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Received, November 30, 2007.
Accepted, June 4, 2008.

CLINICAL AND ANGIOGRAPHIC OUTCOME AFTER ENDOVASCULAR MANAGEMENT OF GIANT INTRACRANIAL ANEURYSMS

OBJECTIVE: Giant (≥25 mm) intracranial aneurysms (IA) have an extremely poor natural history and continue to confound modern techniques for management. Currently, there is a dearth of large series examining endovascular treatment of giant IAs only.

METHODS: We reviewed long-term clinical and radiological outcome from a series of 39 consecutive giant IAs treated with endovascular repair in 38 patients at 2 tertiary referral centers. Data were evaluated in 3 ways: on a per-treatment session basis for each aneurysm, at 30 days after each patient's final treatment, and at the last known follow-up examination.

RESULTS: Ten (26%) aneurysms were ruptured. At the last angiographic follow-up examination (21.5 ± 22.9 months), 95% or higher and 100% occlusion rates were documented in 64 and 36% of aneurysms, respectively, with parent vessel preservation maintained in 74%. Stents were required in 25 aneurysms. Twenty percent of treatment sessions resulted in permanent morbidity, and death within 30 days occurred after 8% of treatment sessions. On average, 1.9 ± 1.1 sessions were required to treat each aneurysm, with a resulting cumulative per-patient mortality of 16% and morbidity of 32%. At the last known clinical follow-up examination (mean, 24.8 ± 24.8 months), 24 (63%) patients had Glasgow Outcome Scale scores of 4 or 5 ("good" or "excellent"), 10 patients had worsened neurological function from baseline (26% morbidity), and 11 had died (29% mortality).

CONCLUSION: We present what is to our knowledge the largest series to date evaluating outcome after consecutive giant IAs treated with endovascular repair. Giant IAs carry a high risk for surgical or endovascular intervention. We hope critical and honest evaluation of treatment results will ensure continued improvement in patient care.

KEY WORDS: Endovascular treatment, Giant intracranial aneurysms, Outcomes

Neurosurgery 63:662–675, 2008 DOI: 10.1227/01.NEU.0000325497.79690.4C www.neurosurgery-online.com

Giant (≥25 mm) intracranial aneurysms have an extremely poor natural history (21, 32) and continue to confound current techniques for management by maintaining a high occurrence of periprocedural neurological injury and death (1, 3, 6–8, 10, 12, 16–19, 23–25, 32). Numerous open surgical series have demonstrated morbidity and mortality rates ranging from 26 to 35% and 15 to 21%, respectively (3, 11, 28). Improving technologies have encouraged endovascular surgeons to attempt treatment of these difficult lesions. Currently, there is a dearth of large series examining endovascular treatment of solely giant aneurysms, but the developing body of literature suggests limited efficacy of this approach as well, with mortality rates reaching 24 to 27% and additional morbidity rates on the order of 12 to 42% (true numbers are difficult to generate, as most studies also include "very large aneurysms" and/or are small selected series) (6–8, 16–18, 24, 25).

As endovascular techniques evolve to deal with problems of coil herniation, delayed coil impaction, and late rebleeding, a large study of giant aneurysms treated with endovascular

Abbreviations: GOS, Glasgow Outcome Scale; **ICA**, internal carotid artery; **SAH**, subarachnoid hemorrhage

repair is needed. To this end, we performed a multi-institutional retrospective review of consecutive giant aneurysms treated with endovascular repair.

PATIENTS AND METHODS

Thirty-eight consecutive patients with 39 giant aneurysms were treated by endovascular means between December 2001 and July 2007 at 2 tertiary referral centers (the University at Buffalo and the University of Pittsburgh). Clinical and radiographic outcome data were collected from prospectively gathered databases at each institution and supplemented by retrospective chart and angiogram review where necessary. The angiographic follow-up period was defined as the time period between the first treatment session and the last angiogram available for each patient. The clinical follow-up period was defined as the time period between the first treatment session and the last clinical follow-up examination results available. Institutional review board approval was obtained at each center for this study.

Morbidity and mortality were evaluated on a per-session basis as well as on a per-patient, cumulative basis. Sessional morbidity was defined as the proportion of sessions with any untoward event occurring within 30 days, divided into sessional events affecting patients in a transient or permanent manner. Cumulative morbidity was defined as the proportion of patients who experienced a procedure-related neurological worsening that did not resolve fully at any time point between the first treatment session and 30 days after the last treatment session. Sessional and cumulative mortality were defined in a similar fashion. Morbidity and mortality at the time of final follow-up examination denote worsening relative to initial presentation and all-cause death, respectively, assessed at the last clinical follow-up examination. Glasgow Outcome Scale (GOS) scores were determined at the last clinic visit or by telephone call. A score of 4 (moderate disability, independent for activities of daily life) was considered a good outcome, and a score of 5 (good recovery, resumption of normal activities despite minor neurological or psychological deficits) was considered an excellent outcome. All outcomes were assessed by means of an intent-to-treat format. Data are presented as mean ± standard deviation. Univariate statistics used Student's *t* test for continuous variables and Fisher's exact test for ratios.

RESULTS

Demographics

Data for 38 patients harboring 39 giant aneurysms were reviewed. The average age of these patients was 60.9 ± 11.7 years. Thirty (79%) patients were women; 10 (26%) patients presented with subarachnoid hemorrhage (SAH). Demographics, presentation, aneurysm size, location, and treatment are summarized in Tables 1 and 2. A total of 10 ruptured and 29 unruptured aneurysms were treated. There were no significant differences in age or sex between SAH and non-SAH groups (Table 3).

Angiographic Outcome

The last angiogram for the patients in our series was obtained at a mean of 21.5 ± 22.9 months (median, 14.8 months; range, 0–94 months). At the time of the last follow-up examination, parent vessel preservation was possible in 74% of aneurysms (9 of 10 ruptured and 20 of 29 unruptured aneurysms) (Table 1). Among the 10 cases in which the parent vessel patency was not preserved, 8 occlusions were planned after successful balloon test occlusion, and 2 were the result of unintentional adverse events. The first unintentional occlusion was discovered incidentally on follow-up angiography and was asymptomatic. The second resulted in several small cortical occipital lobe infarcts that were clinically undetectable against the background of a middle cerebral artery stroke.

Angiographic occlusion rates of 100% were obtained in 12 of 29 unruptured aneurysms and 2 of 10 ruptured aneurysms, for an overall rate of 36% (Table 1). Occlusion rates of 95% or greater (i.e., including 100% occluded aneurysms) were obtained in 19 of 29 unruptured aneurysms and 6 of 10 ruptured aneurysms, for an overall rate of 64%. The remaining aneurysms (14 of 39) were all less than 95% occluded. One of these was never treated because of difficult anatomy, despite multiple attempts at treatment.

Stents were required at some point during the management of 25 aneurysms. Of these, 6 were in ruptured aneurysms and 19 were in unruptured aneurysms. Of stents placed in ruptured aneurysms, only 4 were placed during the initial, post-SAH attempt.

Clinical Outcome

Thirty-nine aneurysms were treated over the course of 76 sessions, resulting in an average of 1.9 ± 1.1 treatment sessions per aneurysm (Table 3). Nine percent of treatment sessions resulted in transient morbidity (ruptured, 4%; unruptured, 12%) and 20% of treatment sessions resulted in permanent morbidity, all of which was neurological (ruptured, 8%; unruptured, 25%). Death within 30 days occurred after 8% of treatment sessions (ruptured, 8%; unruptured, 8%).

At 30 days after their last procedure, 12 of 38 patients had worsened neurological status (10 of 28 non-SAH patients and 2 of 10 SAH patients), whereas 6 patients had died (4 of 28 non-SAH patients and 2 of 10 SAH patients) (Table 2). At the last known clinical follow-up examination (mean, 24.8 ± 24.8 months), 24 (63%) patients had a GOS score of 4 or 5 ("good" or "excellent" outcome; 19 of 28 non-SAH patients and 5 of 10 SAH patients) (Table 2), 10 patients had worsened neurological function, and 11 had died (7 of 28 non-SAH patients and 4 of 10 SAH patients). Six deaths at the last known clinical follow-up were directly attributable to procedural events, resulting in an overall 16% cumulative procedure-related mortality rate among our 38 patients. Final follow-up GOS score was 3.4 ± 1.7 (Table 3). Patients with electively treated unruptured aneurysms had better eventual outcome (GOS score of 3.7 ± 1.7) than those presenting with SAH (GOS score of 2.6 ± 1.5), although this trend did not reach statistical significance ($P = 0.08$) (Table 3).

