Midterm Outcome of Partially Thrombosed Intracranial Aneurysms Treated with Guglielmi Detachable Coils

S.J. KIM *, I.S. CHOI

Department of Interventional Neuroradiology, Lahey Clinic; Burlington, Massachusetts

* Department of Radiology Dankook University College of Medicine; Cheonan

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Summary

We evaluated the results of Guglielmi detachable coil (GDC) treatment in partially thrombosed aneurysms and determined if there is high rate of recanalisation on follow-up.

Among 149 treated aneurysms in 141 patients, 25 CT- or MR-confirmed partially thrombosed aneurysms were selected for evaluation. The features of thrombosed aneurysms and percentage of occlusion were analysed on initial angiograms. Follow-up angiograms, which were available in 18 cases, were evaluated for aneurysm lumen recanalisation. The recanalisation rate was compared with that of non-thrombosed aneurysms treated with GDCs.

Locations of aneurysms were as follows: cavernous carotid ten; ophthalmic four; p-com. two; MCA one; A-com. one; basilar tip four; midbasilar two; PICA one. The size of the aneurysm lumen ranged from 5 to 30 mm (mean 16.8 mm) on angiograms, but on cross sectional images the size of aneurysms ranged from 13 to 70 mm (mean 24.6 mm). The extent of aneurysmal thrombosis ranged from 10 to 90 per cent (mean 46.4 per cent). On initial GDC treatment, total to subtotal occlusion was achieved in 18 cases out of 25 (72%). Of the 18 follow-up angiograms, 14 cases (77.8%) showed recanalisation ranging from 10 to 60 per cent of aneurysm size. Luminal recanalisation was due to migration (10 of 14) or compaction (4 of 14) of coil masses. In two cases, symptoms recurred in association with aneurysm recanalisation, but in no instance was haemorrhage noted. Attempts for

retreatment were made in ten cases with success in six. In comparison, 14 (15.9%) out of 88 non-thrombosed cases revealed recanalisation on follow-up angiography.

Midterm follow-up angiograms in partially thrombosed aneurysms treated with GDC revealed a fivefold higher rate of recanalisation than in non-thrombosed cases. Close follow-up is necessary in patients with thrombosed aneurysms treated with GDCs.

Introduction

Thrombosis is known to occur in 9% to 13% of intracranial aneurysms with the incidence reported as high as 20% in giant aneurysms¹⁻³. Although surgical clipping is a well-established treatment for intracranial aneurysms, a thrombus within aneurysms presents difficulties for clipping⁴. For these surgically difficult or inoperable cases endovascular treatment can provide a reasonable alternative. Occlusion of aneurysms with Guglielmi detachable coils (GDCs) is widely accepted as a safe and effective endovascular treatment. However, thrombosed aneurysms are reported to have a tendency to incomplete initial occlusion and frequent recanalisation after embolisation with GDCs⁵⁻⁷. This information is based on sporadic case reports and the true incidence of incomplete initial occlusion or late recanalisation is not well known.

We evaluated the initial treatment result and

follow-up recanalisation rate of Guglielmi detachable coil (GDC) treatment in partially thrombosed aneurysms and compared these results with those of non-thrombosed aneurysms.

Patients and Methods

Among 149 intracranial aneurysms in 141 patients treated with GDCs, 25 aneurysms with partial intraluminal thrombosis were selected for evaluation. The remaining 124 non-thrombosed aneurysms in 116 patients were also analysed for comparison. Presence of a thrombus was confirmed by CT and/or MR images. MR criteria for presence of a thrombus included (1) concentric or eccentric signal intensity around flow void signal, or (2) heterogeneous signal intensity in the lumen, and CT evidence of an intraluminal thrombus was an area of non-enhancement⁸⁻¹¹. When equivocal, angiography was used to determine whether a thrombus was present or not: discrepant small size of the aneurysm on angiography compared with that on CT or MR was considered as an evidence of partial thrombosis.

Presenting symptoms of thrombosed aneurysm patients were variable. Subarachnoid haemorrhage was noted in four patients. Other presenting symptoms included cranial nerve palsy in ten, headache/dizziness in nine, aphasia/slurred speech in three, confusion in four, facial/orbital pain in two and ataxia in two. Three aneurysms were found incidentally, and in one patient the aneurysm developed as a complication of transsphenoidal hypophysectomy.

