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Preliminary results of embolisation of nonsurgical intracranial aneurysms with GD coils: the 1st year of their use

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Abstract We report 28 patients with nonsurgical intracranial aneurysms referred by neurosurgeons for endovascular treatment using platinum detachable coils (GDC, Target Therapeutics, Fremont, Calif., USA). Because of unfavourable anatomical features or failed hyperselective catheterisation, 4 patients were not treated. In the other 24 patients (26 aneurysms) embolisation was successful. No manufacturing defect was detected in the coils used. Complete occlusion of the sac was obtained in 22 of the 26 aneurysms (85 %) regardless of their size. The glasgow outcome score was good on discharge in 20 of the 24 patients (83 %). The Karnovski score at 3 months after embolisation revealed that 20 patients (83 %) lead a normal life, with minor neurological signs. Morbidity related directly to treatment occurred in 2 cases (8 %): a superior hemianopia due to mass effect on the optic nerve from a carotid-ophthalmic aneurysm, and hemiparesis due to an embolus. Five transient deficits lasting hours to a few days were related to proven or suspected emboli. No death occurred. Anatomical, technical and clinical data are reported; the mean cost of materials (catheters and coils) per patient and aneurysm was FF 13500. The mean hospitalisation time was 6.5 days in the neurosurgical department with 2.8 days of intensive care.

Key words Arterial aneurysm · Guglielmi detachable coil · Embolisation

Introduction

The classical treatment for intracranial aneurysm is still surgical clipping, which is of proven efficacy. The endovascular approach to aneurysms has recently been developed; the use of Guglielmi detachable coils (GDC) [1–3] is currently considered the most efficient embolisation technique for these lesions, allowing preservation of the parent vessel and selective exclusion of the sac. We were asked to participate in an international trial of the method, its efficacy and safety and to treat with GDC only aneurysms considered as nonsurgical by the referring groups, because of medical (clinical status, myocardial infarct) or surgical reasons (location of the lesion, anticipated surgical or technical difficulties, previous failed clipping). Our aim is to present our results after the 1st year of use of GDC and to emphasise the logistics and decisions to be made in management of these lesions.

Materials and methods

Clinical (Table 1)

During 12 months, 28 consecutive patients shown to have at least one nonsurgical intracranial aneurysm (or at a high surgical risk) were referred for endovascular treatment using GDC. The 12 men and 16 women were from 32 to 69 years old (median 47.7 years). The reasons for exclusion from surgical treatment were: location of the lesion (11 cases), anticipated surgical difficulties (9 cases),

failed surgery (4 cases), unfavourable clinical status after subarachnoid haemorrhage (Hunt and Hess grade III) or difficult anaesthetic management for direct surgery (severe high blood pressure and diffuse atherosclerosis, chronic alcoholism).

The surgical contraindications, the alternative endovascular therapy and the possible adverse effects of embolisation were explained to each patient (or to the family, 4 patients being drowsy, 1 stuporous and 1 comatose. Informed consent was obtained in all cases before enrolment; the study received approval from our ethics committee. In all patients, initial and follow-up examination included general and neurological examination, Hunt and Hess score (Table 2), Karnovsky score on admission to our hospital, on discharge (Table 3), at 3 months and 1 year, and Glasgow outcome score on discharge (Table 4). Follow-up angiography was performed at 3, 6 and 12 months after the initial procedure; the aneurysm was again embolised with GDC during one of these sessions if needed for e.g., coil compaction).

Technical aspects

All the procedures were performed under general anaesthesia, the patient being given antibiotics (oxacillin 25 mg/kg per day) for 2 days after the embolisation. A 6F introducer was placed in the femoral artery. The examination began with an angiographic study of the arteries of the base of the brain, including demonstration of the perforating arteries. A 6 F guiding catheter was then positioned in the main arterial trunk harbouring the aneurysm (internal carotid or vertebral artery) and the patient was given intravenous heparin (50 IU/kg) to avoid clotting during the procedure. For the same reason, continuous coaxial saline solution flushing (without heparin) into the guiding catheter and microcatheter was routinely used. Superselective catheterisation of the lesion was then performed.