Illustrative Cases

Patient 16

A 57-year-old woman with a history of lupus erythematosus, rheumatoid arthritis, coronary artery disease, coronary stent placement, chronic obstructive pulmonary disease, and heavy nicotine abuse presented with a decreased level of consciousness, nausea, vomiting, and visual blurring worsening over 2 weeks. Imaging demonstrated acute obstructive hydrocephalus secondary to a giant, partially thrombosed posterior circulation

TABLE 1. Patient demographics and treatment[a]

Patient no.	Age (yr)/ sex	Aneurysm size	Aneurysm location	Presentation			BTO	Treatment session	Treatment summary	Parent vessel preserved	% occluded on last angiogram
				CN deficit	SAH	Other					
1	41/M	29 × 21	L ICA-c	VI	No	H/A	None	2	Stent/coil, coil	No	100
2	66/M	26 × 23	L MCA		No	Dizziness	None	1	Stent/coil	Yes	95
3	73/M	27 × 25	R ICA-c		No	H/A, retro-orbital pain	Pass	4	Coil, abort, stent, coil	Yes	90
4	55/F	30 × 16	L PICA		No	L visual field scintillations	None	2	Stent, stent	Yes	100
5	63/M	30 × 15	R ICA-c		No	TIA	Pass	2	BTO, prox. occl.	No	100
6	60/F	26 × 18	R ICA-p	II	No	H/A	Pass	1	Prox. occl.	No	100
7	69/F	27 × 18	R ICA-p	II	No	H/A	None	1	Balloon/coil	Yes	85
8	69/F	42 × 33	R ICA-c	III, VI	No		Pass	1	Prox. occl.	No	100
9	57/F	27 × 16 25 × 15	R ophth. L ophth.	II	No		Pass None	1 1	Prox. occl. Stent /coil	No Yes	100 95
10	82/F	25 × 21	R AICA	III, VI, VII	No	Brainstem compression	None	3	Stent/coil, stent/ coil, coil	Yes	>90
11	68/F	27 × 21	L ICA-p	II	No		Fail	3	Failed coil × 3	Yes	0
12	74/F	41 × 41	R ophth.	II	No	TIA	None	3	Coil, coil, stent/coil	Yes	>95
13	49/F	25 × 11	L MCA		No	L H/A, L facial pain	None	1	Stent/coil	Yes	95
14	43/F	26 × 16	R PCA		No	H/A, N/V, mass effect	None	2	Stent/coil, coil	No	90
15	46/M	31 × 15	R ICA		No	Severe headache	Pass	2	Coil, prox. occl.	No	100
16	57/F	32 × 18	R P1/P2		No	Drowsy, N/V, visual blurring	None	1	Stent/coil	Yes	100
17	71/F	26 × 26	BA tip		No		None	1	Stent/coil	Yes	50
18	58/F	25 × 15	R ICA-c		No	TIA, H/A	Pass	4	BTO, stent attempt, stent, coil	Yes	100
19	48/F	29 × 21	R ICA-p	II	No		Fail	2	BTO/coil, coil	Yes	70
20	51/F	33 × 23	BA		No	Thalamoperforator stroke	None	2	Coil, stent/coil	Yes	100
21	59/F	31 × 24	L ICA-c	V	No	H/A	Pass	1	Prox. occl.	No	100
22	65/F	36 × 35	L ICA-t		No	H/A	None	3	Stent, attempted stent, coil	Yes	0
23	71/F	25 × 24	L ICA-c	VI	No	Diplopia, pain	Pass	1	Prox. occl.	No	100
24	79/F	36 × 32	BA		No	Paresis, gait difficulty	None	1	Stent/coil	Yes	>95
25	70/F	27 × 20	L ophth.		No	H/A	None	1	Stent/coil	Yes	>95
26	56/F	35 × 35	R ophth.	II	No		None	2	Coil, stent/coil	Yes	>98
27	61/F	39 × 38	L MCA		No	Dysphasia	None	2	Coil, stent/coil	Yes	>90
28	74/F	26 × 24	BA	III	No	N/V, paresis	None	1	Stent/coil	Yes	70
29	48/F	27 × 26	R ICA-p		HH4		Pass	3	Coil, stent/coil, prox. occl.	No	100
30	74/F	26 × 24	L ophth.	III	HH1		None	3	Stent/coil, coil, coil	Yes	85
31	80/M	41 × 33	R MCA		HH2	Refused treatment before rupture	None	1	Coil	Yes	>95

TABLE 1. *Continued*

Patient no.	Age (yr)/ sex	Aneurysm size	Aneurysm location	Presentation			BTO	Treatment session	Treatment summary	Parent vessel preserved	% occluded on last angiogram
				CN deficit	SAH	Other					
32	55/M	35 × 27	L ICA-t		HH5		None	5	Stent/coil, stent/coil, coil, coil, stent/coil	Yes	95
33	55/F	29 × 24	R ICA-p		HH5		None	2	Coil, coil	Yes	>95
34	52/F	32 × 21	R ophth.		HH4		None	1	Stent/coil	Yes	0
35	44/M	25 × 18	L PCA		HH2		None	1	Coil	Yes	100
36	75/F	25 × 25	R ICA-p		HH3		None	1	Stent/coil	Yes	>95
37	57/F	25 × 23	L ICA-p		HH4		Fail	3	Coil, BTO/coil, coil	Yes	>90
38	39/F	27 × 20	BA		HH1		None	4	Coil, coil, attempted coil, stent/coil	Yes	60

[a] CN, cranial nerve; SAH, subarachnoid hemorrhage; BTO, balloon test occlusion; L, left; ICA, internal carotid artery; c, cavernous; H/A, headache; MCA, middle cerebral artery; R, right; PICA, posteroinferior cerebellar artery; TIA, transient ischemic attack; prox. occl., proximal occlusion; p, paraclinoid; ophth., ophthalmic; AICA, anterior inferior cerebellar artery; PCA, posterior cerebral artery; P1/P2, segments of the PCA; N/V, nausea/vomiting; BA, basilar artery; t, terminus; HH, Hunt and Hess grade. Age is at first treatment. Treatments are listed in the order performed.

aneurysm. Her mental status recovered after urgent cerebrospinal fluid diversion. Angiography subsequently demonstrated a giant P1/P2 segment aneurysm arising from a fetal right posterior cerebral artery (Fig. 1, A–C). This was treated with stent-assisted coil embolization (Fig. 1, D–F). Misplacement of the last coil segment within the parent vessel led to sluggish flow that resolved after the intra-arterial administration of a bolus of eptifibatide. Postoperative magnetic resonance imaging demonstrated patchy right occipital infarcts, and the patient was maintained on an eptifibatide infusion for 24 hours. Angiography performed 6 months later demonstrated complete aneurysm obliteration, with preservation of the right posterior cerebral artery (Fig. 1G). She made an excellent recovery, with only mild patchy visual loss (secondary to small right occipital lobe infarcts) (Fig. 1H) at follow-up examination.

Patient 10

An 82-year-old woman with a history of myocardial infarction and paroxysmal atrial fibrillation presented with right ptosis, hemifacial spasm, lateral gaze palsy, and hemibody paresthesias of several months' duration. Angiography demonstrated a giant midbasilar aneurysm arising just above the anterior inferior cerebellar artery (Fig. 2A). She had poor collateral supply to the posterior circulation and was not deemed a good candidate for surgical bypass. Given the preexisting mass effect, we aimed for hemodynamic effect with stent reconstruction of the parent trunk supplemented by partial coiling of the aneurysm. This led to marked stasis within the aneurysm during the procedure, with near obliteration seen on angiography 1 month later (Fig. 2B). She did well but then developed moderate gait ataxia after 4 months had elapsed. Aneurysm recurrence was found on angiography (Fig. 2C). A second stent was placed within the initial stent to further decrease porosity at the aneurysm neck, and further coils were placed within the aneurysm sac to promote thrombosis (Fig. 2D). Her gait ataxia and cranial nerve palsies continued to progress, and angiography 1 month later showed further aneurysm recanalization (Fig. 2E). A third coil embolization (Fig. 2F) was performed after magnetic resonance imaging showed continued brainstem compression but no infarction (Fig. 2, G and H), with the hope that aggressive packing might this time prevent recanalization and permit gradual resorption of her mostly thrombosed aneurysm. The patient's condition continued to deteriorate over the next month, despite high-dose steroid therapy, and, at the family's request, support was withdrawn. She died shortly thereafter.

