

# Coiling of Very Large or Giant Cerebral Aneurysms: Long-Term Clinical and Serial Angiographic Results

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**BACKGROUND AND PURPOSE:** Initial complete occlusion of very large or giant aneurysms often cannot be accomplished, and most will partially reopen over time. This study was performed to assess the clinical and angiographic outcome of patients with very large or giant cerebral aneurysms treated with detachable coils.

**METHODS:** During 6 years, 29 patients with 31 very large or giant (20–55-mm) cerebral aneurysms were initially treated with detachable coils. Nineteen patients presented with subarachnoid hemorrhage (SAH), and eight patients had symptoms of mass effect. One patient had an incidental aneurysm, and one patient had an additional aneurysm.

**RESULTS:** Twenty-three (79%) of 29 patients had a good clinical outcome at a median follow-up of 50 months. One of 19 patients presenting with SAH had repeat bleed (annual rebleeding rate, 1.45%). After initial coiling, seven of 31 aneurysms were incompletely occluded; this rate increased to 20 of 29 aneurysms at 6-month follow-up angiography. After 16 repeat coiling procedures in 13 aneurysms, 12 of 29 aneurysms in surviving patients were still incompletely occluded. After additional treatment other than coiling (parent-vessel occlusion and/or surgery) in eight aneurysms, three of 25 aneurysms in 24 surviving patients were incompletely occluded. Only 13 (42%) of 31 aneurysms had one coiling as a sole therapy.

**CONCLUSION:** Coiling of very large or giant aneurysms can be considered. Long-term clinical outcomes were good in 79% of patients. The stability of the coil mesh over time was poor, requiring repeat coiling, surgery, and/or parent-vessel balloon occlusion in 58% of the aneurysms primarily treated with coils.

Direct surgical clipping of very large or giant cerebral aneurysms poses substantial risks (1–7). On the other hand, if these are left untreated, the prognosis of patients with giant cerebral aneurysms is grim, with 2-year survival rates reported to be as low as 20% (6, 8–10). An effective and safe alternative to clipping is endovascular parent-vessel occlusion, (11–13), which can be preceded by bypass surgery if necessary.

Besides clipping and parent-vessel occlusion, selective endovascular treatment with detachable coils can be considered (14–17). However, initial complete occlusion of very large or giant aneurysms often cannot be accomplished. Moreover, in time, most of these aneurysms partially reopen as a result of compaction of the coils or migration of the coils into intraluminal

thrombus (15). Despite these limitations, in some patients, coiling may be the only therapeutic option with acceptable risks (17).

The purpose of the present study was to determine the clinical and angiographic outcome in patients with very large or giant cerebral aneurysms that are primarily treated with detachable coils, with an emphasis on the clinical outcome, the stability of aneurysm occlusion over time, and the need for additional treatment.

## Methods

### Patients

Between November 1994 and March 2000, 955 intracranial aneurysms were treated in our institution. The allocation to the method of treatment was made in a multidisciplinary working group consisting of three experienced vascular neurosurgeons, two interventional neuroradiologists, and two neurologists. Primary clipping was performed in 665 aneurysms (69.6%); primary coiling, in 252 (26.4%); parent-vessel balloon occlusion with or without previous bypass surgery, in 35 (3.7%); and primary bypass surgery, in three (0.3%). Of the 955 treated aneurysms, 69 (7.2%) were 20 mm or larger. Of these, 69 very large or giant aneurysms, 35 were treated with parent-vessel

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**Summary of Relevant Clinical and Angiographic Data and Outcomes in 29 Patients with 31 Very Large or Giant Aneurysms**

