA bypass was the most common high-flow bypass (14 aneurysms, 26% of all bypasses),

and the superficial temporal artery-to-MCA bypass was the most common low-flow bypass (11 aneurysms, 20%). Intracranial-intracranial bypasses were used with 15 aneurysms (28% of all bypasses), and included in situ bypasses (2), reimplantation of efferent arteries (2), aneurysm excision with re-anastomosis of parent artery (3), and intracranial bypass grafts (8). Radial artery grafts were used in 10 patients and saphenous veins were used in 21 patients.

Three aneurysms were densely calcified at the base and could not be treated surgically. These patients were treated subsequently with coiling.

Aneurysm Outcomes

108 of 141 aneurysms (77%) were completely occluded with no residual aneurysm or neck remnant. 14 aneurysms (10%) had minimal residual aneurysm after clipping (small neck remnant or dog-ear) and further treatment was deemed unnecessary. 16 aneurysms (11%) were incompletely occluded after parent artery clipping with or without bypass (>5% of the original aneurysm lumen remaining). Postoperative angiography demonstrated reduced or reversed flow through the aneurysm, new intraluminal thrombosis, and decreased luminal size. The 3 calcified aneurysms were coiled incompletely or with minimal residual.

Complete aneurysm obliteration was achieved in 79% of cases (52/66) in which direct clipping was performed, and in 72% of cases (56/72) in which indirect aneurysm occlusion was performed. Bypass patency was confirmed in 47 of 54 bypasses (87%), with 7 bypasses occluding acutely (13%). Rates of aneurysm occlusion varied with location, with the lower rates with giant basilar trunk, posterior cerebral artery (PCA), basilar bifurcation, and MCA aneurysms. Kaplan-Meier analysis demonstrated 30 month aneurysm control rates of 96%, 93%, and 84% for completely occluded, minimally residual, and incompletely occluded giant aneurysms, respectively (log rank p=0.05) (Figure 1).

Five patients required additional treatment, 3 for recurrent aneurysms and 2 for incompletely occluded aneurysms (retreatment rate, 3.5%). One thrombotic P1-P2 junction aneurysm recurred 8 months after thrombectomy and clip reconstruction; the initially preserved posterior cerebral artery (PCA) required trapping with MCA-PCA bypass. A similar PCA aneurysm in another patient was clip occluded proximally, preserving PCoA to supply distal PCA flow; PCoA recanalized the aneurysm, which was treated with endovascular coiling. A dysplastic basilar bifurcation aneurysm was clipped directly with no residual aneurysm postoperatively; 2-year follow-up angiography demonstrated recurrent thrombotic aneurysm at the right P1 origin that was coiled. Large PCoA collaterals enabled one dolichoectatic basilar trunk aneurysm to be treated with proximal clip occlusion; although luminal filling was markedly reduced post-operatively, persistent aneurysm filling at 6-month follow-up required coiling. Another dolichoectatic vertebrobasilar aneurysm underwent VA clip occlusion and VA-SCA bypass with intra-aneurysmal thrombosis; she underwent contralateral endovascular VA occlusion when the aneurysm enlarged two years later.

Patient Outcomes

18 patients died in the perioperative period (surgical mortality, 13%) (Table 3). Eight patients died of ischemic complications, which included perforator infarctions associated with basilar artery aneurysms (6 patients), an intraoperative ICA tear that required occlusion (1 patient), and multiple embolic infarctions that were probably cardiogenic (1 patient). Six patients died of hemorrhagic complications, which included 3 postoperative hemorrhages, one hemorrhagic infarction resulting from bypass occlusion, one preoperative aneurysm re-rupture, and one intraoperative aneurysm rupture. Two patients died of medical complications, including one myocardial infarction with cardiac arrest and one coumadin-

related subdural hematoma. Two patients failed to improve despite uncomplicated treatment and support was withdrawn.

Complications related to bypass resulted from bypass occlusion (7 patients), aneurysm hemorrhage due to incomplete aneurysm occlusion (4 patients), or aneurysm thrombosis with perforator or branch artery occlusion (4 patients). Bypass occlusion resulted in MCA territory infarctions in 2 patients, focal ischemic deficits in 3 patients, and no sequela in 2 patients. Aneurysm re-hemorrhage after bypass occurred after proximal clipping in 1 patient, before endovascular occlusion in 1 patient, after endovascular occlusion from a previously unruptured aneurysm in 1 patient, and after endovascular occlusion while on heparin in 1 patient. Intraluminal aneurysm thrombosis occluded perforators on the basilar trunk in 3 patients with basilar trunk aneurysms and the anterior choroidal artery in 1 patient with an ICA bifurcation aneurysm.

Non-neurologic neurosurgical complications occurred in 7 patients and included five wound infections treated with surgical debridement and antibiotics, and two epidural hematomas treated with surgical evacuation. Medical complications occurred in 6 unruptured aneurysm cases and included two pulmonary emboli, one deep venous thrombosis, and three cases of symptomatic tachyarrythmia, none of which were fatal or caused long term morbidity.

15 patients (11%) were neurologically worse at early follow-up 6 weeks after surgery, but 13 patients (9%) were worse at late follow-up (permanent neurological morbidity, 9%). Overall, good outcomes (GOS 5 or 4) were observed in 114 patients (81%) and poor outcomes (GOS 3 or 2) were observed in 8 patients (5%) (Table 3). Relative to preoperative neurological baseline, 109 patients (78%) were improved or unchanged after therapy. The mean length of post-operative follow-up for all patients was 23±1.9 months.

Patients with giant posterior circulation aneurysms experienced higher rates of ischemic injury and neurologic complications than those with giant anterior circulation aneurysms. Posterior circulation location is an independent risk factor for ischemic injury, controlling for aneurysm morphology (OR 2.86, 95% CI 1.2-7.1, p=0.24). Patients with unruptured giant anterior circulation aneurysms were significantly more likely to have the same or improved neurological function at last follow-up than those treated for unruptured posterior circulation aneurysms (86% vs. 53%, χ^2 p=0.007). In general, SAH patients had worse outcomes: with ruptured giant aneurysms, 61% were improved or unchanged, 52% had good outcomes (GOS 5 – 4), and 39% died; with unruptured giant aneurysms, 82% were improved or unchanged, 84% had good outcomes, and 8% died.

DISCUSSION

Trends in Surgical Management

A heavy reliance on bypass techniques (54 bypasses, 38% of aneurysms) distinguishes this contemporary surgical experience with 141 giant aneurysms from earlier ones (Table 4). The increased use of bypasses reflects the impossibility of safely clipping many giant aneurysms. Direct clipping remains the preferred surgical technique for aneurysm occlusion, but it is prevented by dolichoectatic morphology, aberrant branch anatomy, atherosclerotic aneurysm neck, intraluminal thrombus, and in some cases, previous coil deployment. Our experience confirmed that less than half of giant aneurysms (66 aneurysms, 47%) were clippable.

The failure of conventional clipping requires the use of techniques to indirectly occlude the aneurysm or the use of adjuncts that facilitate direct clipping. Deep hypothermic circulatory arrest is one such adjunct that may transform an unclippable aneurysm into a clippable one by collapsing it, eliminating risk of intraoperative rupture, and permitting aggressive

manipulation, even entrance into the aneurysm to remove thrombus and create a supple neck. Giant aneurysms, and in particular those in the posterior circulation, are the most frequent indication for hypothermic circulatory arrest. In the Columbia University experience with 66 procedures in 66 patients, 57 were performed for giant aneurysms [3]. In the Barrow Neurological Institute experience between 1985 and 1994 with 62 procedures in 60 patients, 43 were performed for giant aneurysms [6]. This adjunct increases the rate of direct clipping, which was 62% in the BNI giant aneurysm experience, with hypothermic circulatory arrest applied in 23% of patients. Direct clipping rates are lower without hypothermic circulatory arrest: 39% in Drake's experience with posterior circulation giant aneurysms and 49% in Sundt's overall experience [7].

Deep hypothermic circulatory arrest incurs significant operative morbidity [3]. Cannulation of the femoral vessels can cause dissections, occlusions, and compromise of distal circulation [3, 8, 9]. Prolonged circulatory arrest is associated with cerebral ischemic injury and poor neurological outcomes [3]. Heparinization, slowing of the coagulation cascade by hypothermia, and trauma to red blood cells and platelets by the cardiopulmonary bypass pump contribute to postoperative bleeding complications [3]. In the early BNI experience, initial surgical mortality was 8.3% and permanent treatment-associated neurological morbidity was 13.3% [9]. As indications narrowed to the most complicated aneurysms, associated risks increased further. The most recent description of the BNI experience in 103 patients and 104 procedures reported 14 perioperative deaths (13.3%) and 9 later deaths (8.6%), for a cumulative mortality rate of 22%. The combined rate of permanent treatment-related morbidity and mortality was 32% [10].