Patient 15

A 46-year-old diabetic hypertensive man presented with severe headaches, leading to discovery of a giant right internal carotid artery (ICA) aneurysm on magnetic resonance imaging (Fig. 3, A–C). A computed tomographic scan and lumbar puncture disclosed no evidence of SAH. Angiography demonstrated fusiform involvement of the right ICA, which was deemed not favorable for stent reconstruction because of its stenosed/diseased appearance proximal and distal to the aneurysm neck (Fig. 3, D–F). After the patient had passed a balloon test occlusion of the right ICA, the aneurysm was endovascularly trapped (Fig. 3G), with excellent collateral flow noted to the right anterior cerebral artery and middle cerebral artery (Fig. 3, H and I) distal to the aneurysm. The patient developed right facial nerve palsy postoperatively, leading to exposure keratitis, which was treated medically. Follow-up imaging showed no aneurysm filling at 6 months.

Patient 18

A 58-year-old woman presented with a transient ischemic attack and worsening headaches. She was found to have a wide-necked,

TABLE 2. Patient outcomes[a]

Patient no.	30-day morbidity and mortality	Follow-up (months)		Status at last follow-up	GOS score at final follow-up
		Imaging	Clinical		
1	None	25	26	Symptoms mostly resolved	5
2	None	4	16	Asymptomatic	5
3	None	30	28	H/A resolved	5
4	SAH, PICA infarct, transient cortical blindness, CN VI palsy, R hemiparesis	63	64	Paresis, CN palsy resolved	4
5	None	—	42	Mild H/A	5
6	Transient hyponatremia necessitating readmission	—	33	H/A, CN deficit improved	4
7	Hemianopia	15	15	Hemianopia improving	5
8	None	31	37	Persistent CN palsy	5
9	None	25	25	Persistent visual field loss	4
10	None Worsening mass effect after each procedure	24 5	6	Brainstem edema, CN dysfunction, coma, deceased	1
11	Transient dysphasia, R drift	—	6	Lost to follow-up, died 2 y later	1
12	R basal ganglia stroke; second R MCA stroke	27	30	Dense L plegia, neglect, R hemianopia	3
13	Right-sided hemiplegia, aphasia, SAH, ICH	14	14	Mild dysphasia	4
14	L MCA stroke with dysphasia, hemiparesis	4	4	Mild dysphasia	3
15	R facial nerve palsy, R eye exposure keratitis	6	6	R facial palsy	5
16	Small postoperative R occipital lobe infarct	6	6	Mild visual symptoms	5
17	Postoperative 5-min TIA, rerupture after partial waffle-cone	—	—	Deceased POD 2	1
18	None	65	66	Asymptomatic	5
19	Hemiparesis/visual deficit after BTO (resolved with tPA), emboli during second procedure (resolved with tPA)	94	96	Intact	5
20	None	12	21	SAH, deceased	1
21	None	—	1	No long-term follow-up	5
22	Intraoperative rupture, death during last session	—	—	Deceased	1
23	Facial numbness	6	6	Increased facial numbness	5
24	Posterior circulation stroke	1	1	Deceased	1
25	None	6	6	Intact	5
26	None	25	25	Symptoms stable	5
27	L MCA hemorrhagic stroke	3	3	Worsened aphasia, lethargy	4
28	Postoperative L hemiparesis, R CN III palsy	—	27	Deceased	1
29	R parietal stroke, L U/E paresis	5	7	Paresis improving, walks with cane	3
30	Embolic stroke, mild expressive aphasia after second recoil	16	17	Persistent visual field loss caused by optic nerve compression	4
31	Intra-/postoperative rupture, care withdrawn, deceased <24 hr	—	—	Postoperative death	1
32	None	15	15	Cognitive deficits	4
33	MCA stroke at second session	20	20	Nursing home, deceased	1
34	Intraoperative rupture/deceased	—	—	Deceased	1
35	None	—	1	No long-term follow-up	5
36	None	0	4	Recovering from SAH	4
37	None	11	85	Good functional recovery from SAH	4
38	None	65	65	Rerupture, deceased	1

[a] GOS, Glasgow Outcome Scale; H/A, headache; SAH, subarachnoid hemorrhage; PICA, posterior inferior cerebellar artery; R, right; CN, cranial nerve; MCA, middle cerebral artery; L, left; ICH, intracranial hemorrhage; TIA, transient ischemic attack; POD, postoperative day; BTO, balloon test occlusion; tPA, tissue-type plasminogen activator; U/E, upper extremity.

TABLE 3. Demographics and outcomes summary[a]

Characteristic	Overview (mean ± standard deviation)			*P* value
	Total	Ruptured	Unruptured	
Aneurysms	39	10	29	
Patients	38	10	28	
Women	30.0 (79%)	7.0 (70%)	23.0 (82%)	0.41
Age (y)	60.9 ± 11.7	57.9 ± 13.9	62.0 ± 10.9	0.35
Parent vessel preserved	74%	90%	69%	0.41
Aneurysm 100% occluded	36%	20%	41%	0.28
Aneurysm 95% occluded or more	64%	60%	66%	1.00
Need for stent	66%	60%	68%	0.71
Sessions per aneurysm	1.9 ± 1.1	2.4 ± 1.4	1.8 ± 0.9	0.13
Sessional transient morbidity	9%	4%	12%	0.43
Sessional permanent morbidity	20%	8%	25%	0.12
Sessional mortality	8%	8%	8%	1.00
Cumulative morbidity	12 (32%)	2 (20%)	10 (36%)	0.45
Cumulative mortality	6 (16%)	2 (20%)	4 (14%)	0.64
Cumulative M&M	18 (47%)	4 (40%)	14 (50%)	0.72
Imaging follow-up (mo)	21.5 ± 22.9	18.9 ± 21.5	23.0 ± 24.1	0.69
Clinical follow-up (mo)	24.8 ± 24.8	30.3 ± 31.6	25.9 ± 23.4	0.69
Last follow-up GOS score	3.4 ± 1.7	2.6 ± 1.5	3.7 ± 1.7	0.08
Mortality at final follow-up	11 (29%)	4 (40%)	7 (25%)	0.43
Morbidity at final follow-up	10 (26%)	2 (20%)	8 (29%)	0.70
M&M at final follow-up	21 (55%)	6 (60%)	15 (54%)	1.00

[a] M&M, morbidity and mortality; GOS, Glasgow Outcome Scale. All percentages are rounded to the nearest integer. *P* values are for ruptured versus elective aneurysms.

giant, right cavernous aneurysm with erosion into the sphenoid sinus (Fig. 4, A–C), which was eventually obliterated with stent-assisted coil embolization. She has remained neurologically intact, with stable occlusion and a patent right ICA visible on angiography (Fig. 4, D–F) more than 5 years after diagnosis.

Patient 13

A 49-year-old woman presented with left-sided headaches and facial pain, leading to the diagnosis of a giant, partially thrombosed left middle cerebral artery aneurysm on magnetic resonance imaging, which was subsequently confirmed on angiography (Fig. 5A). During stent-assisted coil embolization, perioperative hemorrhage was encountered (Fig. 5B), resulting in aphasia and hemiplegia postoperatively. Her symptoms were managed medically, and she made a partial recovery during her extended hospital course (18 days). She continued to improve during rehabilitation and at 14 months was ambulating independently with only mild dysphasia; follow-up angiography at this time showed near-obliteration of her aneurysm (Fig. 5C).

DISCUSSION

We have found attempted endovascular treatment of giant aneurysms to carry an overall mortality of 29% and morbidity of 26%, for a cumulative morbidity and mortality of 55% over 24.8 ± 24.8 months of clinical follow-up. Although sobering to consider, these rates reflect better outcomes than the previously demonstrated natural history of these lesions (21) and a comparable outcome to open surgical series (3, 11, 28). Surgical series have reported good or excellent outcome rates ranging from 69 (3) to 75% (22). We experienced a cumulative all-cause good or excellent outcome rate (GOS score of 4 or 5) in 68% of non-SAH patients and in 50% of SAH patients. Of the SAH patient population, 50% presented with Hunt and Hess Grade 4 or 5 status, which doubtless contributed to their poor outcomes.

