Recanalisation of cerebral artery aneurysms treated endovascularly — a midterm follow-up

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ABSTRACT

Endovascular methods of aneurysm treatment, as an alternative to neurosurgical clipping, have proved a welcome opportunity to treat patients with unruptured aneurysms or those disqualified from neurosurgical intervention. This paper presents our own experience of endovascular treatment of cerebral aneurysms in 107 patients. It includes clinical and technical data from the perioperative period and a 12-month radiological follow-up of 78 patients.

Method. Our retrospective evaluation covered patients with intracranial aneurysms treated endovascularly. The following were analysed: age, sex, neurological symptoms, and familial burden of intracranial aneurysm. Multivariate analysis was performed to determine independent factors of recanalisation of the cerebral aneurysm 12 months after embolisation.

Results. The data of 107 patients at a mean age of 61 years [57.09 ± 14.27] treated with embolisation was analysed. The indication for intervention in 16 patients was subarachnoid haemorrhage; in the remaining 91 cases, aneurysms were revealed during diagnostic procedures for different symptoms or during imaging examinations. The intracranial segment of the internal carotid artery and the anterior communicating artery were the most common locations for aneurysms. After embolisation, subarachnoid haemorrhage occurred in one patient, ischaemic stroke in two patients, and one patient died because of acute circulatory insufficiency. The functional status of 94 patients on the day of discharge from the department (on days 4-21) was very good. 78 patients completed a 12-month follow-up period. In 11 of those, a follow-up MR angiography revealed recanalisation 12 months after the intervention. Except for one patient reporting vertigo, aneurysm recanalisation procedures were asymptomatic. The only independent risk factor for recanalisation was the size of aneurysm > 10 mm; OR 3.0; CI [1.15–7.83] p = 0.0255.

Conclusions. Embolisation of cerebral aneurysms is a safe method with few perioperative complications, and most of these are mild and transient. The size of the aneurysm during qualification for embolisation is a risk factor for recanalisation in the subsequent 12 months. Recanalisation of embolised cerebral aneurysms concerns less than 20% of patients in a one-year follow-up and is most often asymptomatic.

Key words: cerebral aneurysm, embolisation, recanalisation

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Background

Intracranial aneurysms, assessed on the basis of post mortem examinations, are estimated to affect 1–5% of the adult population [1]. They are reported three times more frequently in women than in men [2]. Most cerebral aneurysms do not rupture. Improved access to neuroimaging examinations has enabled physicians to find more intracranial vascular abnormalities prior to the appearance of neurological symptoms. Endovascular methods of aneurysm treatment, as an alternative to neurosurgical clipping, have proved a welcome opportunity for patients with unruptured aneurysms and have helped those disqualified from neurosurgical intervention. Compared to surgical clipping of aneurysms, endovascular therapy is substantially less invasive [3]. The risk of aneurysm rupture depends on the patient's clinical profile (i.e. age, location, familial burden of aneurysms) and the morphological features of that aneurysm (size and shape) [3]. Before deciding about the indication for a specific treatment and before the choice of method, the risk of natural course (i.e. will the aneurysm rupture if no intervention is performed) is compared against the risk related to the particular neurosurgical or endovascular procedure.

There are these days many different techniques used in endovascular treatment of cerebral aneurysms, e.g. simple coiling, balloon remodelling, stent remodelling, 2-stent remodelling in various configurations, flow diversion, and intrasaccular flow disruption, as well as combinations of the abovementioned methods.

The choice of a specific treatment technique depends on a number of factors, i.e.: the morphology of the aneurysm and in particular the width of the aneurysm neck, the preference of the operator, the availability of equipment in the laboratory, and the anatomy of the arteries feeding the aneurysm [4]. Aneurysm recanalisation after endovascular treatment affects up to one third of patients, usually within the 12 months following intervention. The risk of recanalisation depends on the parameters related to the morphology of the aneurysm and the technical aspects of the procedure.

This study looks at the effectiveness and safety of embolisation of ruptured and unruptured cerebral artery aneurysms.

Method

This paper presents our own experience with endovascular treatment of mostly unruptured cerebral aneurysms in 107 patients exhibiting clinical and technical data from the perioperative period and at least a 12-month radiological follow-up of 78 patients (72.89% of the 107).

