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ORIGINAL ARTICLE

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Recurrent hemorrhage after initially complete occlusion of intracranial aneurysms

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Abstract Recurrent hemorrhage in the case of incompletely treated aneurysms is well known. The authors present a series of patients in whom rebleeding occurred in spite of totally occluded aneurysms. During a period of 12 years, 1170 patients with intracranial aneurysms were treated using either clipping (n=727) or coiling (n=443). In 11 of them, intracranial rebleeding occurred, in seven of whom routine post-treatment angiography revealed total aneurysm occlusion before the appearance of rehemorrhage. Further analysis focused on these seven patients. Their recurrent aneurysm ruptures happened with a mean latency of 9.5 months (range 21 h–48 months) from initial treatment. All aneurysms belonged to the anterior circulation. Three patients underwent primary clipping, and four experienced coiling first. The intracranial hemorrhages appeared mainly as intracerebral hematomas. The angiographically documented recurrent aneurysm configurations were caused by clip slippage (n=2), coil compaction (n=3), or coil migration/dislocation (n=1). In one case with primary surgery, clip slippage was possible but not confirmed by intraoperative view, because the patient died before therapeutic intervention. Two patients did not undergo therapy because of their poor clinical condition and died. Four of the remaining patients underwent clipping of the recurrent lesions, and one had recoiling. Final outcome was excellent/good in only two patients. The mainly poor outcome after rebleeding was caused by the high incidence of intracerebral hemorrhage.

Keywords Clipping · Coil embolization · Intracranial aneurysm · Recurrent hemorrhage

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Introduction

Even laypersons would consider an incompletely treated intracranial aneurysm as a potential risk for future hemorrhage. In large microsurgical treatment series, postoperative angiography revealed incompletely occluded aneurysms in 1.6% to 14% of cases [6, 13, 18, 21]. The incidence of rerupture for postoperative remnants was as high as 10% [13]. The frequency of significant aneurysm rest after Guglielmi detachable coil (GDC) packing is approximately 14%, with an incidence of rebleeding for the residual aneurysm formations of up to 8% [3].

Recurrent hemorrhage in the case of completely occluded aneurysm is an extremely rare phenomenon and presupposes recurrent aneurysm formation. Whereas recanalization by coil compaction is a frequent long-term complication of endovascular treatment [3, 8, 11], delayed clip displacement from slippage or slackening is an unexpected failure in the use of late-generation clips. The authors present a series of patients in whom rebleeding occurred in spite of totally occluded aneurysms.

Patients and methods

From January 1991 to February 2003, 1170 patients with intracranial, mainly symptomatic aneurysms were treated at our institutions. As primary therapy, 727 underwent microsurgical clip ligation as primary therapy, and 443 underwent endovascular embolization with GDCs. Following microsurgery, postoperative angiography was routinely performed within 14 days, whereas after endovascular treatment, angiographically confirmed control was achieved at the end of the procedure, 3 and 12 months after coil placement.

In 11 of these patients, intracranial rebleeding occurred. In two of them, de-novo aneurysms independent from the previously treated aneurysms caused the rehemorrhages. In another two patients, rebleeding was caused by initially incompletely clipped aneurysms. These four patients were excluded from further analysis, because the study focused on rebleeding from previously totally occluded aneurysms documented by angiography.

Of the remaining seven patients, all had presented with subarachnoid hemorrhage (SAH) before the primary treatment. The location of the aneurysm included the anterior communicating

artery (ACoA) in three cases, middle cerebral artery (MCA) in two, and internal carotid/posterior communicating artery (PCoA) and pericallosal artery (PcallA) in one case each. Three patients underwent primary surgical clip application, whereas four experienced GDC packing first. Recurrent aneurysm rupture happened with a mean latency of 9.5 months (range 21 h–48 months) from initial treatment. The patients were followed up both angiographically and clinically.

Results

Surgery as primary treatment

In this subgroup (Table 1), all three patients had presented with extensive intracerebral hematoma after primary surgery, with poor Hunt and Hess grade (IV/V). The latency between initial treatment and rebleeding ranged near 48 months, 1 month, and 25 days, respectively. Angiography and intraoperative view both revealed clip slippage of 2–3 mm with recurrent aneurysm proximal to the clips in a giant MCA (patient 3, two clips, Sugita Elgiloy) (Mizuho, Japan) and a medium-sized ACoA aneurysm (patient 1, one clip, Yasargil Titanium alloy) (Aesculap, Germany). Both patients underwent emergency surgery with evacuation of the hematoma and additional clip application without removal of the former clips. The ACoA aneurysm was approached via right pterional craniotomy at the first operation. The second surgery was performed through a left pterional approach, because the ICH was located in the left frontal lobe (Fig. 1). In both patients, outcome was unfavorable (Glasgow Outcome Scale II/III). In the third patient (MCA aneurysm, patient 2), last angiography also revealed a recurrent aneurysm proximal to the clip (Sugita Elgiloy). Therefore, clip slippage could be a possible mechanism. However, this could not be confirmed by intraoperative view, because the patient died before therapeutic intervention. None of the three patients underwent magnetic resonance imaging (MRI) after primary surgery.