These risks have resulted in declining use of hypothermic circulatory arrest. During a 15-year period at Columbia University, the number of procedures decreased from 34 during the first 5 years to 12 during the last 5 years. Although experiences at other institutions are unpublished, this trend appears widespread (see comments in [3]). Our giant aneurysm experience used hypothermic circulatory arrest in just 4 patients (3%).

Indirect aneurysm occlusion with or without a bypass has become a more acceptable alternative, with indirect occlusion consisting of proximal occlusion, distal occlusion, or trapping. This strategy has important advantages. The bypass can be performed with predictable ischemia times, cerebroprotection, and relatively low complication rates. The dangers of direct attack are avoided, including perforator dissection and preservation around clips. Clips are applied to afferent and efferent arteries, avoiding pathological tissues at the neck that can cause clip slippage, intraoperative rupture, and branch artery occlusions. Endovascular techniques can be utilized for indirect aneurysm occlusion, allowing the adequacy of a surgical bypass to be tested before permanent occlusion and also allowing for staged aneurysm occlusion with safe use of heparization or optimization of hemodynamics in the ICU.

Complications Associated with Bypass and Indirect Aneurysm Occlusion

Despite its advantages, indirect aneurysm occlusion introduces unique complications related to bypass graft occlusion and aneurysm thrombosis. Bypass and indirect aneurysm occlusion caused thrombotic occlusion of perforators or branch arteries in 4 patients (7%) with flawless bypasses (Figure 2). These surgical maneuvers are intended to eliminate pathological wall shear stress, high pressure variations, and turbulence that initiate aneurysm development and growth. Malignant hemodynamics are replaced with diminished or reversed intra-aneurysmal flows with more normal wall shear stress. These post-surgical hemodynamics reduce the risk of aneurysm rupture and initiate aneurysm thrombosis that is considered protective. However, unlike non-giant aneurysms that can be trapped safely, giant aneurysm occlusion is often deliberately incomplete because of the presence of

perforators or other branch arteries that would otherwise be trapped. Therefore, thrombosis initiated by bypass and aneurysm occlusion can potentially occlude these same arteries too. Giant aneurysm thrombosis is difficult to control temporally or spatially, and can transform a giant sac into a thrombotic and sometimes serpentine aneurysm that can obliterate perforators or small branch arteries at its base (Figure 3).

Our computational fluid dynamics (CFD) studies of giant aneurysm patients demonstrate that regions of slow, recirculating blood flow inside aneurysms are associated with abnormally low wall shear stress and aneurysm thrombosis [11]. Using high resolution gadolinium contrast-enhanced MR angiography and phase mapping velocimetry, regions predicted by CFD to have low velocities and low wall shear stresses correlated with regions of thrombus deposition in longitudinal studies of patients who initially had thrombus-free aneurysms and later developed intraluminal thrombus [12]. Using a “virtual ink” technique to inject aneurysms and follow its wash-out over several cardiac cycles of ink-free flow, estimates of flow residence times in giant aneurysms were elevated in regions where thrombus deposition was observed to occur in vivo [13]. Slow or stagnant flows appear to cause platelet aggregation, activation, and adhesion to aneurysm wall in these recirculation zones. Platelet activity induces thrombus formation, which also attaches to arterial walls, with potential peril to perforators after bypass.

Patency of branch arteries after bypass appears to be caliber-dependent. Thrombotic occlusions resulting in death or neurological deterioration after treatment involved basilar trunk perforators (3 patients) and anterior choroidal artery (1 patient). Flow reversal seems more thrombogenic than flow reduction, but direction of post-surgical blood flow did not correlate with outcomes. Thrombotic perforator occlusion occurred in 2 of 4 cases with preserved anterograde flow in the aneurysm and distal basilar artery occlusion. Thrombotic complications were not observed after bypass and occlusion of cavernous ICA aneurysms, an arterial segment that lacks branches supplying brain.

Postoperative anticoagulation might address this complication in some cases. Patients undergoing endovascular aneurysm occlusion can be anticoagulated 2-3 days after bypass surgery as part of a staged surgical-endovascular treatment. However, we did encounter heparin-induced hemorrhage after such a treatment (1 patient), and even a short delay between bypass and staged aneurysm occlusion by endovascular means can invite rehemorrhage (1 patient). Anticoagulation is problematic in patients immediately after surgical aneurysm occlusion, when most of our thrombotic complications occurred. Post-operative anti-platelet agents may be a remedy for thrombotic perforator occlusion.

CFD may be useful in guiding surgical strategy. Post-bypass hemodynamics can be simulated with both proximal and distal occlusion options and the regions of aneurysm thrombosis can be estimated with wall shear stress maps and virtual ink injections (Figure 4). The preferred treatment strategy will maintain robust flow with normal wall shear stresses in regions where branch arteries originate, while accepting stagnation in perforator-free zones like the dome and fundus. It is conceivable that stents, flow diverters, or other endovascular devices might direct blood flow to perforator zones and shape intra-luminal thrombosis. This technology is in its infancy, and currently the problem of thrombotic perforator occlusion remains unsolved.

Superiority of Surgical Management

This experience demonstrates that good results can be achieved with surgery in the majority of patients with giant aneurysms (GOS 5 or 4 in 81%, and improved or unchanged in 78% of patients). Treatment risks were significant, with a surgical mortality rate of 13% and treatment-associated neurological morbidity rate of 9%. 77% of aneurysms were occluded

completely, there were no late rehemorrhages, and the re-treatment rate was 3.5%. These results are comparable to other surgical series in the literature, where mortality rates range between 5% and 30% and good outcomes range between 60% and 90% [1, 6, 7, 14-37] (Table 6).

Giant aneurysms have not been favorable lesions for endovascular therapy [1, 8, 20, 38-46] (Table 6) because they frequently widen the neck, distort the anatomy of parent and branch arteries at the base, and induce intraluminal thrombosis. These anatomical factors make giant aneurysms dangerous to coil and difficult to obliterate completely, leading to recurrent aneurysm, multiple retreatments, occasional rehemorrhages, and neurological deterioration from progressive aneurysm enlargement. Giant aneurysms require techniques like stent- or balloon-assistance, which increase intraprocedural risks [41]. New technologies and techniques have advanced the efficacy and scope of endovascular therapy. In one of the largest endovascular experiences with 39 giant aneurysms, Jahromi et al. reported a complete occlusion rate of 36%, stent-assistance in 66%, and an average of 2 sessions to treat each aneurysm [40]. Cumulative treatment morbidity occurred in 12 patients (32%), and treatment mortality occurred in 6 patients (16%). Overall, 11 patients died at late follow-up (29%). Follow-up was short (mean duration, 25 months), which can result in underestimates of rates of recurrence, retreatment, and complications.

Endovascular therapy in its current state still transforms a significant number of giant aneurysms into a chronic disease requiring extensive surveillance, multiple retreatments, repeated risk exposure, and a relapsing clinical course. Flow diversion may yet offer some meaningful treatment options, particularly with basilar trunk and some basilar apex aneurysms where surgical morbidity rates are particularly high. Microsurgical aneurysm occlusion still appeals to many patients who prefer a single, definitive, and durable therapy. This contemporary experience with 141 giant aneurysms is a retrospective review and is limited by selection bias, lack of a control group, and short follow-up duration. Nonetheless, it demonstrates that experienced neurosurgeons can achieve excellent results with surgery as the “first-line” management approach, heavy reliance on bypass plus indirect aneurysm occlusion when conventional clipping fails, limited (if any) use of hypothermic circulatory arrest, and liberal use of endovascular techniques as adjuncts to surgery.