All the aneurysms were approached using a microcatheter and occluded with 0.010″ or 0.018″ platinum GDC. With the first coil we tried to obtain a wide knitted cage ("basket"), using a coil whose diameter was equal to or slightly greater than the largest diameter of the aneurysm. The high flexibility of the coil allowed a multidirectional coil to be formed inside the sac, preserving the lumen of the parent artery. Smaller coils were then deposited within the basket to achieve optimum compactness. Before detaching each coil, we assessed and documented the stability of the system and the patency of the parent artery. Detachment was performed by applying a 1 mA electric current, produced by a direct current generator, between the proximal portion of the coil mandril and a needle in the groin, the latter being the negative electrode. Detachment, due to electrolysis, occurred within 2–14 min and was indicated by both a progressive increase in voltage and a sudden decrease in current below 1 mA. Several coils were placed to pack the aneurysmal lumen satisfactorily. The microcatheter was then carefully removed under fluoroscopy so that the coils were not dislodged. We observed no secondary displacement or migration of GDC.

Our first 3 patients were not treated with heparin during the immediate postoperative period. Because of clotting problems, we felt that full heparinisation should be maintained for 24 h to avoid any early embolus originating from the sac itself. The introducer sheath was left in place during this time and removed after the reversal of heparinisation. This did not lead to any local complications, but provided a rapid endovascular access if needed for fibrinolytic procedures. After the first 24 h the patients were finally put on antiaggregant therapy (aspirin 250 mg/day) for 6 months, to avoid platelet embolus originating from the coiled aneurysm, which could produce a transient ischaemic attack. After 1 day, plain X-ray films of the skull were obtained to documented the stability of the coils.

All patients with ruptured aneurysms were treated in the acute phase with calcium-channel blockers. In a few cases, steroids were given when increased mass effect from the coils was feared.

Results

There were four types of presentation: subarachnoid haemorrhage (SAH), mass effect, incidental discovery and transient neurological deficit.

The initial Hunt and Hess grade of 11 patients with *ruptured aneurysms* is shown in Table 2. Five were taken over during the 1st month after the initial SAH; the other 6 underwent embolisation 3.5 months–6 years after the initial bleed, independently of our own protocol, in accordance with the decisions of the referring centres.

Of the 8 patients referred for mass effect, 6 had an intracavernous aneurysm and complained of unilateral blindness or a nasal or temporal hemianopia (3 cases), oculomotor nerve palsy (2 cases) or trigeminal pain (1 case). One patient had hydrocephalus due to a giant A1 aneurysm compressing the third ventricle and the interventricular foramen; another with a basilar aneurysm compressing the cerebral peduncle had a hemiparesis.

Incidental discovery of an aneurysm occurred in 6 patients with vertigo (2 cases), memory disturbance, headache, a microprolactinoma or carotid stenosis.

Two patients had a transient neurological deficit (hemiparesis) thought to be caused by an embolus originating from the aneurysm. One further patient had a transient ischaemic attack from distal arterial occlusive disease, apparently not directly related to the aneurysm.

Embolisation could not be performed in 4 of our 28 patients (14 %). In one of our first patients (case 6), technical problems (difficult catheterisation with proximal bending of the microcatheter and decrease in flow of saline flush) and dangerously increased friction led us to abandon the attempt. In another (case 17), the P1 segment of the posterior cerebral artery and the superior cerebellar artery arose directly from a basilar tip aneurysm (Fig. 1), and it was feared that occlusion of the aneurysm would lead to stroke. In patient 18, a carotid aneurysm 2 mm in diameter could not accept a 3 mm coil; 2 mm coils were not then available. In case 28, an aneurysm 25 mm in diameter on the A1 segment could not be entered safely because of diffuse vascular tortuosity. In none of these 4 cases was any direct adverse clinical effect noted as a consequence of our attempt, although one grade V patient died 2 days later.

We were able to embolise 26 aneurysms in 24 patients. Of these, one was giant (> 25 mm), 7 ranged from 11 to 24 mm, 11 from 5 to 10 mm, 7 were less than 5 mm

Table 1 Clinical and radiological details (*SAH* Subarachnoid haemorrhage, *A1* A1 segment anterior cerebral artery, *TIA* transient ischaemic attack, *AICA* anterior inferior cerebellar artery)