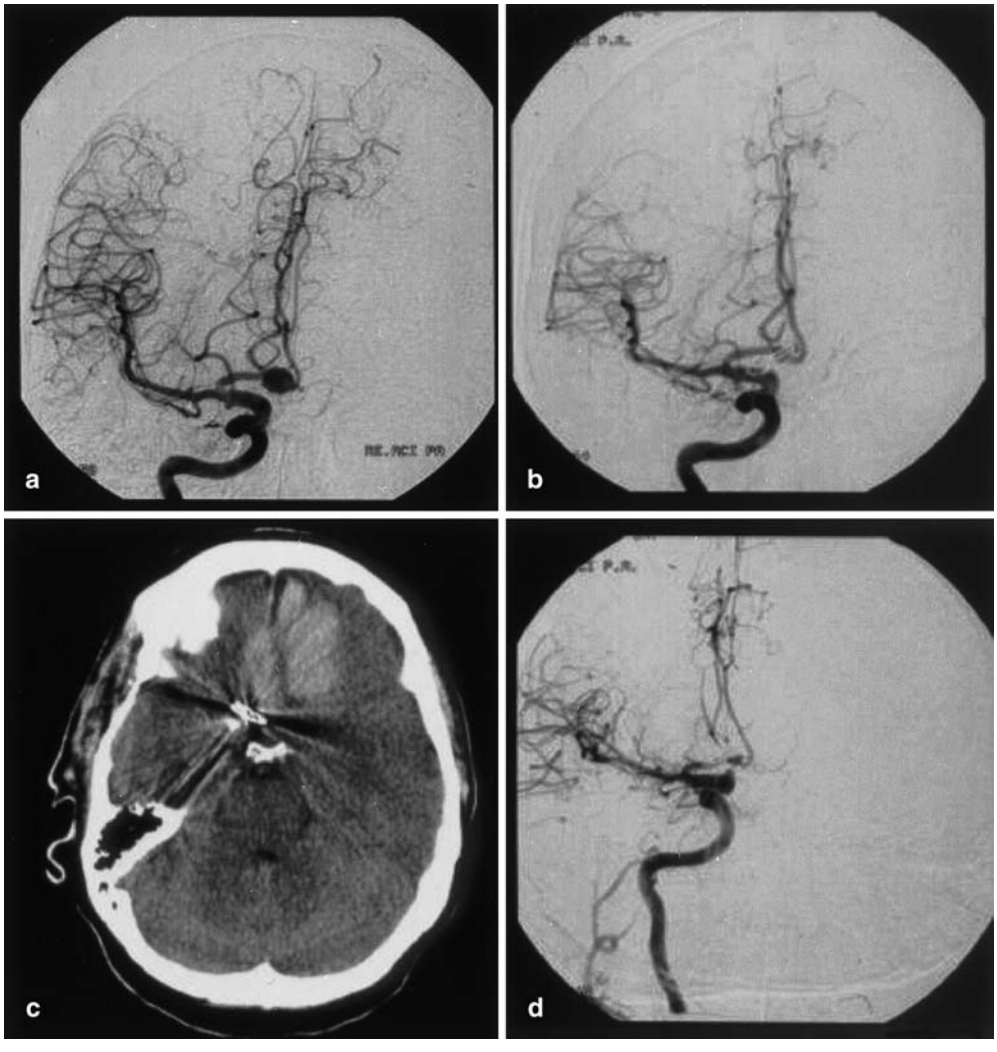

Fig. 1a–d Various imaging in patient 1. **a** Initial angiography revealing ruptured ACoA aneurysm. **b** Control angiography after initial clipping. **c** CCT visualizing rebleeding 1 month after clipping. Note the relationship between ICH and clip artefacts. **d** Angiography (same patient) revealing recurrent aneurysm and 'slipped clip'