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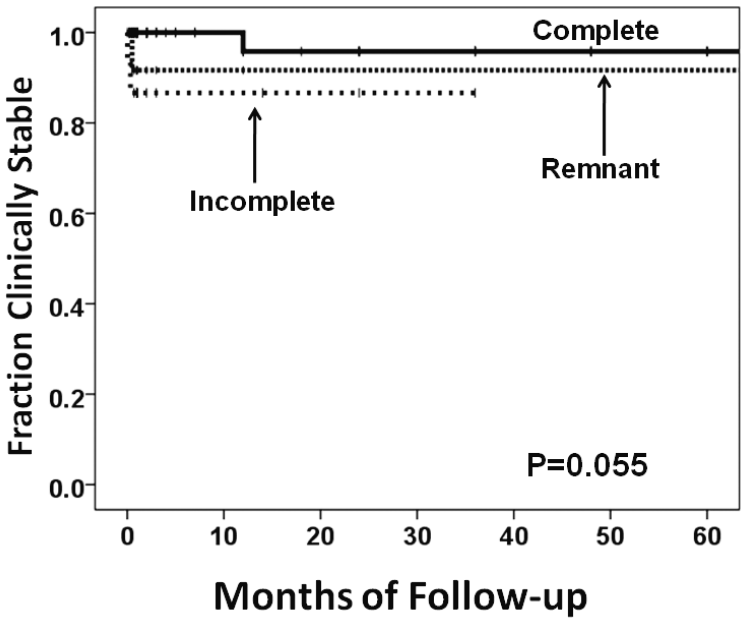
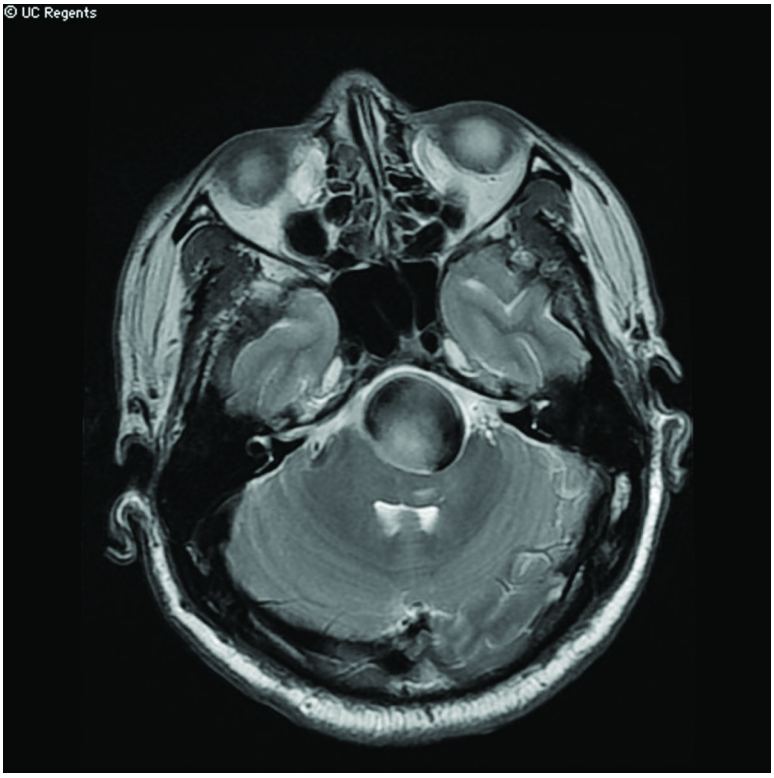
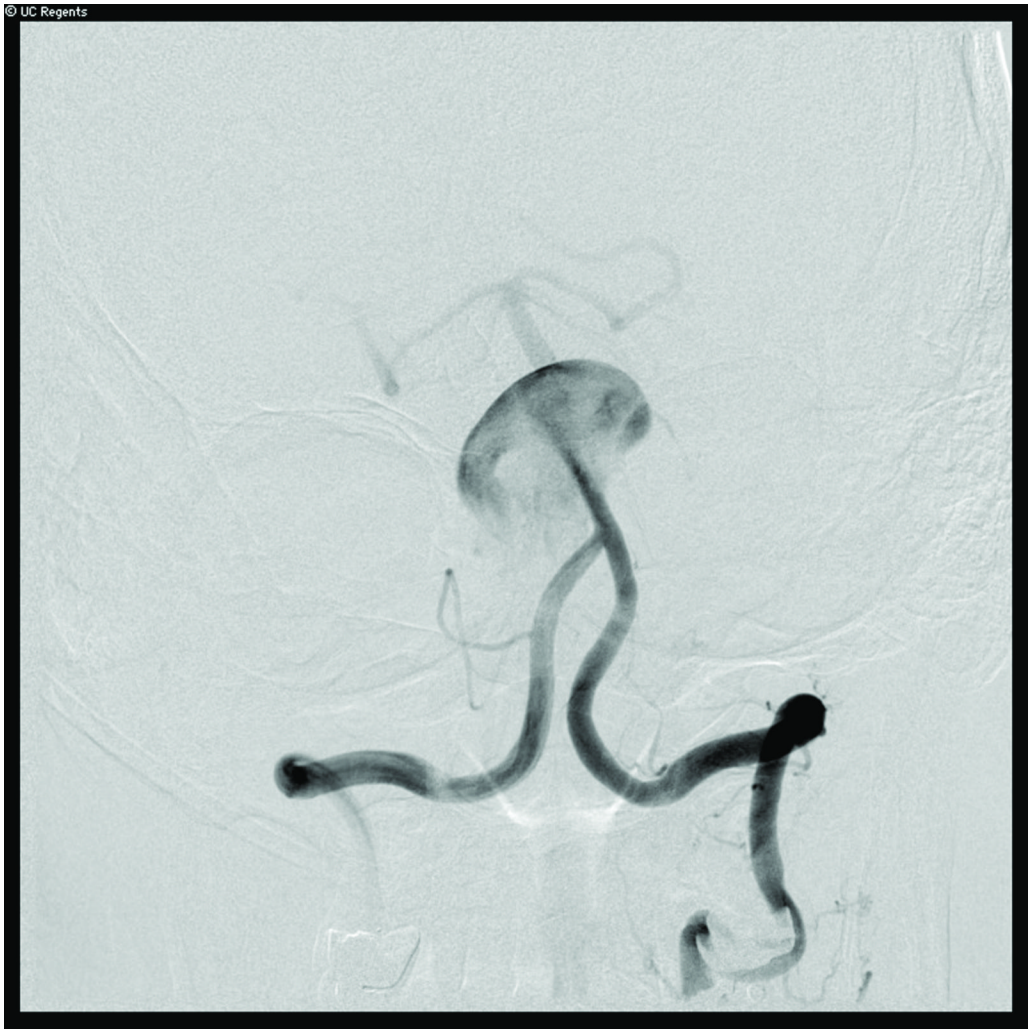
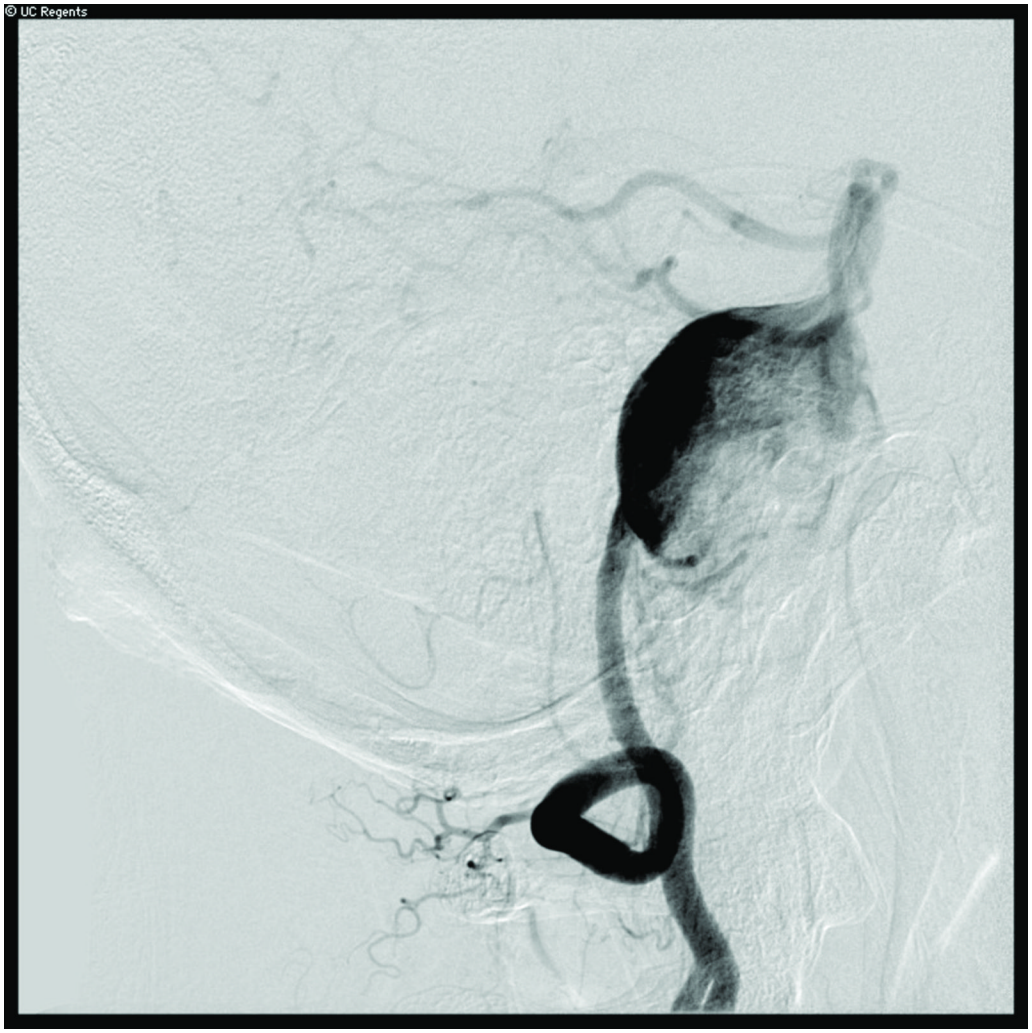
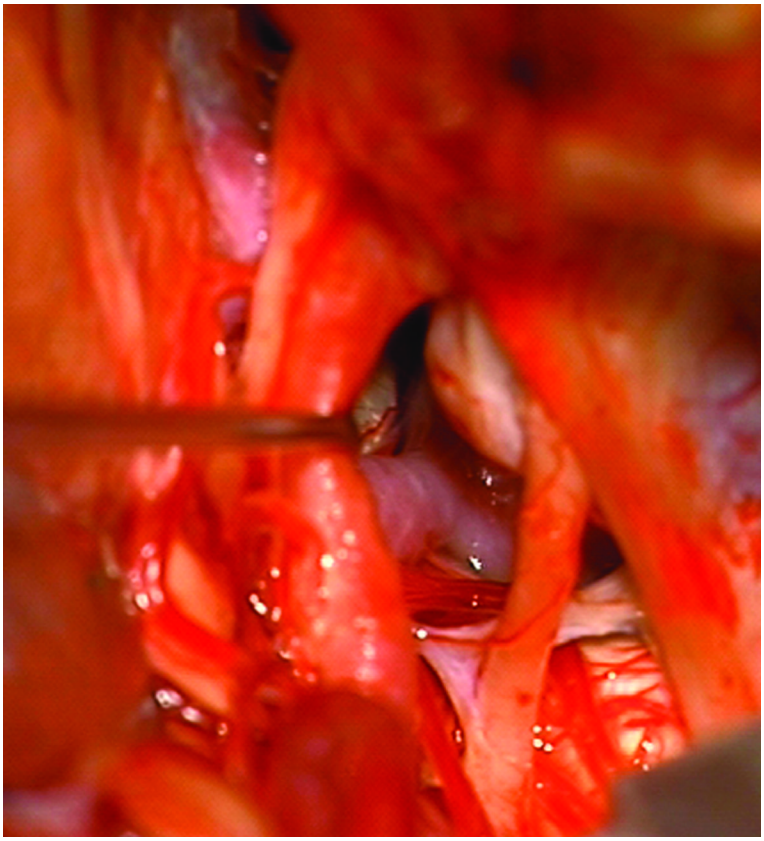