Patient No./ Sex/Age (y)	Clinical Presentation	Aneurysm Location and Size	Timing of Tx after SAH	Initial Occlusion	Complications of Initial Coiling	Occlusion at First Follow-Up
1/F/49	1) ME, ophthalmoplegia and 2) AA	1) Carotid cavernous sinus, 35 mm, and 2) carotid ophthalmic, 20 mm	NA	1) Incomplete and 2) near complete	None	Complete at 5 mo
2/M/46	AA, SAH	Basilar tip, 20 mm	NA	Complete	None	Incomplete at 10 mo
3/F/35	ME, decreased visual acuity	Carotid bifurcation, 40 mm	NA	Incomplete	None	Incomplete at 3 mo
4/F/65	SAH H&H II	Carotid hypophyseal, 35 mm	29 d	Complete	None	Incomplete at 11 mo
5/M/48	ME, mutism, hemiplegia	Carotid bifurcation, 55 mm	NA	Complete	None	Incomplete at 11 mo
6/M/52	SAH H&H II	Basilar tip, 28 mm	16 d	Incomplete	None	Incomplete at 2 mo
7/M/68	SAH H&H II	Basilar tip, 26 mm	15 d	Incomplete	None	Complete at 3 mo
8/F/56	ME, ataxia, facial palsy	PICA, 32 mm	NA	Near complete	None	Incomplete at 8 mo
9/F/51	ME, visual loss, ophthalmoplegia	Carotid ophthalmic, 35 mm	NA	Incomplete	None	Incomplete at 5 wk
10/F/63	SAH H&H II	Basilar tip, 21 mm	33 d	Complete	None	Complete at 10 mo
11/M/40	SAH H&H II	Basilar tip, 30 mm	9 d	Complete	None	Incomplete at 10 mo
12/F/41	SAH H&H II	Supraclinoid carotid, 26 mm	6 d	Near complete	None	Incomplete at 6 wk
13/M/57	SAH H&H II	AcomA, 20 mm	38 d	Incomplete	None	Incomplete at 7 mo
14/F/50	SAH H&H II	AcomA, 23 mm	60 d	Complete	None	Incomplete at 6 mo
15/M/67	ME, trigeminal neuralgia	Superior cerebellar, 27 mm	NA	Complete	None	Incomplete at 6 mo
16/M/41	SAH H&H II	AcomA, 20 mm	22 d	Complete	TE event in A2	Near-complete at 6 mo
17/M/61	SAH H&H II	AcomA, 22 mm	10 d	Near complete	None	Incomplete (at 10 months)
18/F/55	SAH H&H II	Basilar tip, 20 mm	7 d	Complete	None	Near complete at 8 mo
19/F/62	SAH H&H IV	PcomA, 20 mm	4 d	Complete	None	Incomplete at 6 mo
20/M/50	SAH H&H II	Carotid bifurcation, 20 mm	8 d	Complete	TE M1 occlusion, urokinase caused aneurysm rebleed	None
21/M/36	SAH H&H I	AcomA, 24 mm	21 d	Complete	None	Incomplete at 6 mo
22/M/61	ME, frontal syndrome, mutism	AcomA, 55 mm	NA	Complete	Transient paresis, R leg	None
23/M/52	ME, frontal syndrome	AcomA, 30 mm	NA	Incomplete	None	Incomplete at 6 mo
24/F/47	SAH H&H III	Basilar tip, 20 mm	2 d	Complete	None	Complete at 6 mo
25/F/43	Incidental	Basilar tip, 26 mm	NA	Near complete	None	Incomplete at 6 mo
26/F/51	1) SAH H&H I and 2) AA	1) L carotid bifurcation, 25 mm; 2) carotid bifurcation R, 25 mm	1) 18 d and 2) NA	Near complete	None	Incomplete at 6 mo
27/F/54	SAH H&H II	PcomA, 20 mm	7 d	Complete	None	Near complete at 9 mo
28/F/60	SAH H&H II	Basilar trunk, 30 mm	7 d	Near complete	None	Incomplete at 7 mo
29/M/32	SAH H&H I	Carotid bifurcation, 20 mm	7 d	Complete	Transient hemiparesis, aphasia	Complete at 6 mo

Note.—AA indicates additional aneurysm; A2, second segment of the anterior cerebral artery; AcomA, anterior communicating artery; ME, mass effect; H&H, Hunt and Hess clinical grading at the time of treatment; NA, not applicable; PICA, posterior inferior cerebellar artery; TE, thromboembolic; Tx, treatment.

\* After coiling or repeat coiling.

† After additional therapy other than coiling.

balloon occlusion (13, 18), and three were treated with primary bypass surgery. In 29 patients, 31 aneurysms (45%) were primarily treated by means of selective endosaccular packing with detachable coils (Table 1). None of the very large or giant aneurysms were primarily clipped. The present study group consisted of the 29 patients with 31 very large or giant aneurysms that were primarily treated with coils. Fourteen patients

were men and 15 were women, with a mean age of 51.3 years (range, 32–67 years). Eight patients with nine aneurysms presented with symptoms of mass effect, and 19 patients with 20 aneurysms presented with subarachnoid hemorrhage (SAH). One patient had an incidental aneurysm, and one patient had an additional aneurysm after SAH from another aneurysm that was clipped.