Patient	Sex	Age (years)	Symptoms	Site of aneurysm	Sac (mm)[b]	Neck (mm)	Reason for exclusion from surgery	% occlusion[c]	Coils (cm)[d]	Outcome and complications	Glasgow outcome score on discharge
1	M	66	SAH (grade III)[a]	AICA	5	3	Surgical failure	100	40	Asymptomatic	5
2	F	60	SAH (grade I)	Carotid ophthalmic	14	4	Anticipated difficulties	70	100	Transient monoparesis	5
3	F	34	Hemiparesis	Basilar tip	12.5	3	Site	100	200	Complete recovery	5
4	F	57	Transient deficit	Carotid ophthalmic	6.7	2.5	Partly intracavernous	100	80	Asymptomatic	5
5	M	40	Incidental discovery	A1	4	2.5	Clinical judgement	80	15	Asymptomatic	5
6	M	69	SAH (grade V)	Anterior communicating	6.5	2	Clinical judgement	0	0	Dead 2 days later	1
7	M	39	Unilateral blindness	Cavernous carotid	30	8	Site	90	320	Unchanged	5
8	M	49	Incidental discovery	Basilar tip	9.4	3	Site	90	95	Asymptomatic	5
9	F	59	Unilateral hemianopia	Carotid-ophthalmic	10	3	Anticipated difficulties	90	135	Transient hemiparesis	5
10	M	40	Trigeminal pain	Carotid-ophthalmic	14	3	Anticipated difficulties	90	250	Improved	5
11	M	47	Incidental discovery	Basilar tip	7.5	2	Site	90	145	Asymptomatic	5
12	F	68	Oculomotor nerve palsy	Cavernous carotid	15	5	Site	90	250	Transient monoparesis cranial nerve improvement	5
13	F	38	SAH (grade I)	Carotid-ophthalmic Carotid-ophthalmic	9 6	4 2	Surgical failure	100 100	160 50	Asymptomatic	5
14	M	40	SAH (grade II)	AICA	5.6	1.5	Surgical failure	90	15	Asymptomatic	5
15	M	37	SAH (grade IV)	Anterior communicating	8	2	Clinical judgement (grade 4; hemiplegia)	90	140	Hemiplegia (partial improvement)	3
16	51		SAH (grade III)	Basilar tip	11	4	Site	90	110	Transient aphasia	5
17	F	54	Incidental discovery	Basilar tip	8	7	Site	0	0	Asymptomatic	5
18	M	53	TIA	Posterior communicating	2	1	Anticipated difficulties	0	0	Asymptomatic	5
19	F	51	SAH (grade I)	Anterior communicating	9.5	2.5	Anticipated difficulties	90	90	Dead (septicaemia)	1

Table 1 Continued

20	M	36	SAH (grade III)	Basilar tip	5	2	Site	100	40	Asymptomatic	5
21	F	32	Incidental discovery	Carotid-ophthalmic	6	1.5	Anticipated difficulties	100	55	Superior ipsilateral hemianopia	5
22	F	49	SAH (grade II)	Basilar tip Basilar tip	12.7 4	3 3	Site	80 90	100 15	Hemiparesis	4
23	F	35	SAH (grade III)	Basilar tip	5.6	2	Site	100	55	Asymptomatic	5
24	M	49	Incidental discovery	Anterior Communicating	5	2	Anticipated difficulties	100	85	Asymptomatic	5
25	F	54	Unilateral temporal hemianopia	Carotid-ophthalmic	12.3	3	Anticipated difficulties	90	280	Unchanged	5
26	F	39	Transient deficit	Carotid-ophthalmic	4	2	Anticipated difficulties	90	20	Transient monoparesis	5
27	F	37	Oculomotor nerve palsy	Carotid-ophthalmic	4.8	2.5	Surgical failure	70	64	Unchanged	5
28	F	55	Hydrocephalus	A1	25	8	Clinical judgement	0	0	Unchanged	4

[a] Initial Hunt and Hess grading
[b] Largest angiographic diameter
[c] See text
[d] Total length of coil delivered

Table 2 Ruptured aneurysms (13 in 11 patients). Initial Hunt and Hess grading and delay between haemorrhage and embolisation

Delay Hunt and Hess grade	1 week	1 month	1 year	More than 1 year
I	1	1		1
II			1	1
III		1	2	1
IV	1			
V	1			
Total	3	2	3	3

in diameter. There were 10 aneurysms on the posterior circulation (8 on the basilar and 2 on the anterior inferior cerebellar artery) and 16 aneurysms on the anterior circulation (10 carotid-ophthalmic, 3 anterior communicating artery, 2 carotid-cavernous, 1 A1 segment).

Multiple aneurysms were present in 9 patients; 6 of the 9 (67 %) had two aneurysms: 2 were treated during one endovascular session; 2 patients had been operated upon surgically for another aneurysm, one was operated upon after embolisation of one aneurysm, and one patient with two cavernous aneurysms was embolised only on the side of an oculomotor nerve palsy while the contralateral asymptomatic aneurysm was not treated. In the 3 other patients, who had multiple unruptured aneurysms, we chose to treat the largest lesion first.

The procedure lasted between 50 min (1 coil) and 4 h (9 coils), with a mean of 2 h 15 min. The mean number of coils delivered per aneurysm was 4; the total length of the coils is shown in Table 1.

