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The ESMINT Retrospective Analysis of Delayed Aneurysm Ruptures after flow diversion (RADAR) study

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Abstract

Introduction: Rupture following flow diversion treatment has been reported as a complication of this technique. The aim of this survey was to investigate the efficacy of flow diversion by retrospectively analysing the rate of delayed ruptures in all centres using this technology.

Methods: A questionnaire based survey about thromboembolic and haemorrhagic complications related to flow diversion treatment, with focus on delayed ruptures, was distributed to clinical practitioners. Parenchymal haemorrhages were collected and analysed separately from aneurysmal bleeding. Detailed morphological and clinical data was available on a subgroup of 720 of 1421 aneurysms reported (Group 1). In another 581 cases detailed data was available on ruptured cases only. The full cohort of 1421 aneurysms in 1274 patients in Group 2 was used to calculate the incidence of delayed rupture.

Results: In Group 1 procedural thromboembolic complications were reported in 48 of 720 aneurysm treatments (6.7 %). Procedural parenchymal bleeds and subarachnoid haemorrhage occurred in 1.8 % and 14 delayed parenchymal bleeds (1.9 %) were reported. In Group 2, 14 delayed ruptures were reported (1 %), with 13 subarachnoid bleeds and a single case of carotid-cavernous fistula development. All ruptured aneurysms were >10 mm, with a mean maximal diameter of 24 mm. The median time to rupture after treatment was 9 days. The incidence of delayed rupture was 2.1 % in the subgroup of aneurysms >10mm.

Conclusions: Delayed ruptures affected large aneurysms with a mean diameter of 24 mm, suggesting that very large to giant aneurysms are prone to this risk.

Keywords: flow diverter, aneurysm, flow diversion, cerebral, delayed rupture

Introduction

The concept of flow diversion represents a major paradigm shift in the endovascular aneurysm treatment. Aside from conventional endosaccular aneurysm treatment, flow diverters (FD) provide the potential for diseased parent artery reconstruction and cure, with an expectation of lower recurrence risk. The theoretical background for this technology relies on the fact that FDs induce a change in arterial and intra-aneurysmal hemodynamics, which translates to progressive thrombosis, occlusion and healing of the aneurysm. Since the location, morphology and size of aneurysms vary, the induced hemodynamic effect is also expected to vary from one case to another. In general, the time course of the healing process is as yet unclear. Individual differences are likely, however, since healing is influenced by multiple factors including the type of FD and resultant flow change, parent vessel geometry, aneurysm size and

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morphology and applied antithrombotic medication. Consequently, the aneurysm may remain unprotected from rupture for an unknown period after treatment.

Since their introduction into clinical practice, FDs have been shown to be extremely effective in complete and lasting occlusion of large, giant and broad neck aneurysms with acceptably low procedural complication rates [1-3]. However, delayed aneurysm ruptures following seemingly successful treatment have been reported, representing a significant deficiency in the efficacy of the technique in relation to the primary goal of treatment, namely to prevent aneurysm rupture.

Published case series focus on the potential mechanisms of delayed ruptures but do not provide information of its incidence. Therefore, the true magnitude of flow diversion effectiveness remains unclear. Due to the small sample size and the low likelihood of such events, the incidence of delayed rupture cannot be determined from individual case series. Moreover, large study and registry results are not yet available. Thus, in order to estimate the true effectiveness of FD in preventing aneurysm rupture we collected data on the incidence of delayed rupture on a large number of cases using an international survey of FD users.

Materials and methods

A task force established by the Executive Committee of the European Society of Minimally Invasive Neurological Therapies (ESMINT) initiated a survey in December 2010. The survey aimed retrospectively to analyse the rate of aneurysm rupture following FD treatment in all centres applying the technology prior to the end of 2010. Participation in the survey was voluntary, motivated by scientific contribution and did not include any financial compensation for providing data.