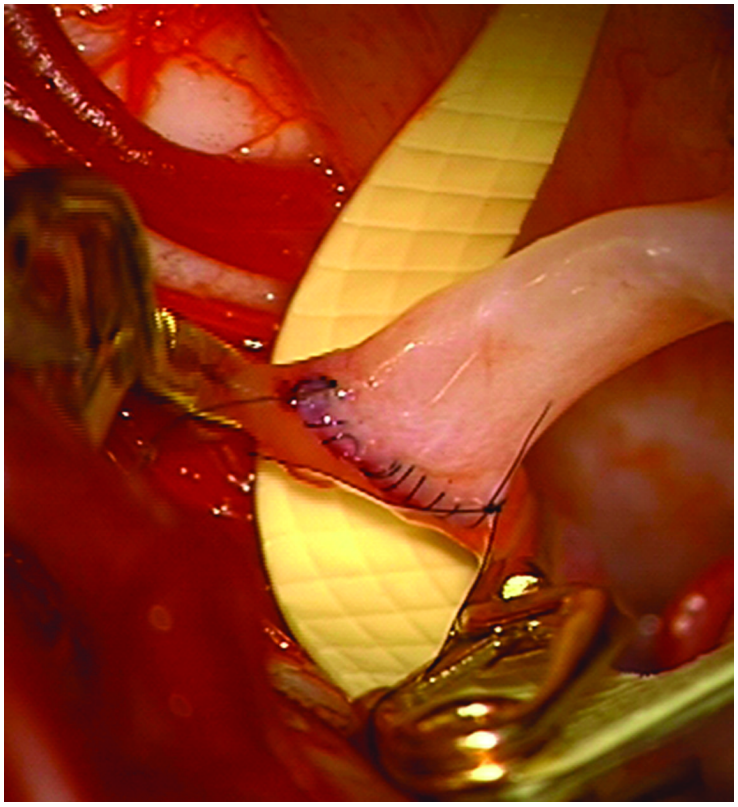
Figure 1.
Kaplan-Meier curves depicting the post-operative durability of aneurysm control of complete occluded, minimally residual, and incompletely occluded giant aneurysms.