Characteristics of the thrombosed aneurysms

With respect to location, thrombosed aneurysms were found in the cavernous carotid artery (10), ophthalmic artery origin (4), posterior communicating artery origin (2), middle cerebral artery (1), anterior communicating artery (1), basilar tip (4), midbasilar (2), and posterior inferior cerebellar artery (1). The actual size of aneurysm on cross sectional images ranged from 13 to 70 mm (mean 24.6 mm), but patent aneurysm lumen size ranged from 5 to 30 mm (mean 16.8 mm) on angiogram.

We classified the aneurysms into three groups by their size. Criteria for small aneurysms were less than 10 mm in the largest di-

ameter, large between 10 to 25 mm, and giant, 25 mm and greater. Angiography was utilized to evaluate initial occlusion rate and follow-up recanalisation rate.

The extent of aneurysmal thrombosis ranged from 10 to 90 per cent (mean 46.4 per cent) of the whole sac based on cross sectional imaging.

Evaluation of initial treatment results

Results of initial GDC treatment were divided into three groups based on the occlusion rate of the lumen: total, 100%; subtotal, 95-99%; and incomplete, under 95%. We compared the occlusion rates between thrombosed and non-thrombosed groups. We also evaluated occlusion rates according to aneurysm size in thrombosed aneurysms. Statistical analyses were performed using chi square test or Fisher's exact test. Complications related to procedure and symptom improvement after treatment was reviewed.

Evaluation of follow-up results

Follow-up angiograms were available in 18 patients and extended from 3 months to 4 years. We estimated aneurysmal lumen recanalisation rate and evaluated the presence of compaction or migration of coil mass on follow-up angiogram. We compared the recanalisation rate between thrombosed and non-thrombosed groups. The recanalisation rate was also compared in different aneurysm size groups. Statistical analyses were performed using chi square test or Fisher's exact test.

Results

Thrombosed aneurysms accounted for 25 cases (16.8%) of the 149 aneurysms treated with GDCs. On initial treatment with GDCs, total to subtotal occlusion (95% plus) was achieved in 18 cases (72%) out of 25 thrombosed aneurysms (table 1). In the non-thrombosed group 104 cases (83.9%) were occluded totally or subtotally. The difference in initial occlusion rate was not statistically significant between the two groups. In the thrombosed group the difference in occlusion rate between small and large/giant aneurysm groups was not significant, either. However, in the non-thrombosed group there was a significant difference

Figure 1 Non-recanalisation. Pre-contrast CT (A) reveals high density mass with rim calcification in the left suprasellar area. The denser area (arrow) represents the lumen and the surrounding low density area represents a thrombus. Left carotid angiogram (B) shows an aneurysm arising from the ophthalmic artery origin. Post-procedure angiogram (C) shows more than 95% occlusion of lumen with GDCs. On seven month follow-up angiogram (D), there is no change in coil position or shape.

between different size groups: that is, small aneurysms showed a higher rate of total to subtotal occlusion than in the large/giant aneurysm group.

Three patients underwent carotid artery trapping or surgical clipping of aneurysm during or shortly after the endovascular procedure

either due to associated vascular pathology or to remove mass effect.

After endovascular treatment with GDCs, symptoms improved in 14 cases and in four cases there was no change in symptoms. The remaining seven cases, which were excluded from symptom review, were as follows: three post-

Table 1 Comparison of initial occlusion rate between thrombosed and non-thrombosed aneurysm groups

Initial occlusion (%)	Thrombosed ¹⁾				Non-thrombosed			
	²⁾ <10	10-25	25≤	Total	³⁾ <10	10-25	25≤	Total
≥ 95	3	13	2	18	57	44	3	104
≤ 95	0	5	2	7	5	12	3	20
	3	18	4	25	62	56	6	124

NOTE: 1) Non-significant by Fisher's exact test, comparison between thrombosed and non-thrombosed groups; 2) Non-significant by Fisher's exact test, comparison between small and large/giant groups; 3) $p < 0.05$ by Chi-square test, comparison between small and large/giant groups.