A questionnaire was created and distributed through the ESMINT Office electronically to all members of the ESMINT Society and to clinical practitioners using the technology worldwide. Two companies producing commercially available FDs at the time of the study, Balt International (Montmorency, France), and Covidien/ev3 Inc. (Irvine, CA, USA), provided a list of active centres.

The questionnaire consisted of two parts. The first part gathered information about the number of consecutive aneurysms treated by flow diversion at the respective centre until the end of 2010. In addition to the locations and size distribution of treated aneurysms with a cutoff at 10 mm, we collected information on FD brands used and the number and type of complications. In order to differentiate aneurysm rupture following treatment from other types of complications, responders were asked to classify each event into one of the following categories: procedural (within 24 h of treatment) thromboembolic or haemorrhagic events and delayed (more than 24 h from treatment) thromboembolic or haemorrhagic events. Haemorrhagic incidents were further divided into subarachnoid or parenchymal bleeds.

The second part of the questionnaire focused on the aneurysm cases with delayed ruptures only and collected data on demographics; symptoms prior to treatment; aneurysm location; aneurysm morphology (neck, largest diameter, dome height and aspect ratio); the number of FDs used at the level of the aneurysm segment; additional intrasaccular coil use; property of FD deployment; thrombosis status of aneurysm prior to rupture as assessed by the last available imaging before the event; and clinical

outcome. The submitted data were accepted without being audited. A database was created for further statistical analysis. Altogether, 53 worldwide centres responded to the invitation: Europe (35 centres), Australia (6 centres), Asia (5 centres), North America (4 centres), South America (2 centres) and Africa (1 centre).

Fifty-one centres submitted detailed questionnaires comprising 697 patients with 720 aneurysms, resulting in a mean of 14 cases/centre (range 1–79). These cases were considered as Group 1 and constituted the basis for detailed procedural risk analysis.

Two other major centres reported another 581 cases, but only provided detailed morphological data for those aneurysms that ruptured in a delayed fashion. These cases could not be used for assessing the procedural risk. However, along with such cases in Group 1, the 581 cases provided an opportunity to analyse the incidence of delayed rupture and specifying the type of aneurysms at risk. Therefore, both Group 1 and the additional 581 cases (totalling in 1421 aneurysms in 1274 patients) were considered as Group 2 and served as the basis of delayed rupture incidence estimation.

Results

Group 1: Aneurysm characteristics and procedural complications

In Group 1 (N=720), detailed data on aneurysm characteristics was available for 668 aneurysms, since one centre failed to submit the location data except for aneurysms >10 mm (N=52). Locations of aneurysms are listed in Table 1. The vast majority of aneurysms (N=531) were located in the anterior circulation (79 %). In total, 511 of 720 aneurysms were larger than 10 mm (71 %). In this subgroup anterior circulation was represented at a similar rate of 82 %. The majority of aneurysms were located at the cavernous and paraopthalmic segments - in 56 % and 59 % of cases in all aneurysms and in the subgroup of aneurysms >10 mm respectively. The internal carotid artery (ICA) was by far the most treated artery, with 73.5 % overall and 76.7 % in the >10 mm aneurysm group.

Procedural thromboembolic complications were reported in 48 of 720 aneurysm treatments (6.7 %). In terms of procedural haemorrhagic incidents, parenchymal bleeds were reported in 7 cases (1 %) and procedure related subarachnoid haemmorhage (SAH) in 6 cases (0.8 %). Ischaemic and haemorrhagic procedural complications were reported in 61 cases (8.5 %) (Table 2). Delayed complications included 14 delayed parenchymal haemorrhages (1.9 %) and 33 delayed thromboembolic incidents among the 720 patients in Group 1 (4.6 %).

Group 2: Delayed aneurysm ruptures

There were 14 delayed ruptures among the 1421 aneurysms treated (1 %), with 13 subarachnoid bleeds and one carotid cavernous fistula (CCF) development. Since all ruptured aneurysms were equal to or larger than 10 mm, the incidence of delayed rupture in this subgroup was 2.1 %. The mean age of these patients was 63 years. Twelve of the 14 aneurysms were symptomatic prior to treatment due to mass effect on adjacent neural structures or headaches, and 1 patient presented with a previous SAH.