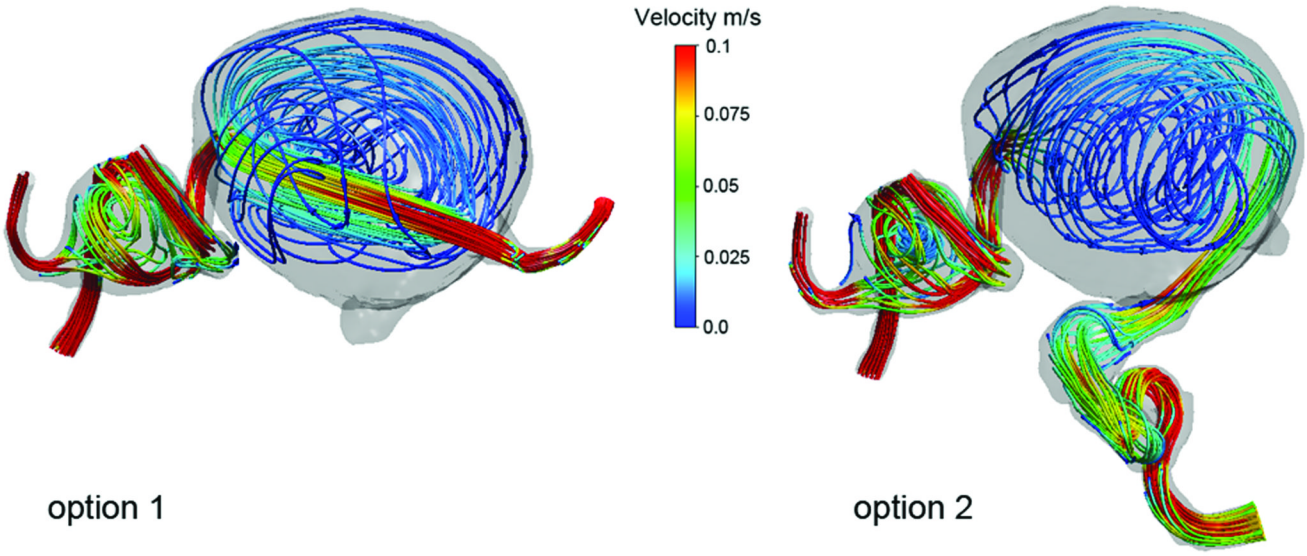


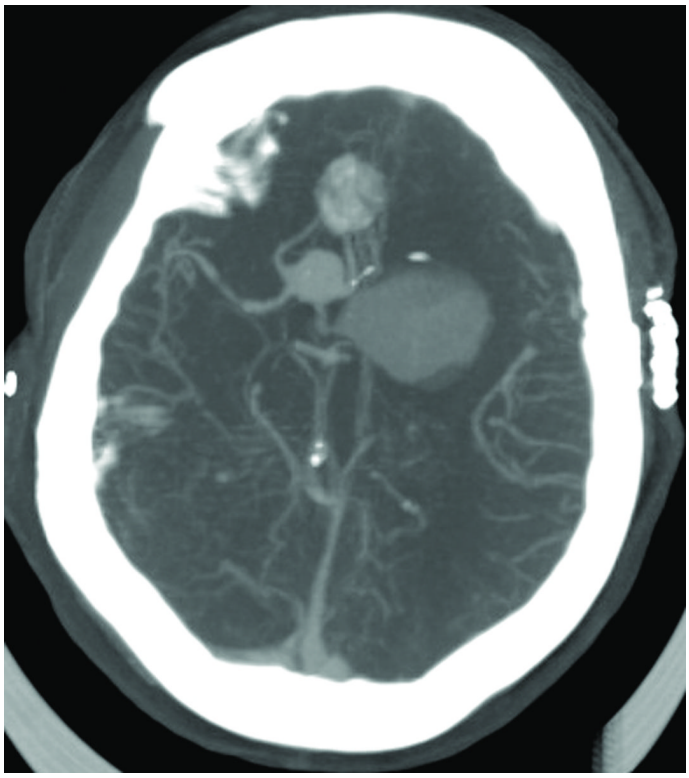












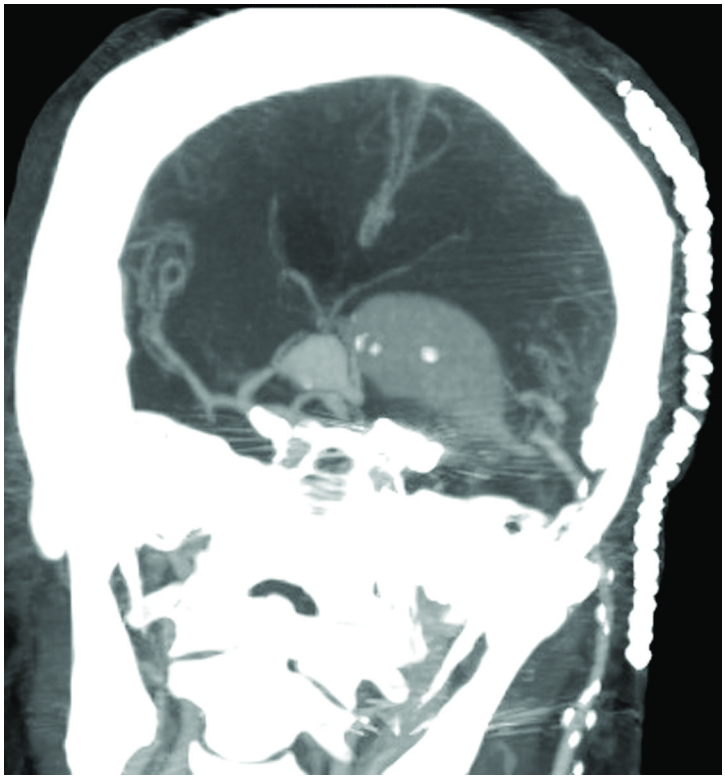
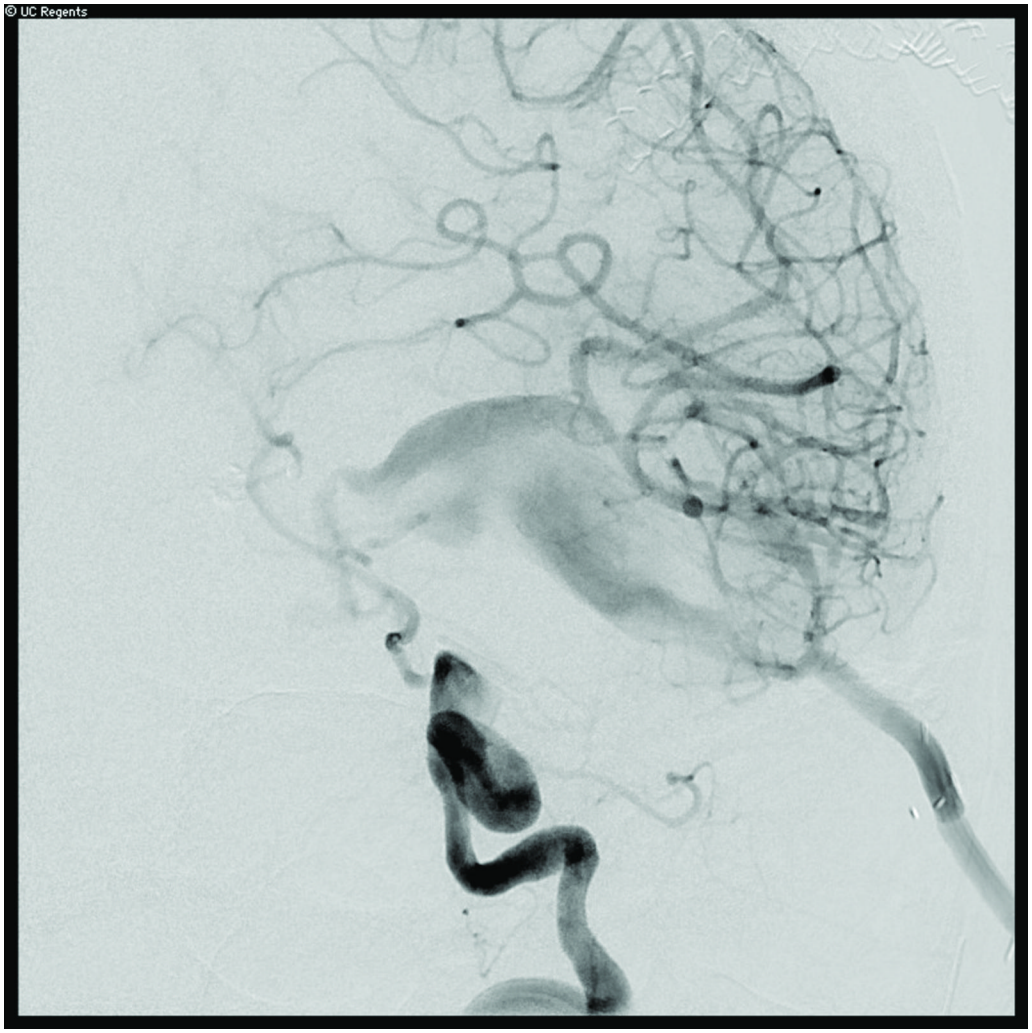
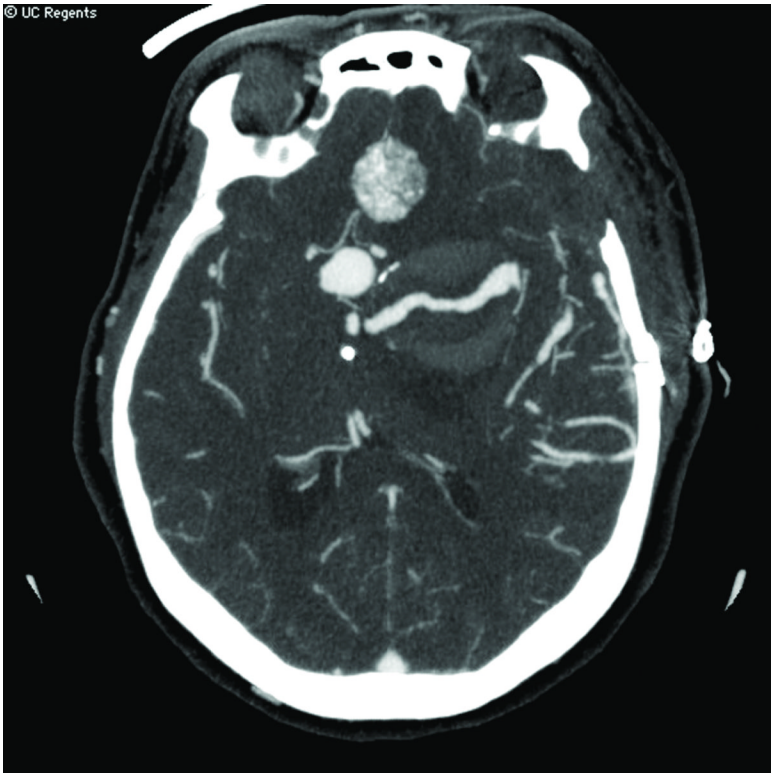
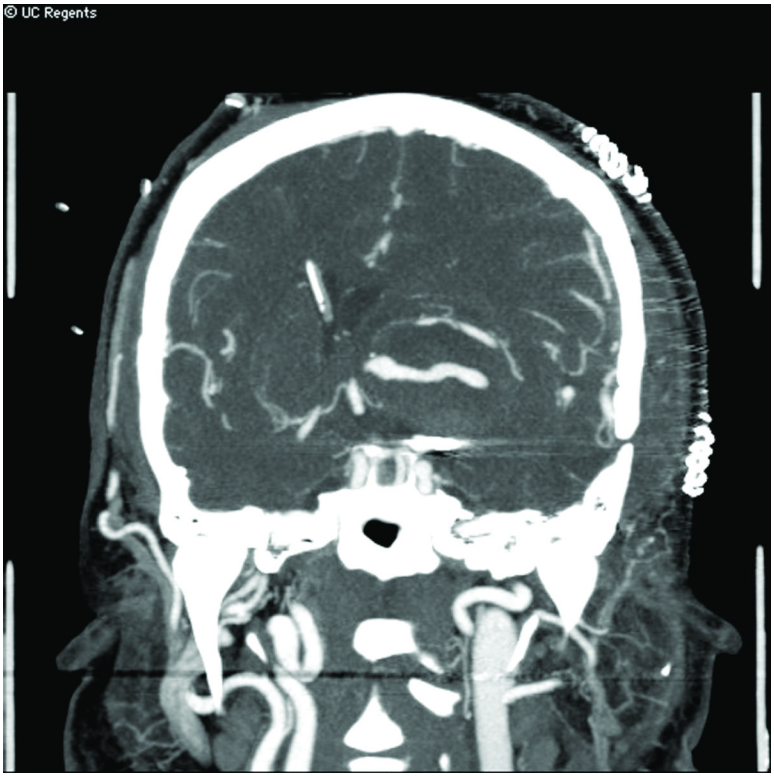
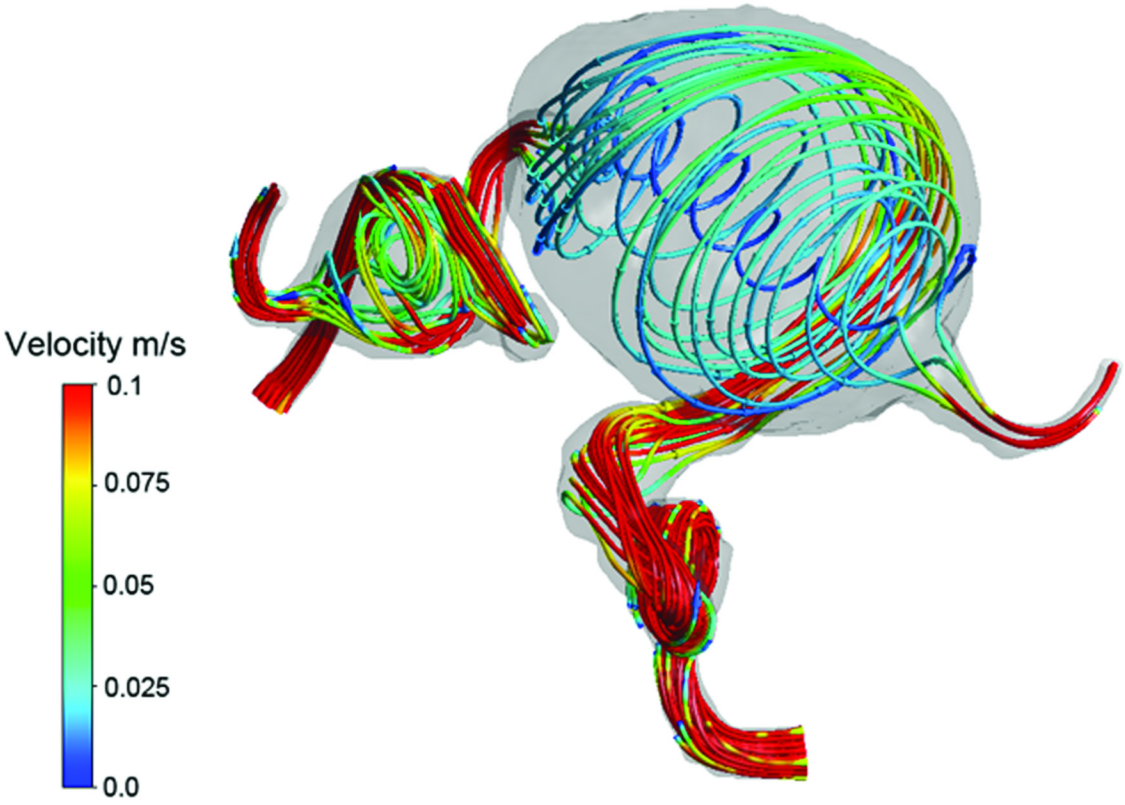