Retrospective evaluation involved 108 patients who, between January 2017 and December 2019, were hospitalised in the Neurology Department of the Upper-Silesian Medical Centre of the Silesian Medical University in Katowice, Poland for embolisation of ruptured or unruptured intracranial aneurysms with the use of coils and/or stents. Preliminary diagnosis of an aneurysm was established on the basis of head computed tomography (CT) or magnetic resonance (MR) angiography (CTA, MRA).

The following data was analysed: the age and the sex of the patient qualified for embolisation, their neurological symptoms, any other diseases, and any family history of intracranial aneurysm. Moreover, the parameters related to aneurysm, i.e. size, location, rupture and individual history, were analysed for the presence of other aneurysms and previous endovascular procedures. For the description of parametric data, the mean and standard deviation, median, minimum and maximum values of a given variable in the study group were calculated. Multivariate analysis was performed to determine independent factors of recanalisation (based on MR angiography) of the cerebral aneurysm 12 months after embolisation. The following parameters were analysed: age, sex, arterial hypertension, diabetes, dyslipidemia and the size of the aneurysm (i.e. < 10 mm, 10–24 mm, or > 24 mm).

Description of cerebral aneurysm embolisation

All patients undergoing diagnostic procedures had a digital subtraction angiography (DSA) performed using X-ray (Siemens Axiom Artis dTa) as per the defined protocols. The other part of the examination involved the use of a DSA workstation for further image analysis. Aneurysms were analysed at the DSA station based on the imaging results. The following features were assessed:

— location;
— aneurysm sac dimensions – the longest dimension of length, height, and width;
— aneurysm neck dimension – the diameter measured in two extreme dimensions;
— sac-to-neck ratio – the longest sac dimension and the longest neck dimension were used for calculations; these were divided into three groups: ratio < 1.2; between 1.2 and 1.5; and >1.5 [5];
— aneurysm size – the aneurysms were divided by size: small aneurysm < 10 mm in diameter; large aneurysm 10–24 mm; or giant aneurysm > 25 mm [6, 7]. The assessment of the aneurysm sac is presented in Figure 1.

Depending on the sac-to-neck ratio, the aneurysms were treated using the following techniques:
a) simple coiling;
b) balloon remodelling;
c) stent remodelling using:
  • a single stent,
  • or two stents in Y, X or T configurations;
d) flow diversion.

The coils were implanted into the aneurysm sac using a micro-catheter and they were detached while using fluoroscopic viewing. The coils were introduced into the aneurysm sac until the aneurysm was filled up and no more coils could be inserted, or until no further inflow of the contrast agent into the aneurysm sac could be seen in angiography.

Figure 1A. Left internal carotid artery aneurysm in the ophthalmic segment (C6) — the sac dimension (length and height)

Figure 1B. Left internal carotid artery aneurysm in the ophthalmic segment (C6) — the sac dimension (width)

Stent embolisation was mainly applied in patients with wide-neck aneurysms. The most commonly used was the hybrid stent (Atlas by Stryker) and the second most commonly used was the braided stent (Leo, Leo Baby by Balt). Flow diverters (Silk by Balt) were used in small quantities and only if no other endovascular technique was suitable for the given aneurysm. A stent was implanted to cover the aneurysm neck; if that aneurysm was located at an artery branching, we chose the branch which enabled us to cover as much of the aneurysm neck as possible. This technique often requires catheterisation of the M2, A2 branches that start at an unfavourable angle of the aneurysm neck. In such cases, a combination of pre-shaped (S, J, C) Sl10 microcatheters (Stryker) with a Hybrid 008J micro guidewire (Balt) was successfully applied. Due to the unique

properties of a hybrid stent, a 2-stent Y or T-configuration placement was very rarely necessary.

Those patients who were planned for stent implantation received pretreatment with antiplatelet agents. On the day before the procedure, the patient was given 600 mg of clopidogrel and 300 mg of aspirin. Patients who underwent stent implantation into the cerebral arteries were administered 75 mg of clopidogrel for three months and 150 mg of aspirin daily for six months following stent placement. Follow-up head MR angiography was scheduled for 12 months post intervention. Residual flow was classified as: A) total obliteration; B) contrast-filled neck without sac-filling; or C) residual aneurysm with sac-filling.