Table 2 Comparison of recanalisation rate between thrombosed and non-thrombosed groups

Reopen	Thrombosed ¹⁾				Non-thrombosed			
	²⁾ <10	10-25	25≤	Total	³⁾ <10	10-25	25≤	Total
Yes	1	10	3	14	2	9	3	14
No	1	3	0	4	45	27	2	74
No FU	1	5	1	7	15	20	1	36
Total	3	18	4	25	62	56	6	124

NOTE: 1) $p < 0.01$ by Fisher's exact test, comparison between thrombosed and non-thrombosed groups; 2) Non-significant by Fisher's exact test, comparison between small and large/giant groups; 3) $p < 0.01$ by Chi-square test, comparison between small and large/giant groups; FU: follow-up.

GDC parent vessel trapping or clipping, three incidentally found aneurysms, and one in which the medical record was not available. Thirteen out of these 18 patients had pre-procedural symptoms which were attributed to mass effect of the aneurysms. Among them, ten showed symptom improvement after the procedure and three patients showed no improvement. Complications related to initial GDC treatment were puncture site haematoma or thrombosis in two.

Follow-up angiograms were available in 18 cases out of 25 in the thrombosed group and 88 cases out of 124 in the non-thrombosed group. Recanalisation of the aneurysm lumen was noted in 14 cases (77.8%) of 18 thrombosed aneurysms (table 2). Four cases showed no luminal enlargement or reopening on follow-up (figure 1). In the non-thrombosed group 14 cases (16%) out of 88 showed recanalisation. The difference in recanalisation rate between the two groups was statistically significant. The degree of luminal recanalisation varied from 10 to 60 per cent in the thrombosed group. Lu-

minal recanalisation was due to either migration of the coil mass (10 cases) (figure 2) into the surrounding thrombus or compaction of the coil mass (4 cases) (figure 3), but it usually occurred in combination at least to some degree (figure 4).

Symptoms recurred in two cases in association with aneurysm recanalisation, but in no instance was haemorrhage noted. Attempt for retreatment was made in ten cases with success in six. However, in one case of basilar tip aneurysm, recanalisation occurred necessitating retreatment with GDCs on three subsequent occasions. In this patient actual growth of the aneurysm was noted (figure 4). Similar post-treatment aneurysm growth was noted in four other cases.

Discussion

Some degree of spontaneous thrombosis occurs in 9% to 13% of intracranial aneurysms. Thrombosed aneurysms tend to be large¹² and conversely, larger aneurysms tend to throm-

Figure 2 Migration of coil mesh. T1-weighted MR (A) shows an elongated shape aneurysm arising from the basilar artery with internal multilayer high signal (arrow). Left vertebral angiogram (B) demonstrates a 7 mm aneurysm arising from the superior cerebellar artery origin. The superior cerebellar artery is not visible. Immediate post-procedure angiogram (C) shows complete occlusion of the aneurysm. On 7 month follow-up angiogram (D), coil mesh migrated laterally without significant change in coil mesh shape and recanalised lumen.

bose more frequently. The size distribution of the aneurysms in general is as follows: small 60-78%; large 20-30%; giant 2-13%⁴. Compared to this, actual size (i.e., on CT or MR) of all thrombosed aneurysms in our series was large or giant and giant aneurysms accounted for 36%. Our result corresponds well with other reports that 40% of thrombosed aneurysms are giant⁴. The distribution of thrombosed aneurysms was very different from that of general

aneurysms. Generally, about 70% of intracranial aneurysms are found in or above the circle of Willis, in the anterior circulation, that is, anterior communicating artery, posterior communicating artery and middle cerebral artery bifurcation⁴. On the contrary, most of the thrombosed aneurysms in our series were found in the internal carotid artery below the posterior communicating artery and in the vertebrobasilar system. Cavernous carotid artery was the

Figure 3 Compaction of coil mesh. Contrast enhanced CT (A) shows an enhancing aneurysm in the left posterior communicating artery origin area. Note focal non-enhancing portion representing a thrombus. Left internal carotid angiogram (B) shows an aneurysm at the same location. The aneurysm was occluded with GDCs and achieved 95% occlusion (C). Eight month follow-up angiogram (D) shows coil mesh compaction without migration.

single most common location accounting for ten cases (40%), followed by the posterior circulation (seven cases, 28%) including basilar tip.