All data are presented in an intent-to-treat format and therefore reflect the eventual death of a patient in whom attempted treatment was aborted, without consequence, because of difficult anatomy, but who later experienced SAH and died. In fact, 45% (n = 5) of the observed deaths occurred beyond the 30-day follow-up period. Of these, 2 were secondary to delayed SAH, 1 occurred as a result of an unknown "stroke" event, and 2 occurred as a result of unknown causes. Two of the 5 patients were considered adequately treated (100 and ≥95% occlusion, respectively); the others had 2 partial embolizations and 1 aborted procedure (without complication). Assuming that the cause (known or not) of all delayed deaths was aneurysm rerupture, our data imply a 7.1% risk of SAH in 100% occluded aneurysms or an 8.0% risk for 95% or greater occluded aneurysms over the follow-up period. Although this SAH rate is better than the natural history of giant aneurysms (21, 32), it is by no means a "cure." With the well-demonstrated likelihood of aneurysm expansion and/or coil compaction, it is crit-

ical that studies of giant aneurysms contain delayed angiographic and clinical outcome data.

Our series contains more than twice the number of giant aneurysms reported in previous comparable endovascular investigations (Table 4). Still, despite having a smaller number of giant aneurysm patients and a slightly younger patient population, 2 of the larger studies published (7, 25), which also included diverse anatomic locations, demonstrated similar clinical outcomes for their giant aneurysm population (morbidity, 42 and 12%; mortality, 27 and 24%; GOS score, 3.2 and 3.8, respectively) (Table 2). Of the remaining series reported here (Table 4), 2 contained a limited number of patients (n = 4) (6, 8) and 2 were limited to selected subpopulations: vertebrobasilar (17) and ICA (16) (Table 4). In addition, parent vessel occlusion was used primarily as the treatment modality in all 4 of these studies (6, 8, 16, 17). There are other reports of giant aneurysms treated with endovascular repair as part of mixed series of variously sized aneurysms or as a report of select aneurysm subtypes (15, 18, 20, 24). For example, the landmark article by Murayama et al. (20) summarizing the first decade of the University of California, Los Angeles experience with Guglielmi detachable coil embolization mentions that 8% (73 aneurysms) were giant, of which 26% could be completely occluded. Specific mean age, presentation (SAH or otherwise), parent vessel status, length of clinical or angiographic follow-up period, morbidity, mortality, and clinical outcome for these giant aneurysms were not provided. Regrettably, the results of such studies are, therefore, not easily compared with our findings, because of either limited aneurysm locality, small number of giant aneurysms, or lack of sufficient detail to extrapolate comparative giant aneurysm data.

A mainstay of giant aneurysm treatment is parent vessel sacrifice (6, 8, 16, 17, 24) (Table 4). Cessation of the inciting inflow of a cerebral aneurysm often results in aneurysm thrombosis. Unfortunately, flow alteration does not always lead to aneurysm occlusion, and the natural history of the disease cannot be assumed to have been improved when the aneurysm remains

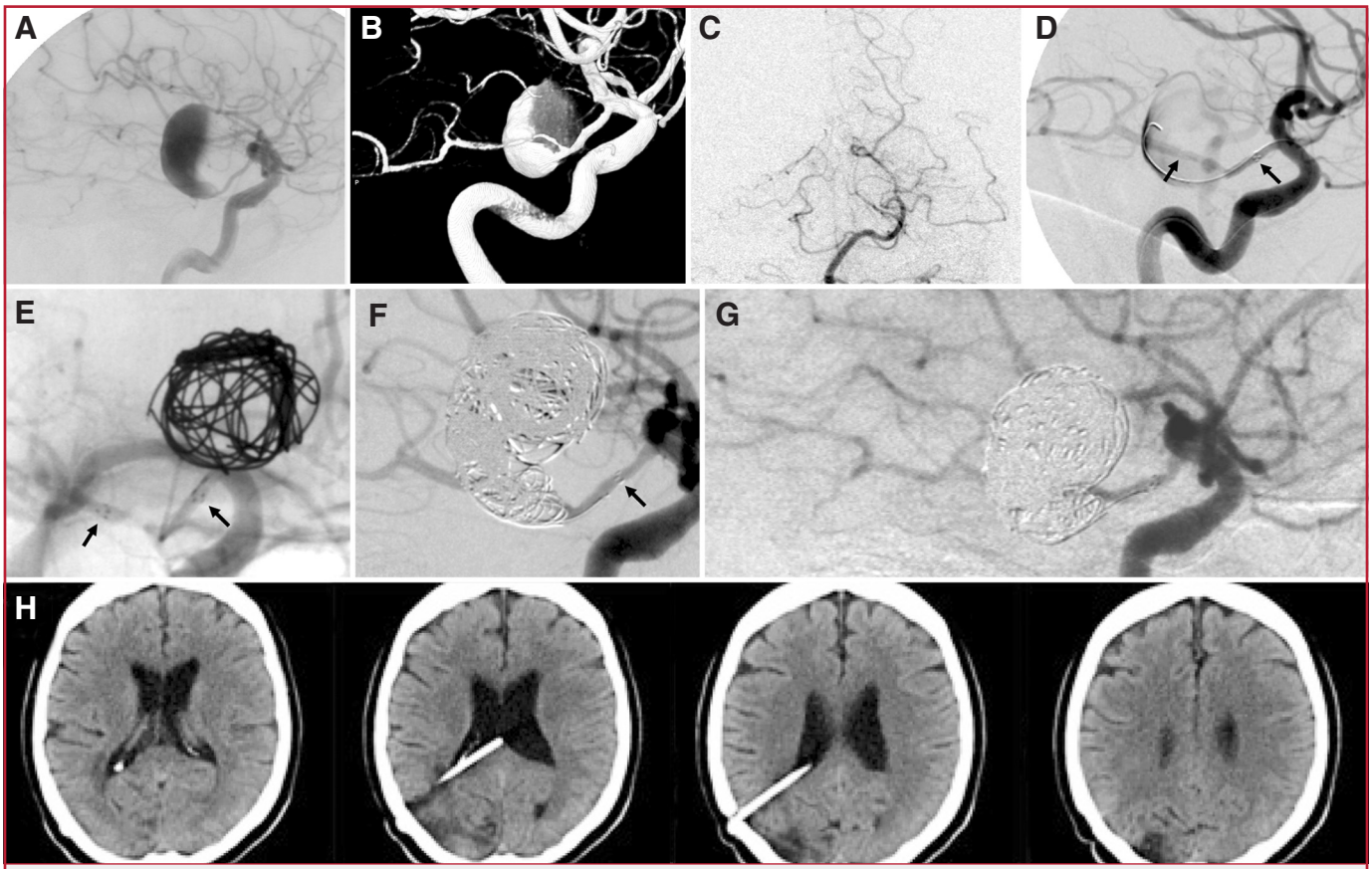

FIGURE 1. *Patient 16. Right carotid angiogram (**A**) and 3-dimensional reconstruction (**B**) demonstrating a giant partially thrombosed aneurysm arising from the right posterior cerebral artery (PCA). **C**, vertebral injection failed to opacify any portion of the right PCA or the aneurysm, suggesting true fetal origin of the right PCA. After stent deployment to ensure parent-vessel patency, the aneurysm was catheterized (**D**) and progressively embolized (**E**, midprocedure; **F**, final coil). Arrows in **D–F** indicate stent proximal and distal markers. **G**, angiogram at the time of the 6-month follow-up examination demonstrating complete patency of the right PCA, along with obliteration of the aneurysm. **H**, computed tomographic (CT) scans at the time of the 6-month follow-up examination showing residual patchy right occipital infarcts.*