The mean duration of hospitalisation was 6.5 days, including 2.8 days in intensive care (excluding one grade IV patient who spent 35 days in the intensive care unit).

The initial radiological results in the 26 treated aneurysms are shown in Table 3. We assessed the rate of occlusion as follows: complete angiographic exclusion of the sac was considered a 100 % occlusion (Fig. 2), a persistent orifice demonstrated on one or two projections was equivalent to a 90 % occlusion (Fig. 3), while partial opacification of the sac corresponds to a less than 90 % disconnection.

We tried to correlate the size of the lesion and the length of the coils necessary for occlusion (Fig. 4). The largest diameters of the aneurysms treated ranged from 4 to 30 mm (average 9.5 mm), while the length of coils delivered ranged from 15 to 320 cm (average 123.4 cm). We found a good correlation by calculation of the regression line parameters: linear regression constant term (A) −0.619; linear regression coefficient (B) 13.005: correlation coefficient (beta) 0.8345 ($P < 0.000001$).

At the 3rd and 6th months after the procedure, 12 follow-up angiographic studies were obtained (Tables 5,

Table 3 Karnovsky score in all patients

Score	100	90	80	70	60	50	40	30	20	10	00	Patients
Admission	5	7	6	4	1	2	1	–	1	1	–	28
Discharge	4	6	3	5	2	2	3	1	–	–	2	28
3 months	8	7	5	3	1	–	2[a]	–	–	–	2[b]	28

[a] One patient not embolised but operated upon and one with SAH grade IV embolised in the acute stage
[b] One patient not embolised and one died 5 days after embolisation

Table 4 Glasgow outcome score (GOS) on discharge

GOS	Treatment attempted	Treatment carried out
5 (dead)	2	1
4 (vegetative)	1	0
3 (severely disabled)	1	1
2 (moderately disabled)	3	2
1 (good recovery)	21	20
Patients	28	24

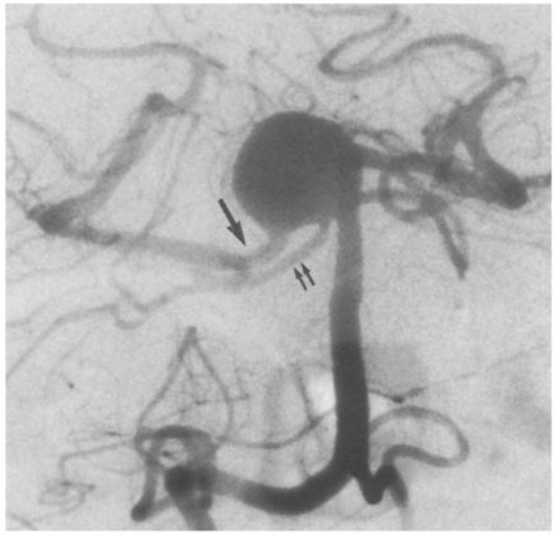

Fig. 1 Case 17. A contraindication to endovascular therapy with GDC. From this 8-mm-diameter aneurysm of the tip of the basilar artery, the P1 segment of the right posterior cerebral artery *(arrow)* and the right superior cerebellar artery *(small arrows)* arise directly

6). One partially occluded aneurysm (80 % initially) was entirely thrombosed (100 %), 8 aneurysms appeared stable and 3 were partially recanalised. In 2 of these 3 cases a basal portion of the sac was seen; in one case the recanalisation was obvious, since the 90 % initial rate of occlusion was only 50 % on follow-up. Compaction of the coils in the dependent distal part of the sac was observed. These three recanalised aneurysms were 9.4, 14 and 15 mm in diameter. Finding two cases incomplete treatment, we performed complementary embolisation using GDC, obtaining 90 % and 100 % occlusions. No aneurysm bled or rebled during follow-up regardless of their degree of occlusion.

We had three types of unfavourable outcome.

No patient died as a direct result of the procedure, although two patients succumbed for reasons unrelated directly to endovascular treatment: one patient, in grade V, not embolised because of failure of catheterisation died 2 days after attempted treatment. Another, in grade I, in whom an anterior communicating artery aneurysm was occluded by 90 % (Fig. 2) died after 4 days from an acute Gram-negative septicaemia; CT and angiography were normal.