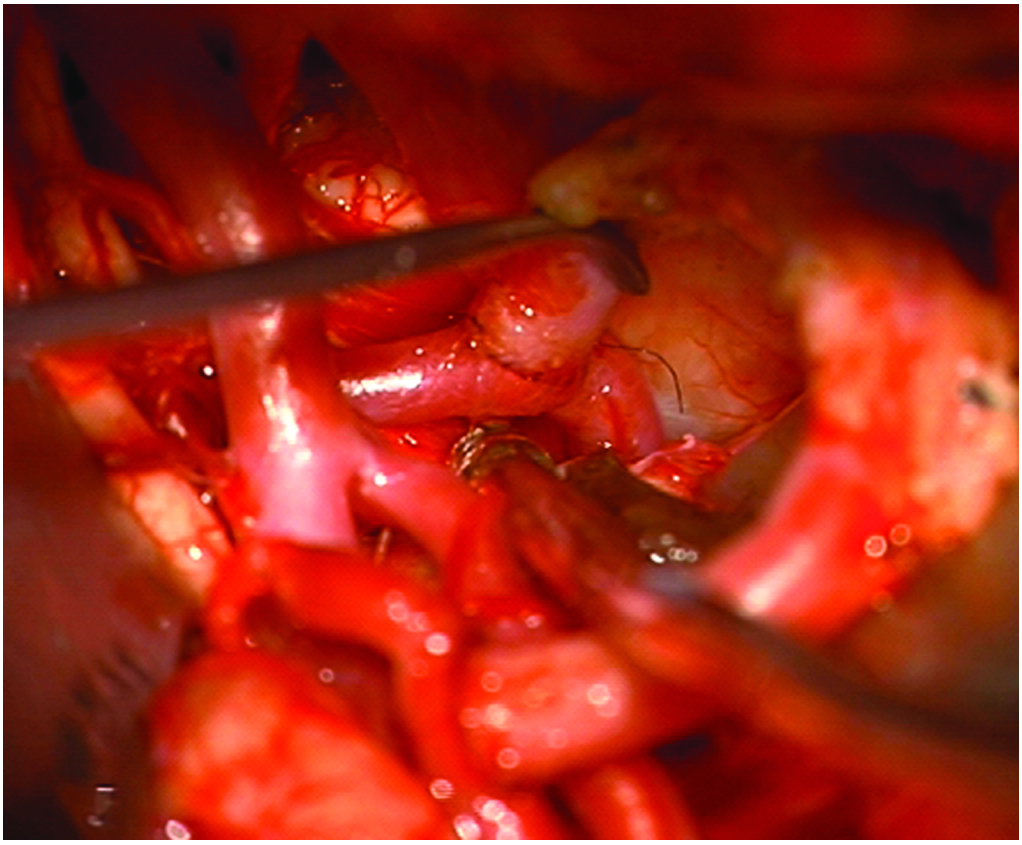
Figure 2.
Case 14. (A) Axial T2-weighted magnetic resonance images demonstrated a giant basilar trunk aneurysm with marked pontine compression in this 52 year-old man. The aneurysm had dolichoectatic morphology with separation of afferent and efferent basilar artery, as seen on digital subtraction angiography (left vertebral artery injection, (B) anteroposterior and (C) lateral views). (D) The distal aneurysm was exposed with an orbitozygomatic-pterional craniotomy and dissection through the carotid-oculomotor triangle. (E) A radial artery graft was anastomosed to the P2 segment, (F) as part of an MCA-PCA bypass. Note the proximal donor anastomosis in the Sylvian fissure. (G) The aneurysm was distal occluded with an aneurysm clip. (H) Postoperative angiography demonstrated slow antegrade filling of the aneurysm and brisk filling of the basilar apex through the bypass (right ICA injection, anteroposterior view). Gradual aneurysm thrombosis also occluded basilar perforators and the patient died. This case demonstrates that post-surgical aneurysm thrombosis can occlude perforators, even with distal aneurysm occlusion that preserves antegrade flow.