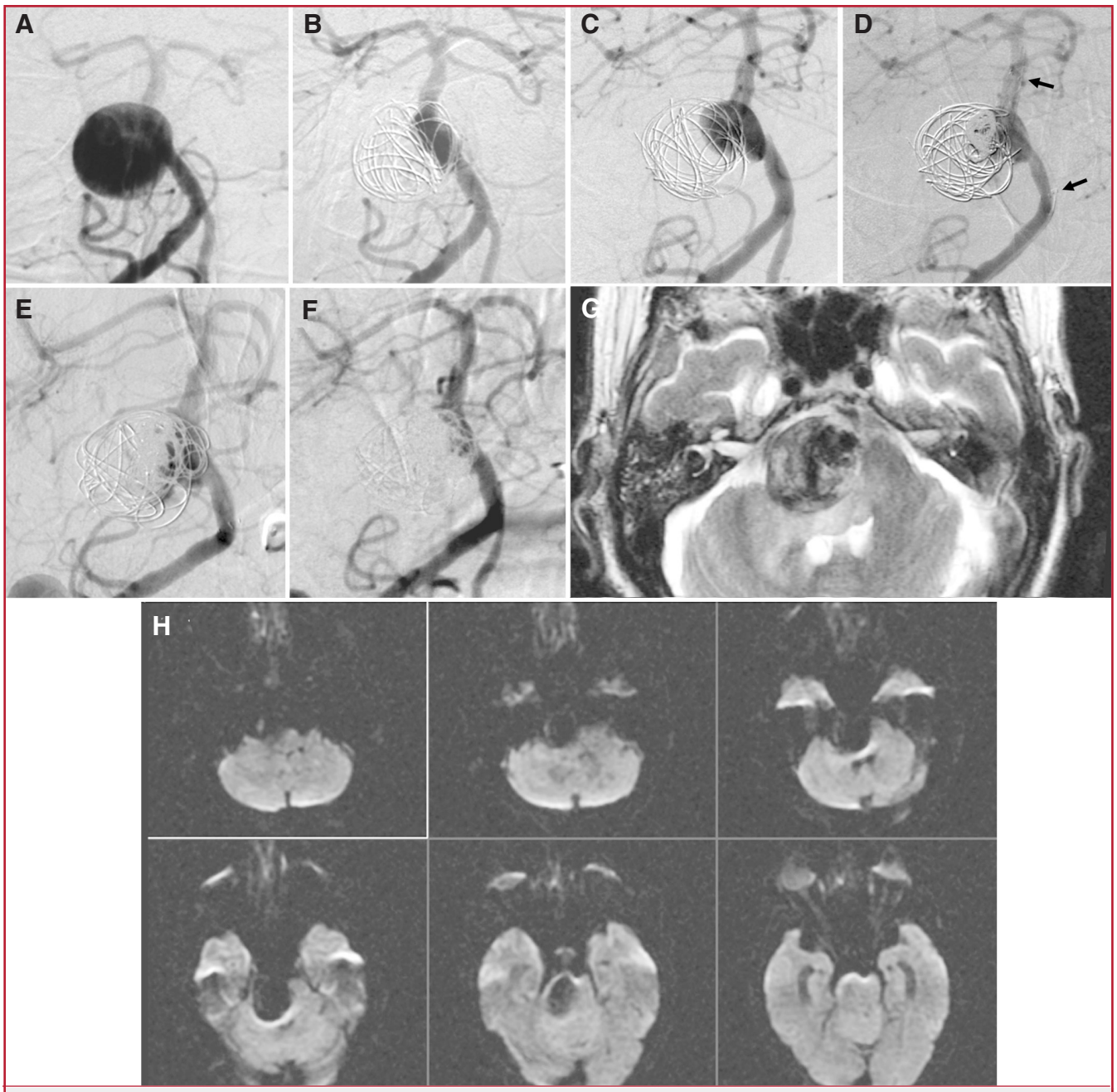

FIGURE 2. *Patient 10.* **A**, *right vertebral angiogram showing a giant midbasilar aneurysm with a poorly defined neck.* **B**, *angiogram 1 month after stent reconstruction of the midbasilar artery supplemented by partial aneurysm coiling. Much of the aneurysm appears thrombosed.* **C**, *aneurysm recanalization 4 months later.* **D**, *this was treated with placement of a second stent (arrows) and further coil embolization. The aneurysm recanalized again (***E***), and a third coil embolization was performed (***F***) 1 month later. T2- (***G***) and diffusion-weighted (***H***) magnetic resonance imaging (MRI) sequences demonstrate marked brainstem compression without evidence of infarction.*

patent. In addition, there are many patients who demonstrate an inability to tolerate parent vessel temporary balloon occlusion and who, as a result, are not candidates for parent vessel occlusion. Balloon- and stent-assisted coiling techniques, as well as better coil designs, have greatly improved the endovascular therapist's ability to coil giant aneurysms and still preserve the parent vessel. We experienced a parent vessel preservation rate of 74%, comparable to rates observed by Gruber et al. (7) (75%) and Sluzewski et al. (25) (65%) and much higher than those seen in other series (6, 8, 16, 17) (Table 4). The disparity among

studies likely reflects both the use of newer techniques as well as a fundamental shift in treatment goals. Some of the previous giant aneurysm series reflect a treatment paradigm wherein parent vessel occlusion was the primary or sole therapy. When primary coiling without planned vessel occlusion is the first-line intervention, with crossover to parent vessel sacrifice being an acceptable alternative should the patient pass test occlusion, we anticipate a parent vessel preservation rate of 65 to 75% on the basis of the findings of our series as well as those of Gruber et al. (7) and Sluzewski et al. (25). In our series, 8 of 10 (80%) parent vessel occlusions were intentional after successful test occlusion, whereas 2 were unanticipated adverse events.

It is important to note that a primary management paradigm of giant aneurysm embolization with parent vessel preservation versus vessel sacrifice (with or without surgical bypass) has been disputed (6, 8, 16, 17, 27). We attempt vessel preservation when technically possible (particularly in younger patients) because of the not insignificant risk of postoperative stroke even when patients pass balloon test occlusion (2, 9, 13). In addition, de novo aneurysm growth and long-term increased risk of SAH have been reported in patients after therapeutic sacrifice of the carotid artery (4, 29, 30), along with increased treatment-related risk should the contralateral carotid artery develop a symptomatic stenosis (5). Nevertheless, the role of parent vessel occlusion, as demonstrated by its use in 8 of our patients, should not be discounted. Indeed, in our series, 1 patient death occurred as a result of direct mass effect from a midbasilar aneurysm on the brainstem (Fig. 2; Tables 1 and 2 [Patient 10]). It is possible that proximal vessel sacrifice would have induced thrombosis of the aneurysm without exacerbating the mass effect; no balloon test occlusion was attempted because of the patient's limited collateral supply and advanced age.

Therapeutic carotid occlusion (with or without bypass) has also been used in treatment of giant cavernous aneurysms (31) and was used in 4 of 7 such aneurysms in our series. Although cavernous aneurysms generally have a lower risk of SAH, all cavernous aneurysms in our series were symptomatic, and their giant size made reliable exclusion of intradural extension difficult. Erosion of the cranial base by such aneurysms (Patient 18, Fig. 4) may also place patients at risk for massive or fatal epistaxis (9, 14). All 7 patients in our series had excellent outcomes, in keeping with the cavernous location of their aneurysms and tolerance of balloon test occlusion in 6 of 7 patients. Excluding these patients, the overall mean GOS score