We had two permanent complications. One patient with a 100 %-occluded right carotid-ophthalmic aneurysm had a right superior hemianopia on waking. Postoperative angiography demonstrated normal flow in the ophthalmic artery, and the ocular fundus showed a pale retina without emboli. T1-weighted MRI showed low signal in the optic nerve (Fig. 3). We attributed the unilateral visual impairment to pressure of the sac on the optic nerve. Despite steroid medication for 6 days no recovery occurred. Another patient, treated early in our experience, in whom a basilar tip aneurysm was occluded by 90 %, had a left hemiparesis on waking. CT showed multiple diffuse low-density areas in the posterior cranial fossa, which we thought due to multiple emboli due to inadequate saline flushing of the thin-walled 5F guiding catheter. There was partial recovery over the following months. Since that accident. 6F guiding catheters have been used routinely.

Transient problems occurred in 5 cases. One of our first patients (not given prophylactic heparin) with a 90 % excluded right carotid ophthalmic aneurysm developed a left hemiparesis 6 h after embolisation. Angiography revealed a filling defect at the orifice of the aneurysm, and amputation of several distal branches of the middle cerebral artery. Superselective fibrinolytic therapy, performed immediately with 900 000 IU urokinase was followed by complete recanalisation and total clinical recovery in a few hours.

Another patient (not on prophylactic aspirin) with a 70 %-occluded right carotid aneurysm developed a transient left monoparesis on the 5th day. CT was normal and angiography demonstrated a clot in the C3 portion of the internal carotid artery below the neck of

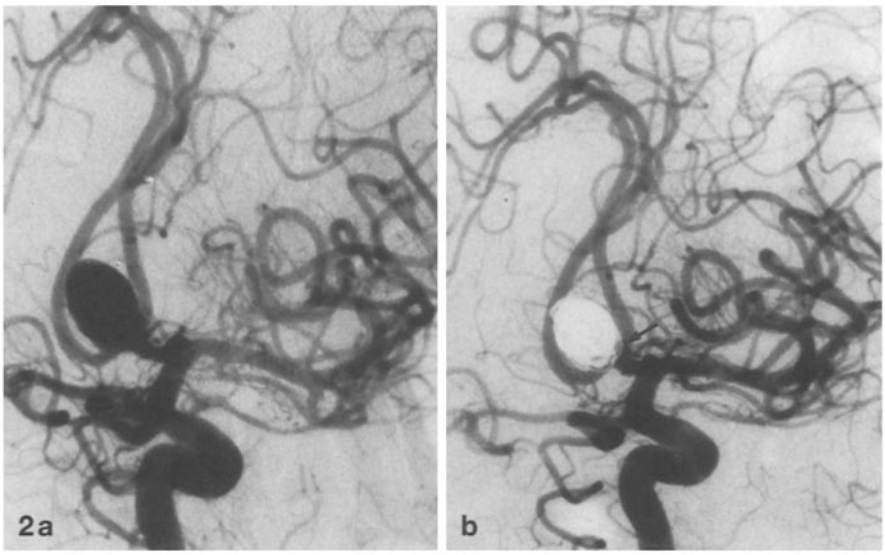

Fig. 2a, b Case 19. **a** Aneurysm of the anterior communicating artery. This oblique projection demonstrates that both the left and right anterior cerebral arteries are fed by the left A1 segment. **b** Persistent opacification of the orifice *(arrow)* indicates a 90 % exclusion