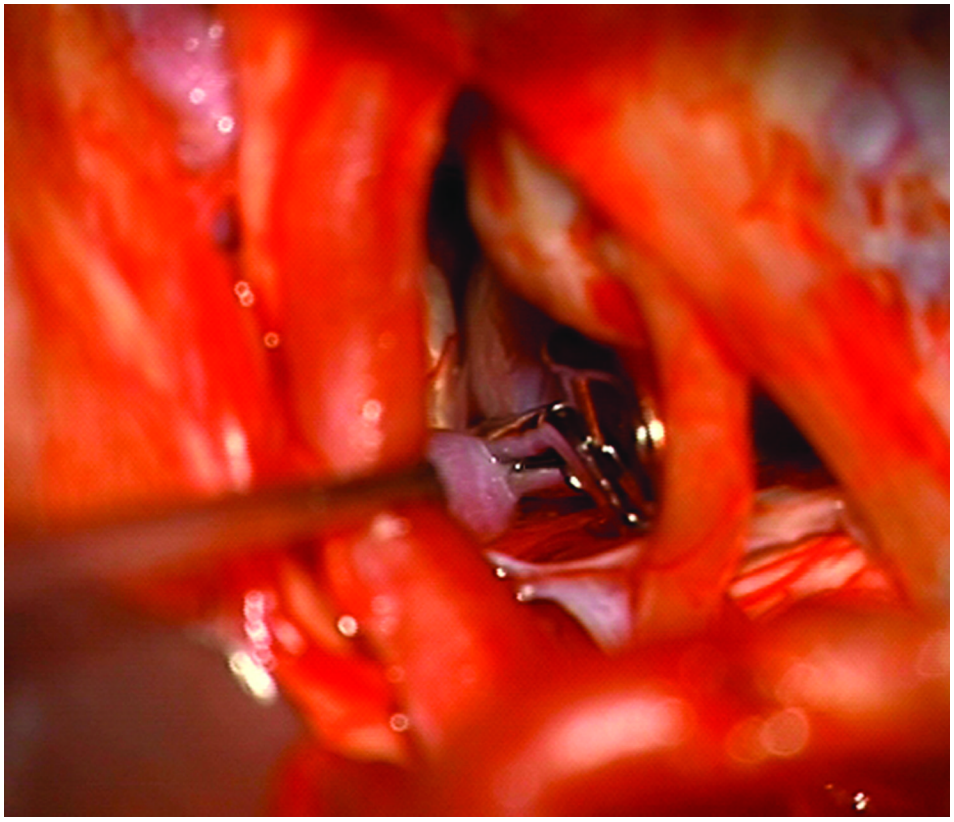


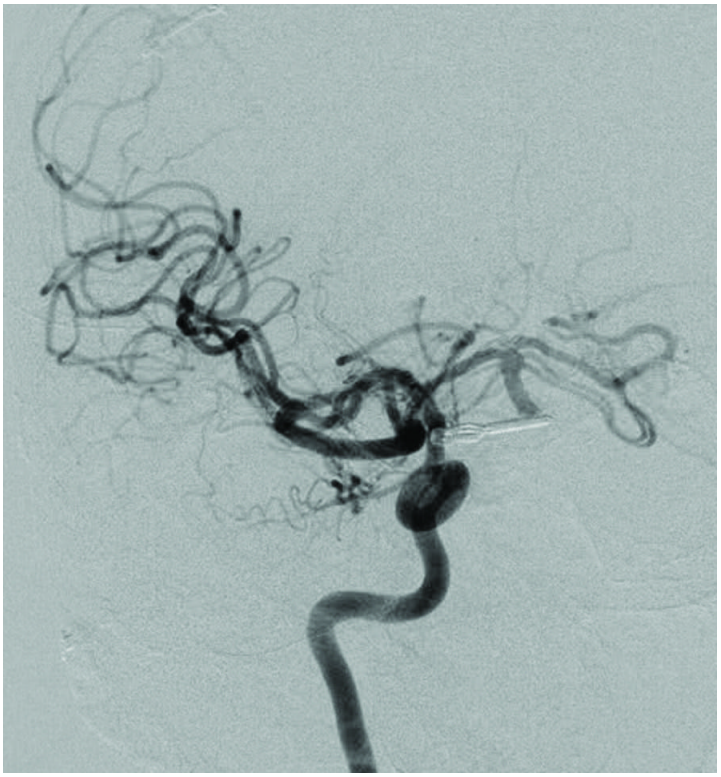












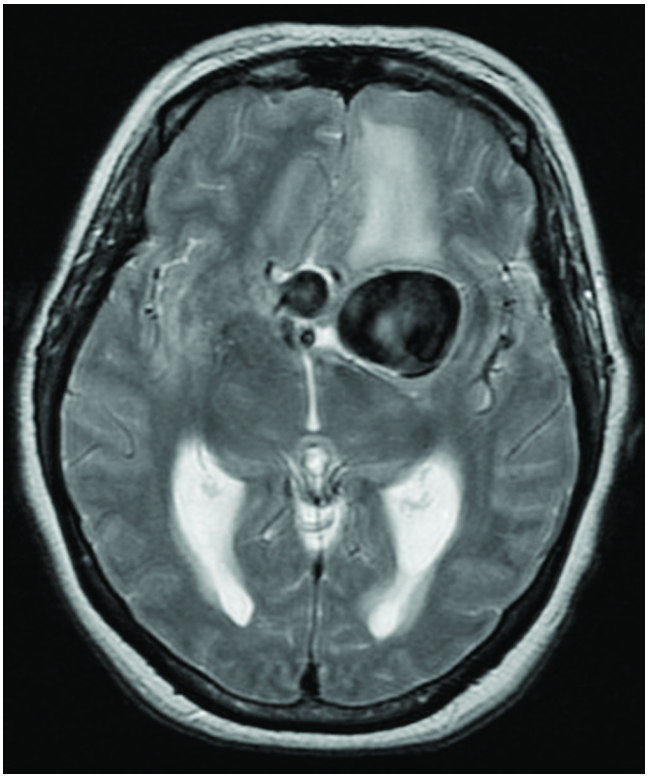
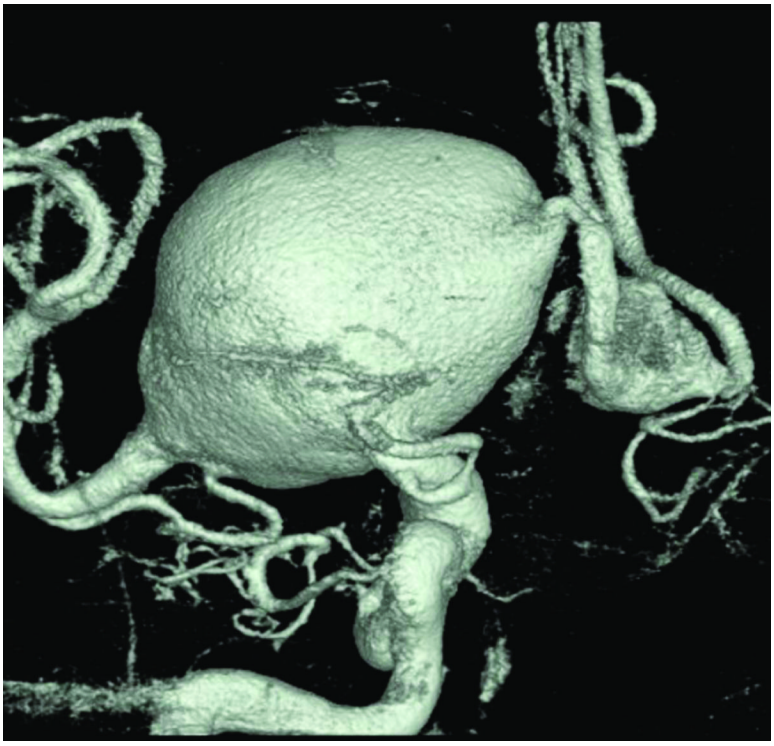


Figure 3.
Case 15. (A) Axial T2-weighted MR imaging revealed a giant left ICA bifurcation aneurysm and a large anterior communicating artery aneurysm in this 51 year-old woman. (B) 3D reconstructed angiogram (left ICA injection) demonstrated its dolichoectatic morphology. An end-to-side anastomosis between radial artery and the efferent MCA was part of an ECA-MCA bypass. (C) Supraclinoid ICA was occluded with a clip as it entered the aneurysm, distal to PCoA. Indocyanine green videoangiography demonstrated patency of the bypass graft, filling of distal MCA branches, filling of the supraclinoid ICA up to the clip, and faint flow of dye within the aneurysm. Postoperative CT angiography showed a thin layer of new intra-aneurysmal thrombus anteriorly, posteriorly, and inferiorly on (D) axial and (E) coronal views. (F) Subsequent digital subtraction angiography demonstrated bypass patency and progressive intraluminal thrombosis (left ICA injection, anteroposterior view). CTA on postoperative day 5 revealed further intraluminal thrombosis with two serpentine channels connecting the bypass with the A1 segment on the opposite side of the aneurysm, as seen on (G) axial and (H) coronal views. Post-surgical thrombosis occluded the anterior choroidal artery, and she suffered a capsular infarct. This case demonstrates that post-surgical aneurysm thrombosis after proximal clip occlusion can occlude small branch arteries.



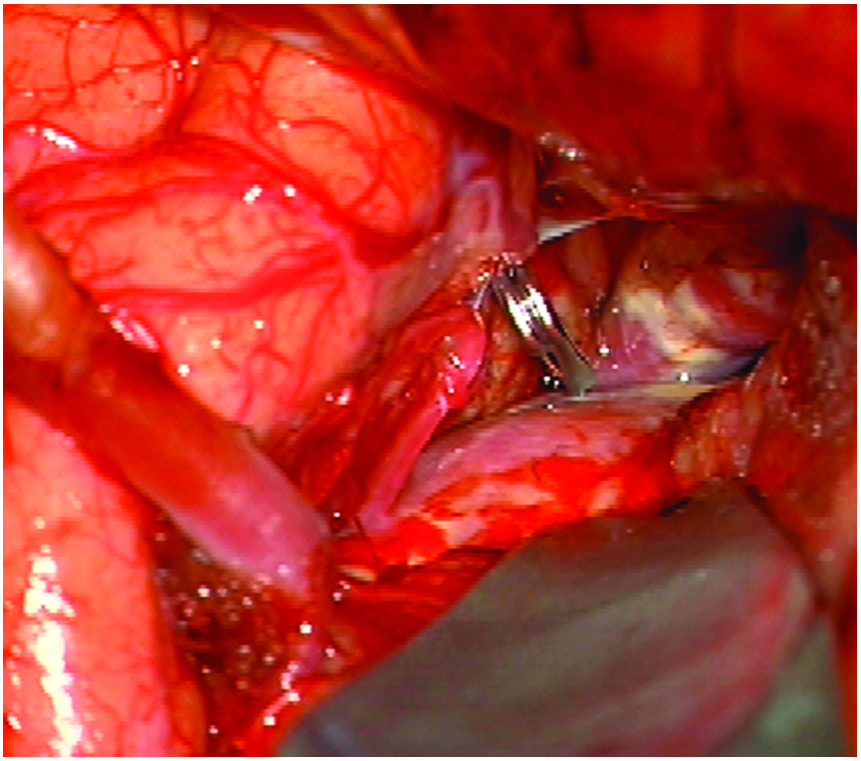


Figure 4.