Table continued

Complications	Additional Tx after Coiling	Occlusion at Last Follow-Up*	Final Occlusion†	Clinical Result in Non-SAH Patients	GOS Score in SAH Patients
None	None	Complete at 15 mo	NA	Ophthalmoplegia improved at 64 mo	NA
None	Repeat coiling	Complete at 16 mo	NA	Asymptomatic at 55 mo	NA
Hemiparesis	Carotid balloon occlusion	Incomplete at 3 mo	Complete at 6 mo	Died of pneumonia after SAH from AA at 14 mo	NA
None	Repeat coiling	Near complete at 22 mo	NA	NA	5 at 52 mo
None	Repeat coiling, carotid balloon occlusion	Incomplete at 11 mo	Complete at 21 mo	Improved at 51 mo	NA
Brainstem compression and edema, fatal rebleed	Bypass surgery to P2, bilateral vertebral occlusion	Incomplete at 3 mo	Incomplete at 3 mo	NA	1 at 6 mo, fatal rebleed
Hydrocephalus, VP shunt	None	Complete at 3 mo	NA	NA	5 at 49 mo
None	Repeat coiling	Complete at 46 mo	NA	Asymptomatic at 48 mo	NA
None	Bypass surgery	Incomplete at 5 wk	Complete	Unchanged at 47 mo	NA
None	None	Complete at 10 mo	NA	NA	5 at 49 mo
None	Repeat coiling	Complete at 33 mo	NA	NA	5 at 45 mo
None	Carotid balloon occlusion	Incomplete at 6 wk	Complete at 7 mo	NA	5 at 45 mo
None	None	Incomplete at 20 mo	NA	NA	5 at 38 mo
Transient hemiparesis	Repeat coiling	Complete at 21 mo	NA	NA	5 at 33 mo
Aneurysm dissection, bypass surgery	Repeat coiling, bypass surgery	Incomplete at 6 mo	Complete at 10 mo	Improved at 36 mo	NA
None	None	Near complete at 17 mo	NA	NA	5 at 31 mo
None	Repeat coiling, clipping	Incomplete at 10 mo	Complete	NA	5 at 16 mo
None	None	Near complete at 22 mo	NA	NA	5 at 22 mo
None	Repeat coiling	Near complete at 12 mo	NA	NA	3 at 14 mo
Death due to TE event and rebleed due to urokinase	None	NA	NA	NA	1 at 1 d
None	None	Incomplete at 13 mo	NA	NA	5 at 22 mo
None	None	None	NA	Death due to pneumonia at 3 mo	NA
None	None	Incomplete at 13 mo	NA	Improved at 13 mo	NA
None	None	Complete at 12 mo	NA	NA	5 at 18 mo
None	Repeat coiling	Near complete at 15 mo	NA	Asymptomatic at 15 mo	NA
1) None and 2) massive infarction resulting in death after bypass surgery	1) recoiling and 2) clipping	1) Near complete at 6 mo and 2) incomplete at 6 mo	NA	1) None and 2) death due to surgical complication at 16 mo	1 at 16 mo
None	None	Near complete at 40 mo	NA	NA	5 at 40 mo
None	2× repeat coiling	Complete at 17 mo	NA	NA	5 at 17 mo
None	None	Complete at 6 mo	NA	NA	5 at 6 mo

#### Aneurysm Characteristics

Fourteen aneurysms were very large (20–24 mm), and 17 were giant (25–55 mm). All aneurysms had a wide neck. Twenty aneurysms (65%) were located in the anterior circulation, and 11 aneurysms (35%) in the posterior circulation. Seven aneurysms were partially thrombosed, as confirmed on CT or MR imaging.

#### Endovascular Treatment

All procedures were performed with the patient under general anesthesia and with systemic heparin administration, which was continued for 48 hours after the procedure and followed by low-

dose aspirin. Twenty-one aneurysms were treated with Guglielmi detachable coils (GDCs) (Target Therapeutics, Boston, MA), seven aneurysms were treated with mechanically detachable coils (Cook Inc, Copenhagen, Denmark), and three aneurysms were treated with both types of coils. Ten aneurysms were treated by using the remodeling technique with a temporary supportive occlusion balloon (19).

#### Evaluation of Treatment

Initial aneurysm occlusion was scored as complete (95–100%), near-complete (90–95%), or incomplete (<90%) (17). The same grading was used to evaluate all results at follow-up

angiography, which was generally scheduled at 6 and 18 months after treatment. In patients with incomplete initial aneurysm occlusion or reopening of the aneurysm over time, different intervals for angiographic follow-up were chosen because these patients additional treatment was considered in these patients.

The clinical outcome was assessed by using the Glasgow Outcome Scale (GOS) (20) at the last clinical follow-up visit, by reviewing the charts, and by conducting telephone interviews. For patients presenting with SAH, the annual rebleed rate was assessed (ratio of the number of rebleeding episodes and the number of patient follow-up years).