Endovascular treatment of partially thrombosed aneurysms entails various difficulties compared with the non-thrombosed group. Recanalisation is frequently reported with GDC treatment. Additionally, initial occlusion is more difficult and packing of the aneurysm carries a high risk of embolism¹³. Some authors

claim it is difficult to eliminate the mass effect in case of large or giant aneurysms. The overall reported incidence of aneurysm luminal recanalisation after GDC treatment varies widely, ranging from 5% in small neck aneurysms and up to 65% in wide neck aneurysms^{5,14-18}. It is well known that the size of the aneurysm neck is the most important factor for the degree of aneurysm occlusion and follow-up recanalisation^{16,19-21}. The size of aneurysm, initial occlusion rate and presence of luminal throm-

bus can also affect the recanalisation rate after GDC treatment. Although recanalisation of the aneurysm lumen after GDC treatment is commonly reported in thrombosed aneurysms, the incidence is not well known. In our study, the rate of recanalisation was 78%, five times higher than in the non-thrombosed group.

The mechanism of luminal recanalisation after occlusion of the aneurysm with GDCs is still unclear. It is generally accepted that coil compaction causes regrowth of the aneurysm²²⁻²⁵. Compaction is believed to occur by arterial pressure and the constant pulsatile flow against the coil mass^{17,19,26}. In our 14 cases of luminal recanalisation after GDC treatment, we observed two patterns of changes in coil mass, which caused recanalisation: one was shrinkage (compaction) of the coil mass (four cases) and the other was migration of the coil mass (ten cases) into the thrombus. In reviewing the literature little distinction is made between coil compaction and migration and the term compaction seems to be generally by many authors used to express both shrinkage and displacement of coils. In our experience they represent separate entities in partially thrombosed aneurysms. We define migration as the entire coil mass moving inside the aneurysm, whereas, compaction represents a shrinking of the coil mass. Typically one predominates, but at least to some degree these phenomena occur in combination.

Compaction of the coil mass may be in part caused by underpacking of the coils²². Szikora²⁷, in his experimental model, showed that only 23-26% endovascular volume is occupied by coil even though packing seemed dense. Compaction occurs more easily when a large or giant aneurysm is partially occluded^{11,14,15}. Because thrombosed aneurysms are usually larger in size, it is easily conceivable that they are often underpacked compared to non-thrombosed ones. The high frequency of coil migration seems to be due to a thrombus in aneurysm. Migration of the coil mass may result from embedding of the coil mass into soft thrombus, partial resolution of a thrombus or enlargement of the aneurysm itself^{7,22,26,28-30}. We consider coil migration one of the characteristics of thrombosed aneurysms. In addition, a wide neck may have influenced more frequent recanalisation after GDC treatment.

Aneurysm recanalisation did not directly re-

sult in clinical recurrence. Although recanalisation of the lumen was noted in 14 patients, only two patients had symptomatic recurrence related to recanalisation. We attempted retreatment in ten patients with success in six. In the four failed cases, the neck was too wide (three cases) or access was not possible (one case). Including four cases without recanalisation, we achieved more than 95% of luminal occlusion in ten cases (40%).

Another possible problem related to endovascular treatment of thrombosed aneurysm is difficulty in eliminating mass effect because thrombosed aneurysms tend to be large or giant in size^{5,31}. A large percentage of clinical symptoms are related to the mass effect of large or giant aneurysms. In one case with a 7-cm giant ophthalmic artery origin aneurysm, surgery was performed after successful embolisation with GDCs because of mass effect. Initial GDC treatment relieved symptoms related to mass effect in ten of 13 patients. The reason for improvement of symptoms is probably that by relieving pulsation, mass effect also was relieved. Others reported an actual decrease in aneurysm volume in some cases on follow-up³². We believe successful treatment with GDC can reduce the mass-related symptoms unless the aneurysm is exceptionally large.

Our initial occlusion rate was slightly lower than in the non-thrombosed group. However there was no statistically significant difference in occlusion rate between the thrombosed and non-thrombosed groups. The main reason for lower initial occlusion rate can be explained as broad neck and large size of the thrombosed aneurysm²².

Risk of thromboembolism is high with surgical treatment of thrombosed aneurysms³³. Although there is also a potential risk of embolism from aneurysmal thrombus during endovascular treatment, no embolic complication was noted in our series³¹.