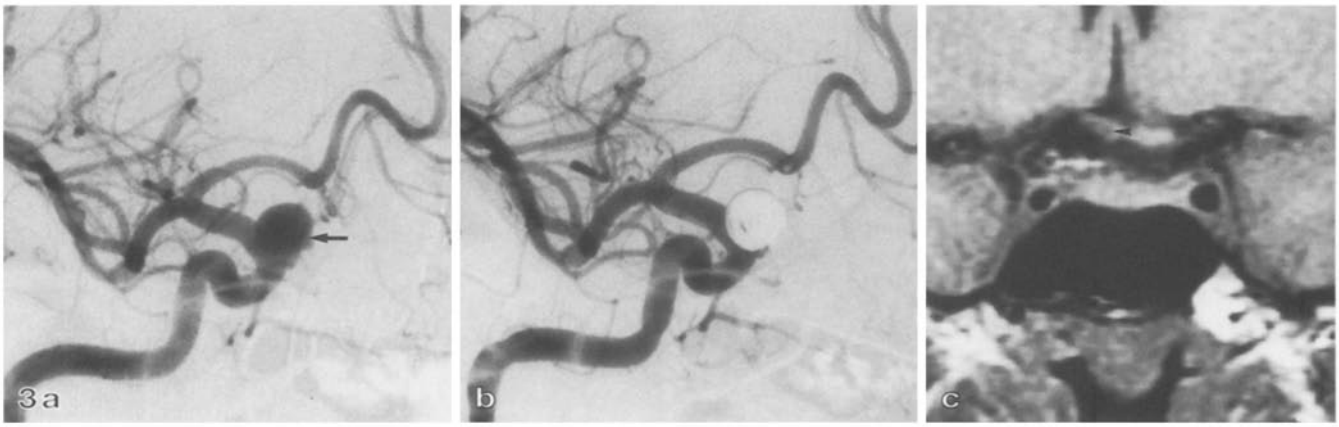

Fig. 3a–c Case 21. **a** Right carotid-ophthalmic aneurysm, oblique projection *(arrow)*. **b** 100 % occlusion: none of the aneurysm sac is filled by contrast medium. This patient had a superior hemianopia after the embolisation, thought to be due to compression of the optic nerve by the coil-filled sac. **c** Postoperative coronal MRI. Low signal in the inferior part of the right optic nerve *(arrowhead)* on this T1-weighted image was taken to indicate local trauma

the aneurysm. No treatment was given, as the patient had recovered spontaneously by the time of the angiogram.

The third patient had a right hemiparesis 1 month before admission. Angiography revealed three aneurysms and distal cortical branch irregularities suggesting diffuse arterial disease. The patient developed a right hemiparesis immediately after complete occlusion of a carotid-ophthalmic aneurysm. The angiogram showed occlusion of the precentral artery. Hyperselective delivery of 1 200 000 IU urokinase over a period of 90 min was radiographically ineffective, but the patient recovered completely over 6 weeks.

Finally, despite prophylactic anticoagulant therapy, one patient developed aphasia and another a monoparesis in the first 24 h. As early angiography was normal, no fibrinolytic therapy was applied. CT demonstrated focal ischaemic lesions, but complete clinical recovery occurred within a few days.

Discussion

The incidence of unruptured aneurysms in the population is around 1 %, according to the incidence of aneurysms seen on angiograms performed for nonaneurysmal disease [4]. The incidence of aneurysmal SAH (10/100 000 per year [5, 6] suggests a yearly risk of rupture of 1 % for asymptomatic aneurysms. Everyone attempting to treat incidental aneurysms should have in mind this low natural risk of rupture.

The aim of early surgical treatment of ruptured intracranial aneurysms is to avoid early rebleeding, which accounts for a cumulative 14-day risk of 19 % [7]; on the other hand, data on long-term follow-up after aneurysmal rupture show rebleeding rates of 50 % during the first 6 months and 3 % per year thereafter [8–10]. Surgery has been considered the main treatment for ruptured and unruptured aneurysms. Nevertheless, technical difficulties during surgery of posterior circulation [11] and carotid-ophthalmic aneurysms [7] lead neuroradiologists to find

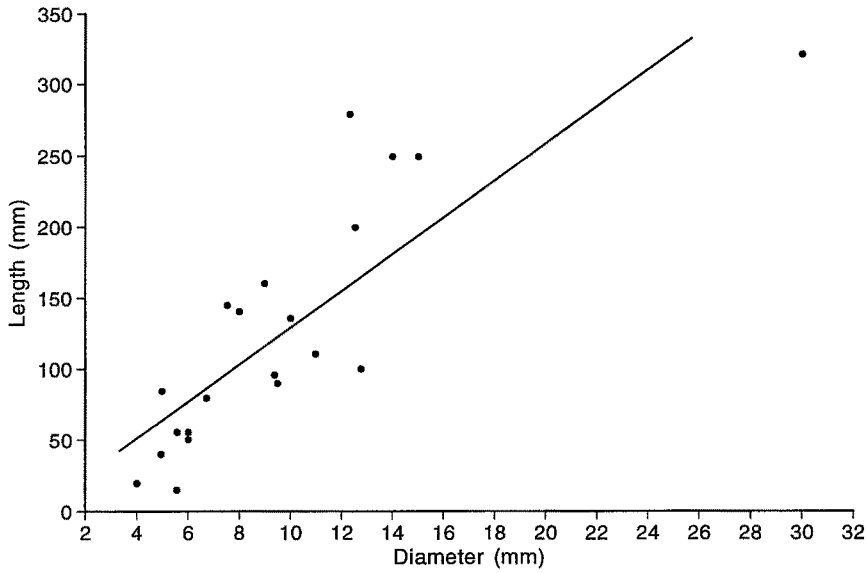

Fig. 4 Relation between the length of the coils and the diameter of the aneurysm in cases in which ⩾ 90 % occlusion was achieved