## Results

Patient and aneurysm characteristics, as well as clinical and angiographic outcomes, are summarized in the Table.

### *Clinical Outcome*

*Patients Presenting with SAH.*—At a median follow-up of 37 months (mean 43.5, range, 0–75 months), 15 of 19 patients presenting with SAH were neurologically intact (GOS score, 5). Thirteen of these 15 patients had lesions of Hunt and Hess grade I–II, and two had lesions of Hunt and Hess grade III at the time of treatment. Coiling occurred at a median of 10 days (mean, 16.8 days) after SAH. One patient (patient 19), whose lesion was initially Hunt and Hess grade IV, was dependent (GOS score, 3). Three patients had died. One of these deaths (patient 20) was caused by thromboembolic M1 occlusion during coiling of a 20-mm carotid bifurcation aneurysm. Superselective intraarterial urokinase administration was needed, and this caused fatal rebleeding during the procedure. One patient (patient 6) with a 28-mm basilar bifurcation aneurysm died due to recurrent SAH 6 months after incomplete coiling and bypass surgery to the P2 followed by bilateral vertebral occlusion (18). The third patient (patient 29) died after surgery for an additional giant aneurysm that was incompletely coiled.

*Patients Not Presenting with SAH.*—Of the 10 patients that did not present with SAH, eight presented with signs of mass effect (patients 1, 3, 5, 8, 9, 15, 22, and 23), one patient had an aneurysm that was found incidentally (patient 25), and one (patient 2) had an additional aneurysm after SAH from another aneurysm that was clipped. Patient 1 also had an additional very large aneurysm that was treated with coils, and one of the patients from the SAH group (patient 29) had an additional giant aneurysm that was treated with coils. Of the eight patients who presented with symptoms of mass effect, four (patients 1, 8, 22, and 23) underwent a coil or repeat coil procedure as a sole therapy. The clinical symptoms of mass effect in these four patients disappeared in one (patient 8), improved in two (patients 1 and 23), and remained unchanged in one (patient 22). The remaining four patients received additional therapy other than coiling or repeat coiling for their aneurysms. The primary coiling procedure alleviated the symptoms of mass effect in one (patient 5), and in three patients (patients 3, 9, and 15), the mass effect remained un-

changed. Overall, in four (50%) of eight patients, symptoms of mass effect improved after coiling.

At a median follow-up of 62 months (mean, 52.3 months; range, 3–96 months) eight of 10 patients were neurologically intact, and two had died. One patient (patient 22) died of pneumonia 6 weeks after complete coiling of a 55-mm, large, anterior communicating artery aneurysm. At the time of treatment, this patient was mutistic and had a hydrocephalus caused by the mass effect of this giant aneurysm. The second patient (patient 3) was initially treated for a giant carotid bifurcation aneurysm, but the signs of mass effect did not improve. After 3 months, subsequent carotid balloon occlusion caused a severe hemiplegia, and she became dependent. She died 14 months later due to complications of a SAH in an additional basilar bifurcation aneurysm. One patient (patient 29), who had two giant aneurysms, died of surgical complications of an additional aneurysm.

Overall outcomes at a median follow-up of 50 months (mean, 46.9 months), comprising 121.2 patient years, was death in 17.2% of patients, dependency in 3.4%, and good outcomes in 79.3%. The annual rebleed rate in patients presenting with SAH was 1.45% (one rebleed in 68.9 follow-up years).

### *Angiographic Outcome*

All surviving patients underwent at least one follow-up angiographic examination. The median time of angiographic follow-up was 18 months (mean, 24.6 months; range, 1–96 months). Seven (23%) of 31 aneurysms were incompletely occluded after initial coiling. At first follow-up angiography performed at a median of 6 months (range, 1–11 months), 20 (69%) of 29 aneurysms in 27 surviving patients were incompletely occluded. All six partially thrombosed aneurysms in six surviving patients showed reopening of the aneurysm lumen caused by either thrombus resolution or migration of the coil mesh into the thrombus. Reopening in the other aneurysms was due to coil compaction. Sixteen repeat coiling procedures were performed in 13 aneurysms, and 10 of these aneurysms were examined during at least one follow-up angiographic examination at 6 months. The final angiographic results after coiling and repeat coiling showed that 12 (41%) of 29 aneurysms were incompletely occluded. Additional therapy other than coiling was performed in eight aneurysms: Parent-vessel balloon occlusion was performed in two; parent-vessel balloon occlusion after repeat coiling, in one; and bypass surgery and parent-vessel balloon occlusion, in two; clipping, in one; clipping after repeat coiling, in one; and bypass surgery after repeat coiling, in one. After these additional treatments, three (12%) of 25 aneurysms in 24 surviving patients were still incompletely occluded (in patients 13, 21, and 23). These three patients had anterior communicating artery aneurysms that were occluded to 80% with coils. Because both A2 segments were incorporated into the base of these aneurysms, further treatment was considered impossible. In 13 (42%) of 31

aneurysms, one single coiling procedure was the sole therapy.