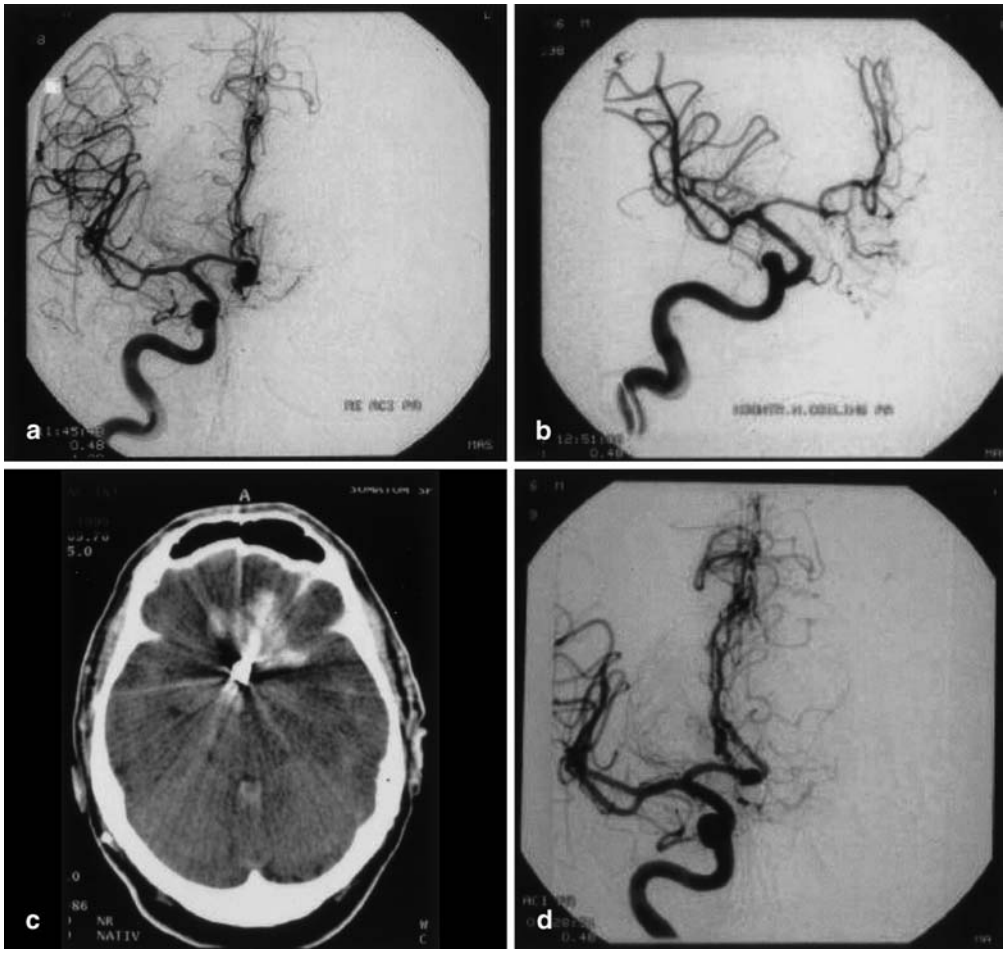

Fig. 2a–d Various imaging in patient 4. **a** Initial angiography revealing ruptured ACoA aneurysm. **b** Control angiography after initial GDC packing. **c** CCT visualizing rebleeding 6 months after coiling. Note the relationship between ICH and coil artefacts. **d** Angiography revealing coil compaction with recurrent aneurysm formation

Table 1 Clinical data of patients of our series (*GOS*Glasgow Outcome Scale)

Case	Age (years), sex	Aneurysm location	Initial treatment	Rerupture	Latency to rebleeding	Mechanism	Retreatment	GOS
1	66, F	ACoA	Clipping	ICH	1 month	Slipped clip	Reclipping	II
2	63, M	MCA	Clipping	ICH	48 months	Slipped clip/?	No	I
3	63, M	MCA	Clipping	ICH	25 days	Slipped clip	Reclipping	III
4	42, M	ACoA	Coiling	ICH	6 months	Compaction	Clipping	III
5	48, F	PcallA	Coiling	SAH	9 months	Compaction	Clipping	V
6	24, M	PCoA	Coiling	SAH	2 months	Compaction	Recoiling	V
7	65, M	ACoA	Coiling	ICH	21 h	Dislocation	No	I