The largest diameter of the aneurysms with delayed rupture had a mean diameter of 24 + 7 mm (range 10 – 37 mm). Only 1 aneurysm with a maximal diameter of 10 mm ruptured 300 days post treatment. The remaining 10 aneurysms were all larger than 19 mm. Nine aneurysms were treated with a single layer FD, 4 aneurysms received 2 layers and 1 aneurysm 3 layers of FD across the aneurysm neck. No additional endosaccular coils were used in either of these cases in conjunction with FD treatment.

The mean time to rupture after treatment was 60 days (range 3 – 300) with a median value of 9 days. Ten out of the 14 delayed ruptures presented in less than 3 months, with a mean time of 12 days after treatment (range 3 to 58 days). Four patients showed a later rupture (> 3 months after treatment) with a mean treatment-to-rupture time of 180 days (range 110 to 300 days). Data on grade of aneurysm thrombosis at the time or shortly before rupture were available in 9 patients, with a mean estimated degree of thrombosis of 64 % of the aneurysm volume. One patient with a cavernous ICA-aneurysm developed a CCF after rupture. From the remaining 13 patients with SAH, 10 died immediately. Two patients remained in a vegetative state and 1 patient survived without clinical consequences. This patient was retreated with further FDs soon after the bleeding.

Discussion

This multicentre, worldwide, retrospective survey on delayed rupture after flow diversion treatment of cerebral aneurysms comprising 1421 aneurysms, showed that delayed rupture occurred in 1 % of all treated cases. In the subgroup of aneurysms larger than 10 mm, the incidence of delayed rupture was 2.1 %. All except 1 aneurysm with delayed rupture were larger than 19 mm, with an average size of 24 mm, demonstrating that this event primarily affects very large to giant size lesions.

The braided structure FDs, designed with low porosity and high pore density, as well as soft structure and flexibility, are able to conform completely to the parent vessel wall. By redirecting the flow into the parent artery and simultaneously reducing the intra-aneurysmal flow, they induce a hemodynamic change in the parent vessel and in the aneurysm. Based on these characteristics, braided structure FD devices can induce complete thrombosis of the aneurysm as a stand-alone treatment. This methodology provided a real paradigm shift in the endovascular aneurysm therapy from endosaccular treatment to parent vessel reconstruction. The case series published to date have indicated significant mid- and long term success in aneurysms which are otherwise difficult to treat or not amenable for endovascular reconstructive treatment [1-3, 5-8].

As the technique has become more popular and increasing numbers of aneurysms treated worldwide, sporadic cases of delayed aneurysm ruptures have been observed. After the first description of a paraopthalmic aneurysm rupturing 3 weeks after treatment [9], a series of similar cases were collected and analysed [4]. According to the results of Kulcsár et al., all aneurysms from this series were very large or giant with a mean dimension of 22 mm. The majority of patients were symptomatic, and 12 of the 13 aneurysms analysed showed a high volume thrombosis prior to rupture. The authors observed 2 groups of aneurysms, rupturing before and after aneurysm treatment. As in the present survey, the majority belonged to the earlier delayed rupture group, with a mean time to rupture of 16 days. Based on histopathological studies, the authors postulated that intra-aneurysmal thrombus was the key factor in inducing inflammation and aggressive autolysis, leading to complete destruction and dissolution of the aneurysm wall. This concept was also supported by previous evidence of aortic and cerebral aneurysm

ruptures related to presence of intra-luminal thrombus [10-14]. Very large and giant aneurysms treated by FD are rapidly able to accumulate great clot volume, which may have an overwhelming inflammatory and enzymatic activity. Intra-aneurysmal thrombosis (>50% of thrombosis) induced inflammatory process with peri-aneurysmal vasogenic brain oedema and blood-brain barrier breakdown was found in a series of FD treated cerebral aneurysm patients, where again large aneurysm size was the most important predictive factor [15].