### Discussion

The prognosis of patients with a very large or giant cerebral aneurysm is considered grim because of hemorrhage, cerebral compression, or thromboembolism, with 2-year survival rates reported to be as low as 20% (6, 9, 10). Because of this poor natural history, treatment is generally considered. Three therapeutic options are available: endovascular parent-vessel occlusion (11–13), direct surgical clipping (5, 6, 21–23), and endosaccular packing with detachable coils (15–17). Endovascular parent-vessel occlusion, whether or not it is preceded by bypass surgery, has been proved to be an effective and safe treatment; however, it is not always possible (13).

Surgical clipping of giant aneurysms can be an adequate therapy. However, large and giant aneurysms are at a higher risk for surgical morbidity compared with small aneurysms, especially when they are located in the posterior circulation (24, 25).

Selective endosaccular packing of cerebral aneurysms with detachable coils was introduced in the early 1990s. Although the effectiveness and safety of coiling of small and medium-sized cerebral aneurysms is increasingly acknowledged (14), its role in the treatment of very large or giant aneurysms is questioned because of frequent instability of the coil mesh over time that results in reopening of the aneurysm lumen (15, 17).

We found that primary coiling of large and giant aneurysms is technically feasible with a low rate of procedural complications. A good clinical outcome was achieved in 79% of patients at a median follow-up of 50 months. The rebleeding rate in patients presenting with SAH was low, and symptoms of mass effect were alleviated in half of the patients. However, initial aneurysm occlusion and stability of the coil mesh over time was poor, and additional coiling was required in many patients. Of note, these repeat coiling procedures did not cause additional morbidity. Even after repeat coiling, 12 of 29 aneurysms were still incompletely occluded, and parent-vessel occlusion or clipping was performed in eight aneurysms. After these additional therapies, three aneurysms were incompletely occluded, but further therapy was considered impossible. The good clinical outcome, poor aneurysm occlusion, and need for additional therapy in many patients are in concordance with observations from other studies of coiling in these aneurysms (15, 17). However, we had rates of procedural complications that were substantially lower than those reported by Gruber et al (17).

Two main reasons may account for why very large or giant aneurysms reopen so frequently over time. First, compared with smaller aneurysms, these are more often partially thrombosed: Six of 31 aneurysms in our patients were partially thrombosed. Both thrombus resolution and coil migration into the thrombus mass is well known to occur frequently (15). The second reason

for reopening over time is the impossibility of obtaining dense packing of giant aneurysms with coils. For instance, if 400 cm of GDC-18 coils are placed in a 25-mm aneurysm, the packing percentage is less than 6% of the aneurysm lumen, while the radiographic result may appear satisfactory. Future developments in endovascular treatments, such as new coils, embolization liquids, and stents, may overcome some of these problems and improve the clinical and angiographic results in patients with very large or giant cerebral aneurysms.

Our study had a retrospective design with inherent shortcomings in its collected data. Moreover, the favorable clinical results in the patients presenting with SAH do not apply to patients with SAH in general because all but three of our 19 patients had lesions of good clinical grades and because the timing of treatment was relatively long. However, given the poor natural history of patients with very large or giant aneurysms, the results indicate that these patients did benefit from treatment with coils.

Direct comparison with surgical clipping of very large or giant aneurysms is not valid because of differences in patient selection, clinical grading, aneurysm characteristics, and outcome assessments. Studies comparing the different treatment options are not available. However, the poor anatomic results and the need for additional therapy has led many authors to conclude that coiling of very large or giant aneurysms is ineffective and should not be performed. Nonetheless, we believe that, in a clinical setting, a reasonable approach may be to balance the poor anatomic results and frequent need for additional therapy of coiling against the low rate of procedural complications, the good clinical outcome, and the low rebleeding rate in individual cases. Patients with very large or giant intracranial aneurysms will benefit from a multidisciplinary approach with a consideration of all possible treatment modalities, depending on many factors such as the patient's age and clinical condition, the characteristics of the aneurysm, and the local expertise.

### Conclusion

In the treatment of large and giant aneurysms, coiling may be considered. Close angiographic follow-up is mandatory to permit the timely detection of any indications for repeat coiling or other additional therapy.

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