Endovascular embolization as primary treatment

In this subgroup (Table 1), two patients developed ICH, and the other two SAH after initially successful GDC packing. The ICH cases presented poor clinical condition (Hunt and Hess grade V), whereas clinical examination of the SAH patients revealed good Hunt and Hess grades (I). The latency between initial GDC packing and rerupture ranged near 9 months, 6 months, 2 months, and 21 h, respectively. In three cases (ACoA, PCoA, PcallA), angiography revealed typical coil compaction as the cause of rebleeding. The PCoA aneurysm (patient 6) was successfully occluded by recoiling. The patient (patient 4) with ACoA aneurysm (Fig. 2) was operated on with ICH evacuation, coil removal, and clip ligation and remained in Glasgow Outcome Scale (GOS) III status. The PcallA aneurysm (patient 5) was completely occluded by surgery (coil removal/clipping). Both patients who suffered from SAH as rebleeding (PCoA, PcallA) remained in excellent clinical condition (GOS V). The last patient of this subgroup (patient 7) suffered from SAH Hunt and Hess grade IV condition caused by a large aneurysm of the ACoA. This patient was treated by endovascular embolization (GDC), and total occlusion was achieved, as was well documented by the last intraprocedural angiography. Because of repeated obliteration of the external ventricular drainage (EVD) and extensive intraventricular blood clots, 5 mg of recombinant tissue-plasminogen activator

Table 2 Clinical data of cases reported in the literature (*GOS* Glasgow Outcome Scale)

First author, year	Aneurysm location	Initial treatment	Rerupture	Latency to rebleeding	Mechanism	Retreatment	GOS
Kossowsky 1982	PCoA	Clipping	ICH	22 months	Clip fracture	No	I
Drake 1984	PCoA	Clipping	?	5 months	Slipped clip	Reclipping	?
	BA	Clipping	?	7 years	Slipped clip	Vessel occlusion	I
Klucznik 1993	MCA	Clipping	ICH	14 years	Magnetic field	No	I
Hodgson 1998	MCA	Coiling	SAH	18 months	Compaction	Clipping	III
Byrne 1999	PcallA	Coiling	SAH	18 months	Compaction	No	I
	BA	Coiling	SAH	30 months	Compaction	Recoiling	IV
	PCoA	Coiling	SAH	35 months	Compaction	Clipping	V
Steiger 1999	BA	Coiling	SAH	5 years	Compaction	Recoiling	?
Bavinzski 1999	BA	Coiling	SAH	2 days	Minimal compaction	No	V

(rt-PA) was given over the EVD the next day. Two hours later, the patient developed massive hemorrhage into the right frontal lobe. Besides, the definitive CCT scan revealed dislocation of a GDC into the subarachnoid space. Finally, this patient died without additive therapy (symptoms of advanced cerebral herniation).

Discussion

Surgery as primary treatment

Since it is apparent that neither the surgeon's intraoperative impression nor collapsing of the dome at surgery is adequate to ensure complete aneurysm occlusion by clip ligation, postoperative angiography is necessary to document the sufficiency of the operative procedure [13, 19]. It is widely felt that a completely clipped aneurysm will not recur. However, little information is available regarding the late outcome of clipped aneurysms. Formerly, use of the problematic Heifetz clip played an important role in delayed aneurysm recurrence. A total of 12 known cases of Heifetz clip fractures from stress corrosion is documented in the literature [12]. Of these, one patient developed rebleeding (Table 2). Drake et al. recognized two patients (Table 2) with rebleeding from recurrent aneurysms due to slipped clips 5 months and 7 years after initial control angiography [5]. In a series of 160 microsurgically treated aneurysms, additional late follow-up angiography was obtained at a mean of 4.4 years [4]. The investigation revealed two patients (1.25%) with true aneurysm recurrence without rebleeding. In the international subarachnoid aneurysm trial of neurosurgical clipping vs endovascular coiling in 2143 patients (ISAT), the incidence of rebleeding from a primarily completely clipped aneurysm was 0.5% [9]. The ISAT did not present detailed information in relation to this form of complication.

In our series, the incidence of rerupture from initially completely clipped aneurysms was 0.4%. However, the real incidence could be higher, because only the symptomatic recurrences were known. Clip slippage was suspected in two of our cases. The latency between initial surgery and rebleeding was 1 month and 25 days, respectively. Latency between initial control angiography and recurrent hemorrhage was even shorter. During this short time, development of de novo aneurysms from a "dog ear" residual, which was not apparent on initial angiography, was unlikely.

In one case, the Yasargil clip was perfectly positioned on the aneurysm neck. The blades embraced the neck with the proximal two thirds of their length. The closing force of the used Yasargil clip was 180 g (1.77 N) according to the manufacturer's declaration (ISO 9713). This clip had possibly experienced repetitive application trials in former operations and lost some strength. In the second case of a giant MCA aneurysm, the closing force of the two Sugita clips used was 150–185 g (1.47–1.81 N), each according to the manufacturer's statement (ISO 9713). In this case, two mechanisms could be responsible for the slippage. Clip ligation was possible after temporary occlusion of the proximal MCA segment. The wide and partially calcified neck exerted stress on the applied clips. Besides, the clips grasped the neck with the distal third of their blades. As the closing force dramatically decreases from heel to the tip of the clip blades [23], the clip positioning was unfavorable in this case. In our third patient, clip slippage and de novo aneurysm formation from a minimal residual neck not seen at initial control angiography both were possible causes of recurrent aneurysm with rebleeding.