Table 5 Initial morphological results in 26 aneurysms treated

% Occlusion Neck type	100 %	90 %	80 %	70 %	Total
Large (A/N ≤ 2)	1	2	1	1[a]	5
Small (A/N > 2)	8	11	1	1	21
Total	9	13	2	2	26

[a] 14 mm diameter aneurysm; to be re-embolised

Table 6 Changes in morphological results in 4/26 aneurysms treated

Patient	% Occlusion			
	Initial	3 months	6 months	After second embolization
5	80	100	100	–
8	90	100[a]	80	90
10	80	70	–	100
12	90	50	–	–

[a] Follow-up angiogram performed elsewhere

alternative endovascular techniques, especially in these difficult locations.

Endovascular treatment, developed by Serbinenko in the early 1970s, consisted of occlusion of the parent vessel using detachable balloons through a cervical carotid approach. Later, Romondanov and Shcheglov [12], Debrun et al. [13], Berenstein et al. [14] and Higashida et al. [15] used, via a femoral approach, detachable latex or silicone balloons to occlude the aneurysm sac while preserving the parent artery. Inflation of the balloon with a polymerising agent conferred a permanent cast of the aneurysm. Nevertheless, the difficult technical management and unsatisfactory results led to a search for easier, safer endovascular therapy.

About 10 years ago Guglielmi et al. [1] conceived a technique of intraaneurysmal electrothrombosis, principle based on the fact that a positively charged electrode (anode) in citrated or heparinised blood will attract the negatively charged components (white and red cells, platelets and fibrinogen), thus promoting clot formation [9, 12]. The technology, experimental results and preliminary clinical experience have been described [2, 3].

Anatomical considerations

Particular attention was paid to vascular anatomy: good demonstration visualization and haemodynamic assessment of the basal arterial circle helped us to detect any hazardous situation and to identify the territories of the perforating arteries. According to Mercier et al. [16], the perforating arteries of the anterior and posterior circulation arise from different parts of the circle of Willis depending on the specific anatomical variations: for example, in the case of asymmetrical P1 segments, it seems that the posterior perforating arteries arise mainly from the P1 segment of cranial type to supply both sides. This may suggest specific technical choices during embolisation. In addition, the morphology of the sac is fundamental. Measurement on two orthogonal projections allowed us to assess its volume. The largest diameter of the sac (A), the size of the orifice of the aneurysm (N) and the calibre of the parent artery (PA) led us to choose a particular type of coil, by defining a theoretical stability rate of the coil (A/N) and a patency rate of the parent artery (PA/N) (Fig. 5). If A/N ≤ 2, the neck of

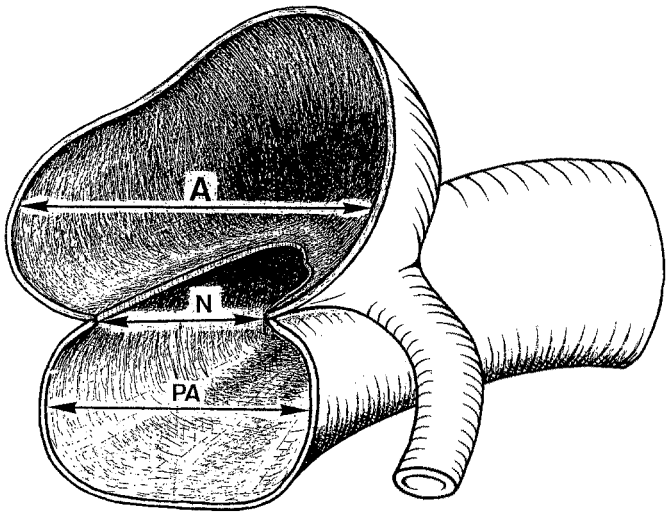

Fig. 5 Diagram of an aneurysm and its parent artery with the three measurements used to predict the likelihood of success (see text); (*A* largest diameter of the sac, *N* neck (orifice) of the aneurysm, *PA* calibre of parent artery)

the aneurysm is large and the stability of the coil uncertain. If PA/N < 2, patency of the parent artery may be compromised by coiling. Thanks to these measurements, primary or secondary occlusion of parent vessel has not occurred. In addition, three-dimensional MR angiography gave us images which helped us to assess these different measurements [17]; they can be useful in hazardous or difficult cases.

Technical considerations

We did not encounter manufacturing defects, rupture of the coil, or problems with the microsoldering between the coil and the mandril were never encountered. Our technical failures were due to anatomical peculiarities or errors in manipulation.