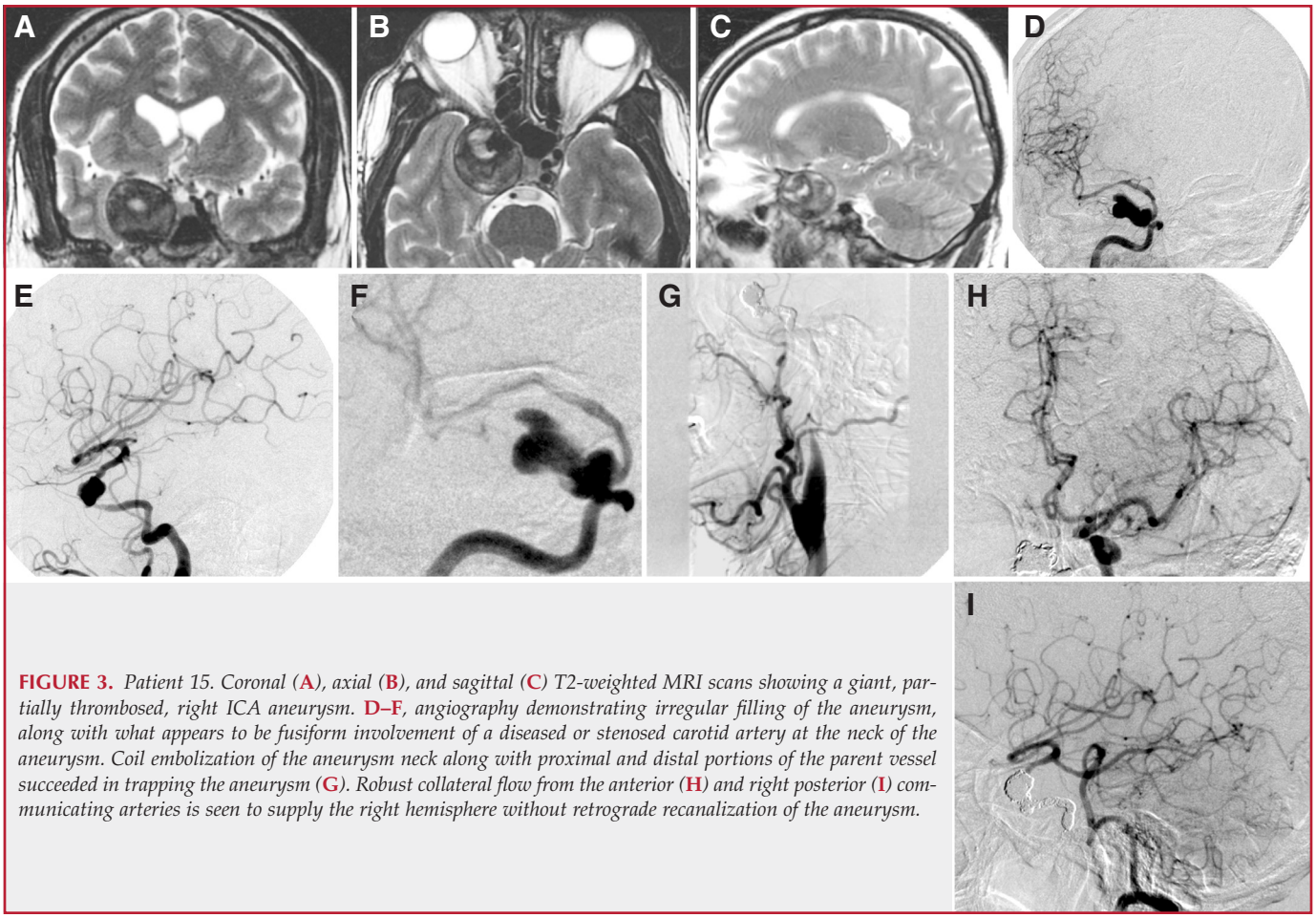

FIGURE 3. *Patient 15. Coronal (**A**), axial (**B**), and sagittal (**C**) T2-weighted MRI scans showing a giant, partially thrombosed, right ICA aneurysm. **D–F**, angiography demonstrating irregular filling of the aneurysm, along with what appears to be fusiform involvement of a diseased or stenosed carotid artery at the neck of the aneurysm. Coil embolization of the aneurysm neck along with proximal and distal portions of the parent vessel succeeded in trapping the aneurysm (**G**). Robust collateral flow from the anterior (**H**) and right posterior (**I**) communicating arteries is seen to supply the right hemisphere without retrograde recanalization of the aneurysm.*

of the remaining patients was 3.1, which was not statistically different from that of the entire series ($P = 0.39$). Similarly, excluding all cavernous and petrous giant ICA aneurysms did not significantly affect the overall outcome of the remaining patients in our series (GOS score of 3.0; $P = 0.39$).

A potential indication of progress with endovascular techniques for giant aneurysm treatment is our relatively high aneurysm occlusion rate compared with the experience of Gruber et al. (7) and Sluzewski et al. (25) (100% occluded, 36% in our series versus 12.5% and unknown, respectively; ≥95% occluded, 64% in our series versus 50 and 25%, respectively) (Table 4). Although our occlusion estimation is based on final angiographic assessment alone, it should be stressed that endovascular treatment of giant aneurysms is a dynamic process, with aneurysm expansion and coil compaction not infrequently converting 95% or more or 100% occluded aneurysms to lower percentage-of-treatment aneurysms. Indeed, 1 of our patients with presumed 100% occlusion eventually experienced SAH. Therefore, we report the degree of occlusion and cumulative mortality and morbidity after each patient's last treatment session. Whether further embolization sessions are recommended depends on clinical and angiographic factors specific for each patient. Our overall rate of aneurysm occlusion is the highest of all giant aneurysm-specific series with primary parent vessel preservation. We hope that higher occlusion rates will lead to better long-term treatment outcomes but emphasize the need for diligent long-term clinical and angiographic follow-up in these patients.

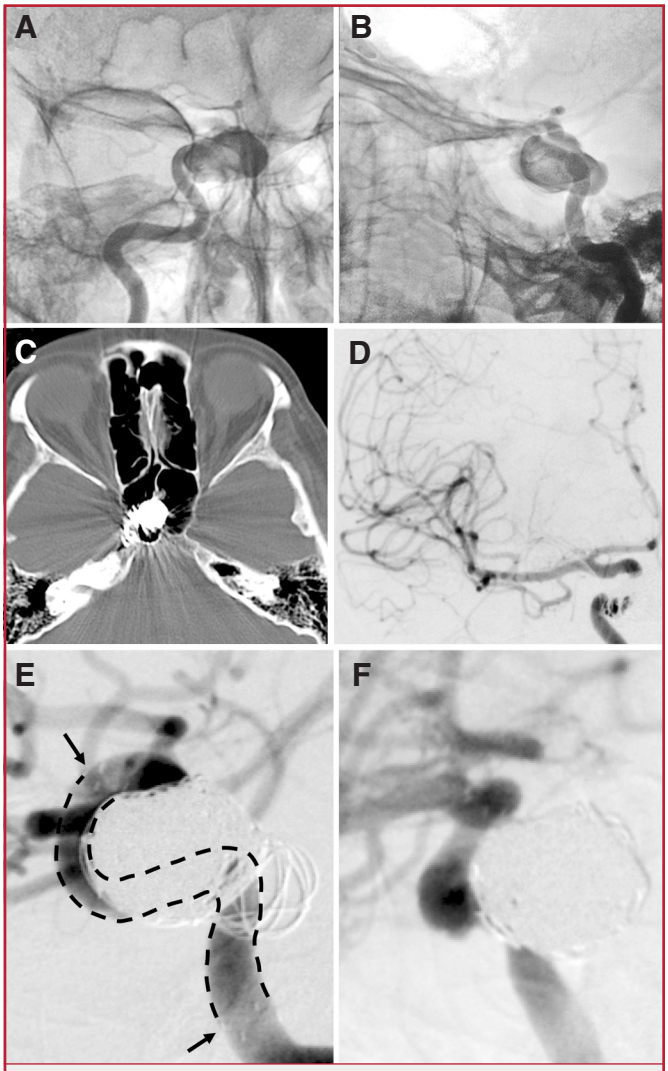

FIGURE 4. *Patient 18. Anteroposterior (**A**) and lateral (**B**) unsubtracted angiograms demonstrating erosion of a right cavernous ICA aneurysm into the sphenoid sinus, further highlighted by the location of the coils on the postoperative CT scan (**C**). Anteroposterior (**D**) and lateral (**E**) images showing obliteration of the aneurysm, along with a patent parent vessel. The dotted line marks a segment of the stented carotid artery obscured by the coil mass, whereas the arrows denote proximal and distal stent markers. **F**, oblique view demonstrating lack of any significant aneurysm neck residual.*

Although no significant differences existed in outcome between ruptured and unruptured aneurysms in our series, there were some interesting nonsignificant differences that may have reached significance with larger numbers. As expected, patients with unruptured aneurysms had better overall GOS scores and lower mortality. Interestingly, these patients did experience higher morbidity, but this is likely because a poor premorbid condition masked postprocedural deficits in those with SAH. Also, as a group, the SAH patients required almost 1 full treatment session more per aneurysm. This is probably secondary to our preference of avoiding stent deployment, and its associated antiplatelet therapy, for wide-necked aneurysms when possible in patients immediately after SAH because they may require other high-risk procedures (e.g., external ventricular drains, lumbar drains, and ventriculoperitoneal shunts). One could consider parent vessel occlusion instead of stent-assisted coiling under such circumstances; however, acute parent vessel occlusion in the setting of SAH is limited by frequent inability to perform adequate balloon test occlusion as well as loss of both endovascular access and collateral flow should symptomatic vasospasm subsequently occur. As a result, in patients with wide-necked giant aneurysms, we were more cautious in how aggressively we would fill an aneurysm, in some cases returning 1 to 2 weeks after the SAH ictus for further stent-assisted embolization of a residual neck left behind for parent vessel protection.