On principle, MRI investigation on a patient with clip-ligated intracranial aneurysm could cause clip movement and rehemorrhage in spite of initially complete aneurysm occlusion (Table 2) [10]. In vitro studies revealed that the Sugita Elgiloy clips used in our patients showed a deflection angle of 9° in a 3.0 Tesla MR system, whereas the Yasargil titanium clip did not show any movement [20]. In our series, we can exclude this mechanism, since none of our patients underwent MRI study between primary surgery and rerupture.

In all cases, rerupture resulted in ICH. We explain this phenomenon by pre-existing dense arachnoid scarring of the subarachnoid space due to the primary SAH and operation. Doubtless, reoperation was the treatment of choice in our patients because of the space-occupying

ICH. Additionally, the recurrent aneurysm formations frequently were located proximal to the previously applied clips, and the "neck:dome" ratios were unfavorable for complementary endovascular management [1].

Endovascular embolization as primary treatment

The frequency of significant aneurysm after GDC packing is approximately 14% [3, 24]. The rebleeding rate up to 36 months after endovascular embolization in 403 patients due to initially incomplete aneurysm occlusion with consecutive coil compaction was reported to be 2.2% [24]. Some case reports with occurrence of coil compaction and rebleeding indicated the risk of initially incomplete coil embolization [15, 16]. However, even in recent reports the rate of coil compaction was reported to be between 14% and 28%, depending on several factors [3, 8, 11]. Initially complete coil embolization could have resulted in coil compaction even 18–24 months later. In our own experience, the rate of compaction was nearly 10% [1]. So-called inflow aneurysms such as lesions of the BA and paraclinoid internal carotid artery are at a much higher risk of compaction [14]. Lack of endothelialization of the luminal surface at the neck of the aneurysm could play a role in the mechanism of coil compaction [15, 17].

In the literature, six cases with initially complete coil embolization and true rebleeding (SAH) are known (Table 2). In our series, the incidence of rerupture from an initially completely GDC-packed aneurysm was 0.9% and comparable to the data of Byrne et al. [3]. The ISAT report indicated an incidence of rebleeding for completely coiled aneurysms of 0.7% [9]. As in three of our four patients, typical coil compaction was the mechanism of rebleeding in five of six reported cases 18 months–5 years after primary treatment [3, 7, 22]. In our fourth case, the rebleeding mechanism remained unclear, since the patient's family refused autopsy. During the period of postembolization heparinization, 5 mg of rt-PA was given over the EVD. Two hours later, the fatal rehemorrhage occurred, and CCT revealed a coil dislocated into the subarachnoid space. Possibly, acute coil compaction with aneurysm perforation by the coil under systemic anticoagulation and intrathecal fibrinolysis caused this catastrophe. Bavinzski et al. described a case (Table 2) of basilar artery (BA) aneurysm completely embolized by a coil with rebleeding (SAH) on the second postprocedural day during routinely performed systemic heparinization [2]. Immediate control angiography revealed minimal recurrent neck filling implicating minimal coil compaction in this wide-necked aneurysm. Twelve months later, control angiography demonstrated complete aneurysm occlusion without any sign of compaction. This case proves that a wide aneurysm neck and anticoagulation and/or fibrinolysis can lead to coil movement in spite of complete aneurysm obliteration during the first 2 days after the procedure.

Principally, repeated GDC packing is the treatment of choice in cases of coil compaction [1, 3], as done in one of our patients (PCoA) and in two reported in the literature (Table 2). Of course, our patient (ACoA) with space-occupying ICH underwent surgery. Additionally, the patient with the PcallA aneurysm underwent clip ligation, because angiography revealed extensive protrusion of the compacted coils through the aneurysmal wall. However, the intraoperative view confirmed that two thirds of the coil material had penetrated the aneurysmal wall.

Conclusion

Recurrent aneurysm formation with rebleeding after initially complete occlusion is a rare but possible complication of both surgical and neuroradiological applications. The incidence is twice as high after GDC packing as after clip ligation. Whereas coil compaction as the main mechanism in rerupture after endovascular aneurysm treatment still is frequent, clip failure with resulting slippage is extremely rare. The mainly poor outcome after rebleeding was caused by the high incidence of ICH.

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