We have been unable to put more than 90 cm of coil in an aneurysm whose diameter is less than 7 mm whereas aneurysms whose largest diameter is more than 7 mm accommodate at least 90 cm of coil. Large aneurysms seem the most difficult to treat and, moreover those whose long-term occlusion is most uncertain.

Clinical assessment

Complications (5 transient and 2 permanent deficits in 24 patients) observed occurred immediately after the procedure in 3 cases, during the first 24 h in another 3 and in only one patient in a delayed fashion, on the 5th day. Since the complication in patient 9, prophylactic heparin has been instituted for 24 h after the procedure. Of the 30 patients embolised since then, 2 (7%) had post-therapeutic deficits, both of which regressed. One should make a distinction between therapeutic risks and complications proper. The former are an almost inevitable adverse effect (e.g., anosmia after a surgical subfrontal approach), to be balanced against the natural history of the disease prior to the intervention; the latter is an unexpected, technically related adverse effect which can be transient or permanent. Our patient who had a unilateral visual field defect following successful treatment of a carotid-ophthalmic aneurysm (postoperative Karnovsky score 80) and another who suffered peroperative distal embolism leading to a deficit from which he recovered only partially (postoperative Karnovsky score 60) illustrate respectively therapeutic risk and a complication of treatment.

The transitory problems in some patients could be related to red cells cylinder formation and emboli. We have not seen any migration of metallic fragments from the microsoldering during electrolytic detachment; such fragments could cause artefacts on MRI.

No vasospasm was observed in the 5 patients with ruptured aneurysms treated in the acute phase; it cannot, however, be affirmed that embolisation avoids vasospasm. This possibility should nevertheless be studied further in larger series.

The Glasgow outcome score at discharge (Table 4) indicates a good recovery in 83% of our patients (20/24), and the Karnovsky score at 3 months reveals the same ratio (20/24) leading a normal life (Table 3).

We have initially achieved satisfactory ($\geq$ 90%) occlusion in 85% of aneurysms (22/26) regardless of their size. No embolised aneurysm has rebled. The 3 patients with initial visual or ocular deficits did not improve while one with trigeminal pain and another with a facial palsy did recover. We therefore believe that embolisation stops the growth of the aneurysm sac, decreasing its pulsatility but possibly increasing its consistency. Delayed partial recanalisation was rare, occurring in 3 of 26 aneurysms treated and amenable to re-embolisation; it always occurs near the neck of the aneurysm. Partial initial occlusion of large aneurysms is most likely to lead to recanalisation. In one case (patient 5) we saw delayed total occlusion due to further thrombosis.

Cost: An average of four coils represents a price of FF 9500 (taxes included) and FF 13500 if one includes the price of the microcatheter and guidewire. In our institution the charge for one patient staying 6.5 days in the neurosurgical department, (including 2.8 days in intensive care) is assessed at FF 37300.

Addendum

During the second year, i.e. after this first group, 40 patients were referred for endovascular management of their aneurysm. Treatment was completed in 2 patients from the first group (nos. 10 and 26). Of the 38 new patients, 26 (69%) presented with subarachnoid haemorrhage (SAH), 2 (5%) with headaches, 4 (10%) with neurological deficits; 6 (16%) had aneurysms discovered incidentally. In 9 patients with multiple aneurysms the symptomatic one was treated first, the others being considered for coiling or surgery. Technical failures occurred in 4 patients (10%); two aneurysms could not retain the coils because of large necks, one with an old bleed is scheduled for another attempt, and one causing an acute SAH was operated upon directly after the failed procedure.

Of the 36 treated patients, 16 (45%) had their aneurysm totally occluded; 14 (39%) could be occluded to 90% and 6 (17%) to less than 90%. Glasgow outcome scale was 5 (good recovery; full independent life with minimal neurological deficit) in 33 treated patients (92%) and 3 (severely disabled; conscious but totally dependent upon others) in 3 (8%), the deficits in 2 of these being related to their disease (vasospasm and hemiplegia, initial Hunt and Hess grade IV) and not to embolisation itself. There has been no death during this 2nd year, and no rebleed.

Complications occurred in 4 cases (11%): in one patient, diplopia resolved spontaneously in 48 h after coiling of a large basilar tip aneurysm; a hemiplegia due to emboli into Sylvian vessels necessitated rapid fibrinolysis and recovered totally. Strokes occurred in 2 other patients: one has recovered totally; one remains aphasic despite early fibrinolysis (Glasgow outcome score 3). This brings our definitive complication rate in this second group to 3%, and in the series overall, since we started, to below 5%.

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