There are some limitations in this study. Since the MR appearance of the aneurysms is variable, there is a chance of mistaking non-thrombosed aneurysms as thrombosed if they have atypical MR signal characteristics^{8,10}. To minimize this possibility we used more or less strict criteria for the presence of a thrombus on CT or MR and if they were equivocal, we matched with angiographic findings to determine the presence or absence of a thrombus.

On the other hand, because we used strict criteria, there is a chance of missing patients with thrombosis if CT or MR was not available or they had a small size thrombus with equivocal CT/MR and angiographic appearance.

Another possible source of error is the small sample size of the thrombosed aneurysm group. To reduce possible error, we simplified the size group into small and large/giant in comparing initial occlusion rate and recanalisation rate within the group and with the non-thrombosed group. In our series of thrombosed aneurysms, although there was a tendency for lower initial occlusion rate and higher recanalisation rate, statistically, there was no significant difference between different size groups. This seems to be mainly due to the small number of

patients. We were also unable to analyse different behaviors of aneurysms based on their locations, again because of small sample size.

Conclusions

In thrombosed aneurysms initial occlusion rates with GDCs are slightly lower than those in the non-thrombosed group, but the incidence of recanalisation in the thrombosed group is up to fivefold greater than in the non-thrombosed group.

The pattern of recanalisation was mainly migration of coil mass into the thrombus. Close follow-up is required in patients treated with GDC and if necessary, retreatment should be performed.

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In Sup Choi, M.D.
 Department of Interventional Neuroradiology
 Lahey Clinic
 41 Mall Road
 Burlington, MA 01805 U.S.A.

COMMENT

The authors intended to evaluate Guglielmi detachable coil (GDC) treatment in partially thrombosed aneurysms. They performed a retrospective study in 141 patients and studied the rate of recanalisation on the outcome in thrombosed aneurysm in comparison to non-thrombosed aneurysms also treated with GDC.

- The small sample size of the thrombosed aneurysm group in comparison to the non-thrombosed one remains problematic for the analysis of the data, although non-thrombosed aneurysms are clinically more frequent. The authors attempted to resolve this problem by dividing the groups in small/large/giant aneurysm. There is a good methodological approach, but the number of patients lost for follow-up represents almost 25% in the group of thrombosed aneurysms and 30% in non thrombosed one. Thus, the difference in recanalisation rate described as statistically significant, loses in power.*
- The proportion of thrombosed aneurysm is greater in large/giant ones, as it is described in the literature, so a question arises whether non thrombosed giant aneurysms recanalize more often than non thrombosed small aneurysms. The authors say that there are no differences in recanalisation rate within the group in non-thrombosed aneurysm, but this argument is difficult to accept as 40% of patients of the group of large size aneurysm have no follow-up. The relation between the thrombotic aspect and the size of the aneurysm might be discussed as a confounding factor in the differences between non-thrombosed and thrombosed aneurysms, the latter type being more frequent in larger aneurysms.*
- This study brings interesting information about the incidence of recanalisation in thrombosed aneurysms, and useful features in the treatment of recurrent recanalisation (success in 40% of the cases).*
- Several authors in the literature report that thrombosed aneurysms are usually larger in size and therefore often underpacked. This study shows that there is no difference in the comparison of initial occlusion rate between thrombosed and non-thrombosed aneurysm. The discussion of morphological aspects of the aneurysm, like the neck (small or wide), might be useful in the understanding of the recanalisation process.*

The study of Kim and Choi brings useful informations about the incidence and the treatment of the recanalisation of thrombosed aneurysm, despite some weaknesses concerning the follow-up of patients which decreases the importance of the difference described with non-thrombosed aneurysms.