Aneurysms required an average of 1.9 treatment sessions to achieve our reported end result. Consequently, per-session permanent morbidity and mortality were substantially lower than the overall cumulative morbidity and mortality (28 versus 55%) (Table 3). When comparing attempted endovascular treatment with the natural history of disease, it is the overall total treatment-related morbidity and mortality that are the most important factors; per-session results were derived as an aid to the patient and physician about to embark on an embolization attempt by providing an estimate of risk incurred for that spe-

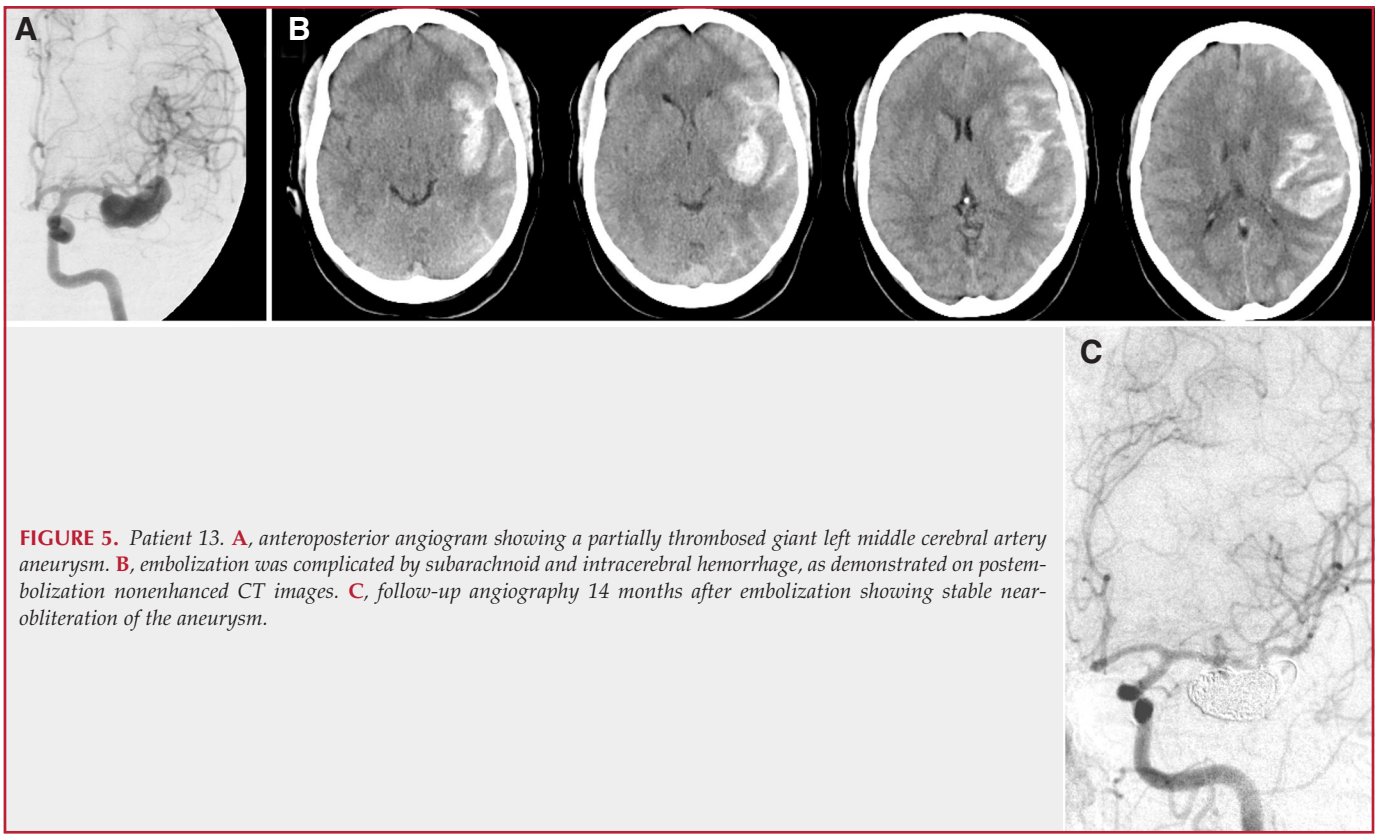

FIGURE 5. *Patient 13.* **A**, *anteroposterior angiogram showing a partially thrombosed giant left middle cerebral artery aneurysm.* **B**, *embolization was complicated by subarachnoid and intracerebral hemorrhage, as demonstrated on postembolization nonenhanced CT images.* **C**, *follow-up angiography 14 months after embolization showing stable near-obliteration of the aneurysm.*

cific episode, as well as providing a measure for assessing particular techniques or strategies used during each session. It should be emphasized that although more than 1 session is often required (i.e., because of coil compaction or aneurysm regrowth), this does not imply a deliberate strategy of staged intervention for all patients, but rather each session attempts to maximize embolization as deemed safe. Patients should be made aware during initial treatment discussions that giant aneurysms often require repeated efforts at treatment. They are, in essence, harboring a chronic disease that may require multiple treatment sessions and will certainly require a significant course of follow-up well beyond the standard period of 12 to 24 months.

Our work has significant limitations:

1) Although prospectively collected in databases, our data remain subject to the biases of retrospective review.
2) Given the tertiary high-volume endovascular referral nature of the centers involved, our results may not be applicable at other centers (11, 26).
3) Seven (18%) of the aneurysms treated were classified as "cavernous." Although cavernous aneurysms are often not treated because of their low risk of SAH, all 7 patients were symptomatic. All 7 had excellent outcomes, potentially because of the higher tolerance for (and use of) parent vessel occlusion in these cases. Such uniformly excellent results are not likely when treating giant aneurysms at other locations and were not achieved across the remaining aneurysms in our series. Nevertheless, exclusion of cavernous aneurysms from analysis did not significantly worsen the outcome of the remaining patients (representing 82% of our series as described previously, under Discussion).
4) Because no randomization to surgery or observation was performed, our data cannot directly address the relative superiority or inferiority of an endovascular approach to giant intracranial aneurysms and only portray a snapshot of current techniques and their associated results. Giant intracranial aneurysms remain formidable lesions, and it is possible that, in expert hands, surgical ligation with or without bypass would have had more favorable results. Observation alone may be a reasonable option in patients with limited life expectancy or major comorbidities. A large number of our patients were aged 65 years or older, although all had symptomatic aneurysms, which portends a very poor natural history when left untreated.
5) As previously mentioned, giant aneurysm embolization is a dynamic process. Our occlusion rate is only that for the last known angiogram and does not necessarily represent the "durability" of the final degree of aneurysm obliteration.
6) Consequently, with the associated morbidity and mortality that comes with this therapy, it is likely that some of these patients will require further treatment in the future. Thus, our results are not necessarily a static representation of the

TABLE 4. Previous investigations of endovascular management of giant intracranial aneurysms[a]

Series (ref. no.)	Anatomic locations	No. of giant aneurysms	Mean age (yr)	SAH (%)	Parent vessel occlusion (%)	Angiographic follow-up (mo)	100% occlusion (%)	≥95% occlusion (%)	Clinical follow-up (mo)	Morbidity (%)	Mortality (%)	Clinical outcome (GOS score)
Gobin et al., 1996 (6)	All	4	37 ± 11	0	75	Unknown	Unknown	Unknown	7.5 ± 3.0	0	0	4.5 ± 1.0[b]
Gruber et al., 1999 (7)	All	12	52 ± 12	42	25[b]	17 ± 21	12.50	50	21 ± 22[b]	42	27	3.2 ± 2.0[b]
Sluzewski et al., 2003 (25)	All	17	52 ± 10	41	35[b]	12 ± 12	Unknown	25	32 ± 20	12	24	3.8 ± 2.0[c]
Lubicz et al., 2003 (16)	ICA	18	42 ± 17	17	94	31 ± 16 (MRA)	Unknown	Unknown	31 ± 16	Unknown	6	Unknown
Lubicz et al., 2004 (17)	Vertebrobasilar	13	48 ± 15	31	100	28 ± 14 (MRA)	8	8	28 ± 14	31	8	4 ± 1.1
Hassan et al., 2004 (8)	Basilar	4	42 ± 16	25	100	Unknown	100	100	Unknown	0	0	4 ± 0

[a] SAH, subarachnoid hemorrhage; GOS, Glasgow Outcome Scale; ICA, internal carotid artery; MRA, magnetic resonance angiography.
[b] Best estimate from data or descriptors.
[c] Reported for SAH patients only.

eventual treatment burden of these patients, which is best represented by cumulative, rather than sessional, morbidity and mortality.

7) The natural history of small aneurysm remnants encased within a large coil volume is not known; whether such aneurysms enjoy significant reductions in rupture rates or whether further intervention would be of additional benefit is not clear.
8) Lastly, our data roughly cover a 5.5-year period during which technological advances may have affected our results. Unfortunately, the limited number of patients precluded further stratification by year. It remains to be seen whether future advances will improve future clinical and radiographic outcomes.