Computational fluid dynamics explains the post-surgical aneurysm thrombosis observed in Case 15. (A) Preoperative velocity streamlines demonstrate the hemodynamics of this giant MCA aneurysm,. Following ECA-MCA bypass with radial artery graft, two surgical options

were considered: proximal aneurysm occlusion with the clip on the ICA distal to PCoA (option 1); or distal aneurysm occlusion with the clip on the MCA as it exits the aneurysm (option 2). (B) Proximal aneurysm occlusion results in flow through the center of the aneurysm and stagnation near the walls. Distal aneurysm occlusion results in flow along the walls of the aneurysm and stagnation in the center. Proximal aneurysm occlusion (option 1) was selected because it more dramatically altered intra-aneurysmal flow, and CFD predicted the pattern of peripheral aneurysm thrombosis based on stagnant flow and low shear stress near the walls. These simulations demonstrate that CFD can predict postoperative thrombotic complications and might be useful in planning surgical strategy.

Table 1

Location giant aneurysms. Abbreviations: ICA = internal carotid artery; PCoA = posterior communicating artery; MCA = middle cerebral artery; ACoA = anterior communicating artery; PCA = posterior cerebral artery; SCA = superior cerebellar artery; PICA = posterior inferior cerebellar artery.

Anterior Circulation	**N**	**%**
Cavernous ICA	19	13.5%
Supraclinoid ICA	18	12.8%
Ophthalmic	15	10.6%
Superior Hypophyseal	5	3.5%
PCoA	5	3.5%
MCA	23	16.3%
ACoA	13	9.2%
Pericallosal	2	1.4%
Posterior Circulation	N	%
Basilar Bifurcation	14	9.9%
PCA	7	5.0%
SCA	2	1.4%
Basilar Trunk	9	6.4%
PICA	4	2.8%
Vertebral Artery	5	3.5%
Total	141	

Table 2

Surgical management of giant aneurysms

Surgical Treatment	N	%
Direct Aneurysm Occlusion		
Neck Clipping	64	45%
Neck Clipping + Bypass	2	1%
Indirect Aneurysm Occlusion		
Parent Artery Clipping	20	14%
Parent Artery Clipping + Bypass	29	21%
Endovascular Occlusion + Bypass	23	16%
Exploration (calcified) → Coiling	3	2%
Thrombectomy	34	24%
Parent Artery Clipping		
Proximal Occlusion	19	
Distal Occlusion	6	
Trapping	24	
Hypothermic Circulatory Arrest	4	3%

Table 3

Patient outcomes after surgical intervention. Abbreviations: GOS = Glasgow outcome scale; NA = not applicable.

	Preoperative		Early Postoperative		Late Follow-up	
	N	**%**	**N**	**%**	**N**	**%**
GOS 5	90	64%	83	59%	93	66%
GOS 4	28	20%	14	10%	21	15%
GOS 3	22	16%	22	16%	6	4%
GOS 2	0	0%	3	2%	2	1%
Dead	0	0%	18	13%	18	13%
Total	140		140		140	
Improved	NA	NA	6	4%	22	16%
Unchanged	NA	NA	101	72%	87	62%
Worse	NA	NA	15	11%	13	9%
Dead	NA	NA	18	13%	18	13%
Total	NA	NA	140		140	

Table 4

Published experiences with surgical and endovascular treatment of giant aneurysms.

	Author	Year	Patients	Complete Occlusion	Bypass	Excellent/Good	Fair/Poor	Mortality	Morbidity	F/U	Retreatment	Rehemorrhage
		N	N	%						(yrs)	%	%
Endovascular Coiling												
Higashida	1990	39	63%	63%	0/39	n/a	n/a	n/a	n/a	0.5	n/a	n/a
Gobin	1996	9	5/9	56%	0/9	100%	0%	0%	11%	0.5	33%	0%
Gruber	1999	28	17/28	61%	0/28	73%			18%	3.3	82%	4%
Tateshima	2000	10	0/10	0%	0/10	n/a	n/a	n/a	n/a	2.6	20%	20%
Hallacq	2002	5	1/5	20%	0/5	60%	20%	20%	20%	1.0	0%	0%
Sluzewski	2003	29	5/29	17%	0/29	75%	4%	21%	10%	3.1	55%	7%
Henkes	2004	47	10/47	21%	0/47	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Kolasa	2004	7	4/7	57%	0/7	85%	14%	0%	n/a	n/a	n/a	n/a
Murayama	2006	33	19/77	25%	0/33	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Jahromi	2008	39	14/39	36%	0/39	63%	8%	29%	20%	1.3	54%	5%
Shi	2009	9	9/9	100%	6/9	78%	11%	11%	11%	1.0	0%	0%
Lylyk	2009	8	4/6	67%	0/8	n/a	n/a	n/a	0%	0.5	0%	0%
Microsugical Clipping												
Peerless, Drake	1982	118	97/118	82%	0/118	58%	26%	14%	n/a	n/a	n/a	n/a
Kodama, Suzuki	1982	49	n/a	n/a	n/a	61%	16%	22%	n/a	n/a	n/a	n/a
Yasargil	1984	30	26/30	87%	6/30	67%	23%	10%	n/a	n/a	0%	3%
Hosobuchi	1985	82	80/82	98%	15/82	84%	9%	7%	38%	10.0	0%	0%
Heros	1986	25	25/25	100%	n/a	72%	12%	16%	n/a	n/a	n/a	n/a
Sundt	1990	315	310/315	98%	81/315	80%	6%	15%	n/a	n/a	n/a	n/a
Ausman	1990	62	62/62	100%	23/62	84%	11%	5%	n/a	n/a	n/a	n/a
Tamaki	1991	4	4/4	100%	0/4	100%	0%	0%	0%	n/a	n/a	n/a
Lawton, Spetzler	1995	136	132/136	97%	40/136	85%	9%	6%	11%	2.2	1%	0%
Shibuya, Sugita	1996	29	n/a	n/a	n/a	84%	7%	8%	n/a	n/a	n/a	n/a
Kattner	1998	29	29/29	100%	1/29	87%	10%	3%	20%	7.0	0%	0%
Samson	1999	44	94%	94%	n/a	n/a	n/a	n/a	n/a	0.5	0%	0%
Osawa	2001	12	n/a	n/a	0/12	50%	16%	33%	n/a	n/a	0%	0%

	Author	Year	Patients	Complete Occlusion	Bypass	Excellent/Good	Fair/Poor	Mortality	Morbidity	F/U	Retreatment	Rehemorrhage
		N	N	%						(yrs)	%	%
Lawton	2002	28	28/28	100%	1/28	75%	11%	14%	5%	1.6	0%	0%
Jafar	2002	29	29/29	100%	29/29	93%	3%	3%	10%	5.0	0%	0%
Lozier	2004	19	6/16	38%	2/19	36%	47%	16%	89%	7.4	0%	0%
Gonzales	2004	8	8/8	100%	1/8	63%	38%	0%	50%	1.0	0%	0%
Kolasa	2004	13	13/13	100%	0/13	76%	23%	0%	n/a	n/a	n/a	n/a
Krisht	2007	11	11/11	100%	0/11	88%	12%	2%	27%	0.5	0%	0%
Hauck	2008	62	56/62	90%	9/62	68%	32%	15%	42%	1.0	0%	3%
Sharma	2008	181	106/118	90%	11/181	86%	5%	9%	12%	n/a	0%	0%
Cantore	2008	99	99/99	100%	41/99	89%	3%	8%	3%	8.5	1%	1%
Xu	2009	51	51/51	100%	0/51	84%	14%	2%	n/a	0.5	n/a	n/a
Sano	2010	109	109/109	100%	0/109	63%	16%	22%	4%	n/a	0%	0%
Current study	2010	140	118/140	84%	52/140	80%	7%	13%	10%	1.9	1%	1%
Combined Endovascular-Surgical Treatment												
Hacein-Bey	1998	5	4/5	80%	1/5	80%	20%	0%	0%	1.9	0%	0%
Arnautovic	1998	8	8/8	100%	0/8	87%	13%	0%	13%	2.0	0%	0%
Ponce	2004	8	n/a	n/a	5/8	76%	0%	25%	38%	1.5	0%	0%