Results

There were 108 patients with a mean age of 61 years [57.09 ± 14.27] qualified for embolisation. In one patient with an unruptured aneurysm, intervention was abandoned for technical reasons (after angiography), meaning that eventually data from 107 patients was collected and analysed. Half of the patients were aged 50–70 and nearly 80% of the whole group were female. The indication for intervention in 16 patients (15%) was subarachnoid haemorrhage in the course of aneurysm rupture; in the remaining cases, aneurysms were revealed during diagnostic procedures for headaches (most commonly) or during imaging examinations unrelated to neurological symptoms. The intracranial segment of the internal carotid artery and the anterior communicating artery were the most common locations for aneurysms. Nearly one in four of the patients revealed another intracranial aneurysm; one in 10 patients had previously undergone endovascular therapy due to an aneurysm in another location. The great majority of patients (73%) underwent small aneurysm embolisation (small being defined where the largest aneurysm was < 10 mm).

Clinical and radiological description of the patients who underwent embolisation is presented in Table 1.

In 51 patients (53.76%), at least six coils were implanted, and stents were placed in 45 patients (42.05%). The most common symptoms reported by the patients in the 72 hours following intervention were: diffuse headache, vertigo, and visual disturbances. Subarachnoid haemorrhage occurred in one patient. Ischaemic stroke occurred in two patients. One patient with a ruptured ACoA aneurysm died due to acute circulatory insufficiency.

The functional status of nearly 90% of patients on the day of discharge from the department (on days 4–21) was very good. Those patients who required caregiver assistance were previously qualified for embolisation due to a ruptured cerebral aneurysm. Table 2 presents the parameters in the perioperative period.

Seventy-eight (72.89%) patients completed a 12-month follow-up period. In 11 of those (14.10%), a follow-up MR angiography revealed recanalisation after 12 months. The degree

Table 1. Characteristics of patients undergoing cerebral aneurysm embolisation

Parameter	N (%)
Mean age [range]	61 [25–82]
< 30	5 (4.67)
30–49	29 (27.1)
50–69	55 (51.4)
≥ 70	18 (16.51)
F	83 (77.57)
Arterial hypertension	61 (57)
Diabetes	8 (7.48)
Dyslipidemia	30 (28.03)
Neurological signs before qualification for embolisation:	
Headaches	46 (58.97)
Vertigo	31 (39.74)
Visual disturbances	6 (7.68)
Tinnitus	20 (25.64)
Seizures	3 (3.84)
No complaints	23 (29.48)
Cerebral aneurysm in a family member	2 (2.56)
Aneurysm location:	
ACoA	17 (15.89)
MCA	14 (13.08)
BA	10 (9.35)
ICA intracranially	53 (49.53)
ACA	4 (3.74)
VA	3 (2.8)
Lateralisation (without ACoA)	
L	39 (50.65)
Ruptured aneurysm	16 (14.95)
Hunt and Hess Scale score	3
I	5
II	5
III	3
IV	
Presence of other cerebral aneurysms	24 (22.43)
Previous procedure for (another) cerebral aneurysm	12 (11.21)

F — female; ACoA — anterior communicating artery; MCA — middle cerebral artery; BA — basilar artery; ICA — internal carotid artery; ACA — anterior cerebral artery; VA — vertebral artery

Table 2. Parameters related to cerebral aneurysm embolisation in 78 patients undergoing a follow-up of at least 12 months

Parameter	N (%)
Aneurysm size*	
< 10 mm	78 (72.89)
10–24 mm	28 (26.16)
> 24 mm	1 (0.94)
Number of coils**	
< 6	43 (45.74)
≥ 6	51 (54.26)
Stent implantation	45 (42.05)
Antiplatelet therapy:	
ASA monotherapy	34 (31.77)
CLP monotherapy	1 (0.93%)
Duotherapy	53 (49.53)
Complications / clinical status of patients, 1–3 days after intervention:	
Haematoma at puncture site	5 (4.67)
Headaches	11 (10.28)
Vertigo / visual disturbances	3 (2.8)
Ischaemic stroke	2 (1.87)
Death	1 (0.93)
SAH	1 (0.93)
Heart failure	3 (2.8)
Functional status on day of discharge on mRS:	
0–2	94 (87.85)
3–5	12 (11.21)
Death during hospitalisation	1 (0.93)

ASA — acetylsalicylic acid; CLP — clopidogrel; SAH — subarachnoid haemorrhage; mRS — modified Rankin Scale; *based on head MR angiography; **data from 94 procedures

The only independent risk factor for recanalisation was the size of aneurysm > 10 mm (Tab. 3).

The internal carotid artery aneurysm before and after embolisation is presented in Figure 2.

Discussion

The most important conclusion that we draw from our study is that cerebral aneurysm embolisation is a safe method. Recanalisation affects 14% of patients 12 months following the intervention. The great majority of patients were asymptomatic and did not require urgent management. Instead, radiological follow-up was indicated.