CONCLUSION

We have presented, to our knowledge, the largest series to date evaluating outcome after consecutive giant intracranial aneurysms treated with endovascular repair. Giant intracranial aneurysms carry a devastating natural history as well as a high risk for intervention, whether via endovascular or open surgical means. We believe endovascular therapy can provide a useful alternative to open surgical procedures, particularly in patients with systemic comorbidities and excessive surgical risk. Nevertheless, microsurgical treatment (or no treatment at all) remains a serious option for an individual patient, after consideration of factors such as age, neurological condition, and other comorbidities. Further angiographic long-term follow-up is required to assess the durability of endovascular treatment (with parent vessel preservation) for giant aneurysms.

Disclosures

L. Nelson Hopkins, M.D., receives grant support from Boston Scientific, Cordis, and Micrus; and honoraria from Bard, Boston Scientific, and Cordis; has an ownership interest in AccessClosure, Boston Scientific, Micrus, and Square One, Inc.; and serves as a consultant to/member of the advisory board for Abbott, Bard, Boston Scientific, Cordis, and Micrus. Elad I. Levy, M.D., receives grant support, other research support (devices), and honoraria from Boston Scientific; holds shares in Micrus Endovacular; receives grant support from ev3; serves as a consultant to Cordis Neurovascular and Micrus Endovascular; and receives fees for carotid stent training from Abbott Vascular and ev3. J Mocco, M.D., M.S., has received a research grant from the Brain Aneurysm Foundation. The remaining authors have no potential financial conflicts of interest to disclose. Adnan H. Siddiqui, M.D., Ph.D., has received a research grant from the University at Buffalo and honoraria from Genentech, an American Association of Neurological Surgeons' course and an Emergency Medicine Conference.

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COMMENTS

Jahromi et al. report on endosaccular treatment of 39 aneurysms at two different centers. Although it is known, on the basis of sporadic case reports, that endosaccular occlusion results in complications and poor long-term occlusion and outcome, other modalities such as parent vessel occlusion with or without bypass and perhaps even newer stent technology offer different options and perhaps will result in better outcomes.

However, regarding the experience of Jahromi et al., it was quite sobering to see that 20% of the interventions resulted in permanent morbidity and death within 30 days in 8% of the treated patients. In 64% of patients, 95% occlusion was documented and 100% occlusion was documented in 36%, but in giant aneurysms the hemodynamic change clearly resulted in recanalization virtually 100% of the time. In fact, with current endosaccular technology and the experience of Jahromi et al., the cumulative morbidity and mortality were 55% over 24 months of clinical follow-up.

As Jahromi et al. indicated, these results are better than the natural history of these symptomatic lesions left untreated. It is clear, however, that these lesions are formidable whether they are treated with endovascular technology or direct surgical repair of the vessel. In many of the cases that the authors illustrated, parent vessel occlusion with or without bypass is a reasonable alternative and probably is underutilized in these types of patients.

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Jahromi et al. critically reviewed their combined experience in the endovascular management of giant intracranial aneurysms. These aneurysms remain a vexing problem to both endovascular neurosurgeons and microsurgeons. As this article validates, these patients often require multiple treatment sessions and therefore are subjected to higher risks. These risks are only compounded by the severity of their cerebral vascular pathological conditions. The results of this series are even more sobering when one considers the types of aneurysms treated.

Jahromi et al. treated 7 patients with giant cavernous segment aneurysms, 6 of whom had successfully passed balloon test occlusion. All 7 of these patients went on to have excellent outcomes with Glasgow Outcome Scale (GOS) scores of 5. One would expect that patients with cavernous aneurysms would have a low rate of morbidity and mortality after treatment, especially if they have passed balloon test occlusion. Removing these patients from this series would render the morbidity and morality rates even higher than currently presented. This study underscores the need for careful patient selection for appropriate endovascular or microsurgical treatment. The rapid evolution of endovascular technology, however, may eventually lessen the treatment-associated risks of these lesions.

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Phoenix, Arizona

In this article, Jahromi et al. have presented the clinical outcome of 38 patients with 39 giant aneurysms treated by endovascular means, including parent vessel occlusion, coiling, and stent-assisted coiling. There were 10 aneurysms that were ruptured, and 29 that were unruptured. This may be the largest reported series of endovascular treatment of giant aneurysms and is very important. Jahromi et al. have provided an honest presentation and discussion of their results.

The results of this series must be viewed from the perspective that the natural history of unruptured giant aneurysms is very bad and that of ruptured giant aneurysms is also poor. The results of microsurgical treatment have improved, primarily because of bypass with proximal occlusion/trapping. However, treatment mortality and morbidity are significant. Very few North American centers now have the experience of Charles Drake or other such surgeons. The reason is that giant aneurysms in unselected series are uncommon (3% in our experience of approximately 600 patients treated in a 3-year period) and may be becoming less common owing to earlier detection of ruptured and unruptured aneurysms in the United States population. This fact becomes obvious when one looks at the experience of surgeons from countries such as India, where the incidence of giant aneurysms in referral centers is much higher.

However, the results of endovascular treatment of these aneurysms in this series were disappointing. The best group of patients were those with intracavernous aneurysms (7 patients), who had favorable results. Of the remaining 31 patients, one-third died, one-third had strokes, and one-third did well as a result of treatment. Regarding paraclinoid aneurysms, 8 patients were treated. Of these, 2 died and 5 had strokes, including the 2 who died. Coiling in 1 patient failed, and 1 patient had 70% occlusion, which indicates treatment failure (the coils will eventually compact). Of 5 patients with 6 carotid ophthalmic aneurysms, 1 died and 2 had strokes; one recovered to a GOS score of 3 and another to a GOS scale of 4. All 4 patients with basilar tip aneurysms died. One 39-year-old patient with a basilar tip aneurysm had 4 treatment sessions (coil, coil, attempted coil, and stent/coil) and, despite them, had a rerupture and died. Should this patient have had attempted clipping or terminal basilar occlusion with bypass? Many patients treated in this series were older than 65 years. Would some of these patients have done better without any treatment?

How does a center accumulate the kind of results that allow equal consideration of microsurgical and endovascular treatment options of these difficult cases? Most centers are likely to have some kind of bias, based on their expertise and experience.

Newer endovascular technologies (such as the "pipeline stent") are being introduced and may advance the results of treatments of giant aneurysms. Until then, this article will remain as a benchmark for endovascular treatment results.

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Giant aneurysms have not been favorable lesions for endovascular therapy because they frequently broaden the neck, distort the anatomy of parent and branch arteries at the base, and induce intraluminal thrombosis. These anatomic 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. New technologies and techniques have advanced the efficacy and scope of endovascular therapy, and, in this report, Jahromi et al. examined whether these advances translated to improved endovascular outcomes.

In a combined experience from Buffalo and Pittsburgh, 38 patients with 39 giant aneurysms were managed endovascularly with sophisticated techniques that included staged therapy in 21 patients, stents in 66% of patients, parent artery reconstructions with overlapping stents, and aggressive "waffle cone" coiling in selected cases. Complete angiographic occlusion was achieved in just 36% of aneurysms. Follow-up was short (mean duration, 25 months), which can result in underestimates of rates of recurrence, retreatment, and complications. Treatment-related morbidity occurred in 32% of patients, and treatment-related mortality occurred in 16%. Overall, 29% of patients were dead at the last follow-up examination. When these results are compared with other endovascular reports in the literature, they demonstrate significant forward progress. However, contemporary microsurgical experiences from experienced neurosurgeons show combined morbidity and mortality rates of approximately 20%, indicating that endovascular therapy is not ready to replace microsurgery as the treatment of choice for giant aneurysms.

Jahromi et al. clearly state that their effort was not meant to demonstrate superiority of one therapy over another, but this article implicitly challenges the microsurgical management of giant aneurysms. In my experience, most anterior circulation giant aneurysms can be favorably treated microsurgically because surgical exposure is excellent and bypass options abound. In contrast, most posterior circulation giant aneurysms (except posterior inferior and superior cerebellar artery aneurysms) are less favorable for microsurgery because surgical exposure is limited and brainstem perforators are a dangerous problem. The endovascular progress showcased in this report is welcomed for tough aneurysms at posterior circulation sites. Stents and stent-assisted techniques clearly have had an impact on the outcomes of endovascular therapy, and I am encouraged by these developments. However, 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. Microsurgical aneurysm occlusion still appeals to many patients who prefer a single, definitive therapy. Jahromi et al. have discussed their technical innovations with an honest reporting of their results which, at present, do not portend retirement for vascular neurosurgeons.

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