Mikaël Mazighi
Service de Neurologie
Hôpital Lariboisière
2 Rue A. Paré
75018 Paris - France

COMMENT

The article by Kim and Choi is the second report in this issue of the journal that deals with GDC treatment of intracranial aneurysms, with this one being focused on a specific subgroup of patients. Their retrospective review of 25 patients with partially thrombosed (PT) aneurysms treated with GDC aims to enumerate the incidence of incomplete initial occlusion and subsequent recanalisation and compare these to the rates in 124 aneurysms which were not thrombosed (NT), postulating that there may be a higher rate of recanalisation in the former group. Several key variables, such as criteria for "thrombosis" and the method for measurement of aneurysm size, are clearly defined. The PT group had a higher percentage of large or giant aneurysms (22/25, 88%) than the NT group (62/124, 50%). For the PT group, the initial total or subtotal occlusion rate ($\geq 95\%$ occlusion) was lower than for the NT group, although not significantly so (72% vs. 84%, respectively), and the rate of recanalisation on follow-up was higher (78% vs. 16%, respectively, which was statistically significant). The authors conclude that the recanalisation rate is five times higher in partially thrombosed aneurysms, and recommend close follow-up of these patients. A useful discussion of the distinction between coil migration and compaction, and the postulated mechanism of each, is included (with the former being more often responsible for recanalisation in their series). The beneficial effects of treatment on symptoms of mass effect are alluded to, with improvement seen in ten out of thirteen patients.

The numbers provided are suggestive of a worrisome pattern of behaviour post-treatment in the group of aneurysms that contain thrombus. Few would argue with the need for follow-up in the type of aneurysms described, given concern about GDC treatment for large, wide-neck and partially thrombosed aneurysms^{1,3,6,7,8,9} and subsequent changes in aneurysms which are not completely occluded with the initial treatment^{1,2,3,5,6,7}. From this study alone, we cannot definitively state the magnitude of the difference or to which factor(s) the higher recanalisation rate should be attributed. The mean length of angiographic follow-up, or number of aneurysms studied beyond a given length of time, is not specified for either group. A discrepancy in follow-up between the two groups could account for some of the differential recanalisation rate (specifically, if the PT group has a longer mean follow-up, and if recanalisation is a function time after treatment). The authors also clearly recognize and try to account for potential confounding due to differences in size of the aneurysms in the PT and NT groups, since there is a documented relationship between aneurysm size and the rate of initial incomplete treatment and subsequent recanalisation^{3,4}. The fact that they did not find a significant relationship between size and either initial occlusion rate or recanalisation on follow-up may relate to sample size, as they mention. This is supported by the fact that for the NT group, where subgroups are larger, significant differences were found for both outcomes, with lower initial success and higher recanalisation in larger aneurysms, as one would expect. This emphasizes the statistical caveat that failing to find a significant difference between groups does not imply equivalence, since there may be inadequate statistical power to have demonstrated a difference unless it is extreme. Although the authors acknowledge the potential importance of another variable, neck width, in aneurysm occlusion

and follow-up recanalisation, no data on neck width is provided here to support the results previously found by other groups^{1,7,9}. Because the variables of aneurysm size, neck width and thrombosis are associated with each other, it becomes very difficult to definitively sort out which factor accounts for an observed difference without having very large sample sizes and a distribution of patients among the various categories of each variable. Until those distinctions are clear, we will not be able to be dogmatic about a management strategy based on individual attributes of an aneurysm.

The reference here to the postulated mechanism of coil compaction raises a similar question to the discussion by Fuse et Al and many other authors^{1,3,5,7,8} regarding the importance of the degree of packing and its influence on the subsequent course. The point beyond which packing will fill enough of the volume of the aneurysm to prevent compaction is unclear. Regarding the degree of occlusion, one wonders whether it would be safer to categorize aneurysms with 95-99% occlusion together with the "incomplete" group rather than the "total occlusion" group, considering any residual as a potential risk. The answer to this of course depends on the subsequent behaviour of those aneurysms, and whether one considers radiographic or clinical criteria (or a combination of the two) for defining failure and the need for retreatment.

Ultimately, this article provides a look at a recurrent problem, incomplete treatment and recanalisation, with a systematic focus on one potential culprit, pre-treatment aneurysm thrombosis. The advice for close follow-up is prudent, based on this report and previous experience related to aneurysm size and other factors, which thus far remain inextricably confounded. Furthermore, such a high rate of recanalisation encourages the consideration of other treatment alternatives for this subgroup of patients.

Phillip J. Porter, M.D.

Neurosurgery and Interventional Neuroradiology
The University of Toronto Brain Vascular Malformation Study Group
University Health Network, Toronto Western Division
Toronto, Canada

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