Anterior intracranial vascular supply proved to be the most common location of aneurysms qualified for endovascular therapy. This accords with the findings of epidemiological studies related to the location of cerebral aneurysms. In our group of patients, diseases causing angiopathy (arterial

of recanalisation in 10 patients was defined as grade B, and in one patient as grade C. This grade C patient required management during reintervention. For the remaining patients, a check-up using MR angiography at six months was recommended. Except for one patient reporting vertigo (probably of multifactorial aetiology), the patients requiring a second procedure due to aneurysm recanalisation were asymptomatic.

Table 3. Risk of recanalisation of aneurysm 12 months after embolisation*

	p (factor)	RR
Size of aneurysm	0.0255	3.0
> 10 mm		95% CI [1.15–7.83]

*The following co-variables were analysed: age, sex, diabetes, arterial hypertension, dyslipidemia, and size of aneurysm ≤ 10 mm or > 10 mm
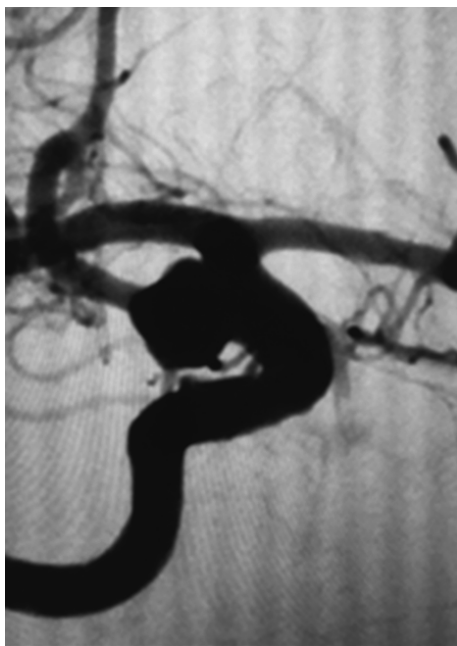
Figure 2A. Right internal carotid artery aneurysm with a wide neck before embolisation
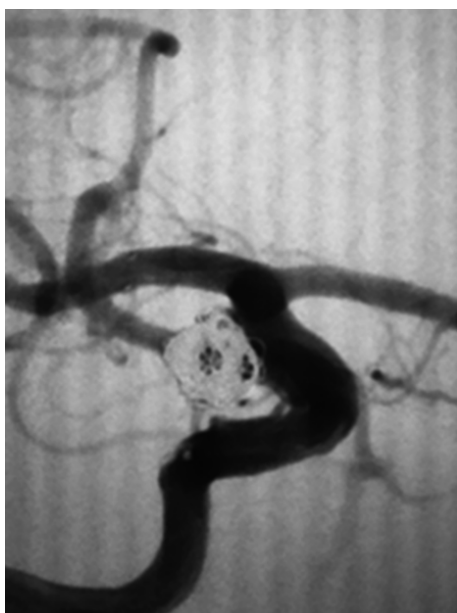
Figure 2B. Right internal carotid artery aneurysm with a wide neck after embolisation with stent securing the aneurysm neck and coils

hypertension, dyslipidemia) were common in 60% and 28%, respectively. Women made up the majority of embolised patients, which is consistent with the gender distribution among patients with cerebral aneurysms [2].

The most common symptoms reported by patients within the first few days following the intervention were headaches with vertigo and atypical visual disturbances. Considering the type and transient nature of the complaints, these probably resulted from vasoconstriction. Two patients suffered ischaemic stroke in the peri-interventional period; one had a subarachnoid haemorrhage. Inguinal haematoma was found in 5% of patients and this was the most frequent extra-neurological complication of the procedure. One patient died during hospitalisation due to acute circulatory insufficiency. The functional status of the great majority of patients on the day of discharge was very good. According to the literature, the most common complications after embolisation of cerebral aneurysms are haematoma in the groin, false aneurysm, and infection. Less frequently reported are arterial dissection (0.7%), embolism (2.5%), and arterial occlusion (2%) [8]. According to observations reported by other authors, the incidence of ischaemic stroke in the perioperative period is higher than that of intracranial haemorrhage: 4.81% vs 1.87%, respectively (National Inpatient Sample Database) [9]. The mechanism of ischaemia in this group of patients may have multiple origins. Hypercoagulability status plays a significant role, along with the following: inappropriate anticoagulation before and during intervention, unstable atherosclerotic plaque, and coil extension into the proximal artery [10, 11]. The risk of thrombosis increases if the stent does not appose well to the vessel wall and/or two stents are used (especially in the Y or X configurations). Endothelial damage during the procedure may stimulate thrombus formation. Cerebral stroke is more likely to occur when managing large aneurysms. The size of the thrombus is a risk factor for its propagation and/or distal embolisation. The above-mentioned complications are reported more often in relation to previously clipped aneurysms than to coiled aneurysms [9, 12].

In accordance with the results of meta-analysis of data from 7,172 patients, poor functional condition/death is observed in 4.7% of patients undergoing embolisation [13]. A significantly higher percentage of patients with a good therapeutic outcome was found among those treated after 2004 (the analysis related to the period 2000 to 2011). A higher risk of poor functional status was demonstrated after treatment with liquid embolic agents (8.1%; 99% CI: 4.7%, 13.7%) than after coil placement (4.9%; 99% CI: 3.8%, 6.3%; p = 0.002) and the highest risk was demonstrated after treatment with flow diversion (11.5%; 99% CI: 4.9%, 24.6%) [14].

In the BRAT study, embolisation was associated with a higher percentage of patients in good functional status than in those after microsurgical clip occlusion in a 1.3 and 6-year observation, particularly in the analysis of the effects of

aneurysm treatment in the posterior part of cerebral circulation [14–16]. However, analysis of the results 10 years post intervention did not reveal any significant differences in this respect [17, 18]. The results clearly proved significantly more frequent recanalisation after endovascular therapy: 16.4% *vs* 4.6% (p < 0.0001) after a 6-year follow-up [16].

The incidence of recanalisation after embolisation of cerebral aneurysms varies between 5% and 34% [19]. The risk of recanalisation of an aneurysm depends on the parameters related to its morphology (shape/size) and the technique applied for the procedure [19, 20]. This risk is higher when the aneurysm sac size is > 10 mm and/or neck size is > 4 mm [5]. In our study, the size of the aneurysm was the only independent risk factor for recanalisation. The results of one study showed that an aneurysm sac > 10 mm multiplies the risk of recanalisation x 4.5 times [21]. It is probable that a larger aneurysm volume makes it more difficult to stabilise and increase the density of coil placement, thus making it easier for coils to slide out of their original positions. According to many authors, coil filling density of 20-25% protects against recanalisation [6, 8]. In our presented paper, 5/11 patients after recanalisation had an aneurysm sized 10–24 mm; seven of them had at least six coils implanted. According to the published data, recanalisation is observed in 35% of patients with 11–25 mm aneurysms, and in as many as 59% of patients with giant aneurysms (> 25 mm) after embolisation [8].

One of our patients had a giant aneurysm, and in that case recanalisation also occurred.

Evaluation of haemodynamic parameters in the aneurysm sac (i.e. flow velocity and wall shear stress) indicates a significantly lower amplitude reduction of the velocity on the neck plane in patients undergoing recanalisation [22, 23]. The role of inflammation in the recanalisation process is not yet established, which is probably important bearing in mind other factors in the process of formation, progression and rupture of aneurysms [24, 25].

According to some authors, the differences in the incidence of recanalisation result from the interval between the intervention and follow-up examination. In most study sites, it is recommended that patients undergo a follow-up every six months (usually involving a head MR angiography). Given the low probability of delayed recanalisation, careful monitoring is still warranted, although at less frequent intervals (every 2–3 years) beyond 36 months post embolisation [26]. Some observations have suggested that the radiological condition reported six months after embolisation remains stable for at least two years [27].

According to studies carried out by Canadian authors, most recanalised aneurysms are visualised in MR angiography within a follow-up period of 12.31 months [28].

Recanalisation is most often asymptomatic. In this case, a routine radiological check-up allows for the assessment of the long-term effects of the intervention.

Limitations

We are aware of the limitations of our study, the most significant being its retrospective nature. No doubt the use of DSA angiography to assess the degree of recanalisation would be a more valuable procedure. However, due to the invasiveness of the examination and its limited availability, we decided to adopt head MR angiography instead.

Conclusions

Embolisation of cerebral aneurysms is a safe method with few perioperative complications, most of them being mild and transient.

The size of the aneurysm during qualification for embolisation is a risk factor for recanalisation in the subsequent 12 months.

Recanalisation of embolised cerebral aneurysms afflicts fewer than one in five patients in a 12-month follow-up, and is most often asymptomatic.

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