In a computational flow analysis study of aneurysms treated with FDs showing immediate or delayed ruptures, the authors suggested that the FD-induced increase of intra-aneurysmal pressure was a possible cause of rupture, not taking into account the presence of an eventual thrombosis in cases with delayed bleeds [16].

The results of the ESMINT Retrospective Analysis of Delayed Aneurysm Ruptures after Flow Diversion (RADAR) survey presented here reflect previous findings. Except for a single aneurysm measuring 10 mm, all other lesions with delayed rupture were very large or giant sized, with a mean diameter of 24 mm. As in the previous series of Kulcsár et al. [4], this cohort also showed that the majority (10 of 14) of aneurysms ruptured relatively early (within 3 months) after treatment, with a mean time-to rupture of 12 days. Interestingly, the symptomatic 10 mm basilar artery aneurysm ruptured 300 days post treatment. Another shared finding with the previous series is that almost all lesions were symptomatic prior to treatment, which may be size and thus mass effect related, but may also suggest recent growth and instability of the aneurysm.

The main purpose of the survey was to estimate the incidence of delayed ruptures after flow diversion treatment. Due to repeated announcements and requests sent out to practitioners registered to have performed flow diversion aneurysm treatments, a high response rate of 53 centres was achieved, accounting for the large number of 1421 aneurysms treated with this technique. Although the sample does not cover all cases treated worldwide, due to the large number, it may still be considered representative. The results indicate an incidence of 1 % of delayed ruptures in the whole group. Keeping in mind previous results showing that delayed ruptures were affecting large and giant aneurysms, a cut-off value of 10 mm was predefined in the survey. In the subgroup of aneurysms larger than 10 mm the incidence was 2.1 %. These data concur with previous estimates [4]. In the largest prospective single arm study on flow diversion (Pipeline for Uncoilable or Failed Aneurysms, PUFS) 4.6 % patients suffered parenchymal haemorrhages for various reasons, but none resulted from delayed aneurysm rupture. Two patients (1.9 %), however, developed CCF, likely to be related to delayed rupture of a cavernous carotid aneurysm. Of note, the mean number of FDs placed per aneurysm in this study was 3.1 [17].

Interestingly, in the present RADAR series, no additional coils were used in any of the aneurysms with delayed rupture, which replicates previous data, where additional coiling was performed in only 1 of the 13 cases [4]. This does not necessarily reflect the usefulness of additional aneurysm coiling, since we had no data of coil use in general in conjunction with FDs in this survey. Endosaccular coiling alone has, however, already proven its bleeding preventive effect over the years, so in aneurysms at risk, they may be used to prevent delayed aneurysm ruptures.

Investigating the complication rate of FD treatment in general was not the aim of this survey. Yet, it was important to collect data on several kinds of periprocedural and delayed adverse events in order to clearly

separate aneurysm rupture from other complications. Thromboembolic complications were collected separately from haemorrhagic events. Furthermore, complications presenting in the first 24 h (procedural) were separated from those which became evident later (delayed). Complication resulting in haemorrhage may occur during the procedure and be related to mechanical perforation of either the aneurysm itself or of a distal arterial branch. Such perforation may result in slow leakage, becoming symptomatic hours following completion of the procedure, but is highly unlikely to remain silent for more than 24 h. In order to exclude these events from the analysis of aneurysm rupture following successful FD treatment, we used 24 h as a cut off for procedural complications.

The RADAR survey also yielded information on procedural complications. These data reflect the results of previous series both for ischaemic and haemorrhagic events [1, 3, 5, 8, 18, 19]. Delayed parenchymal haemorrhage has been reported following FD treatment and is a cause of increasing concern about the technique. The reported incidence of delayed parenchymal haemorrhage of 1.9 % was similar to the findings of the PUFS study, although lower than results of a recently published series [20].

The major limitations of this study are related to the voluntary participation in the study, the retrospective data collection and the fact that we were not able to audit the data provided. However, the rationale to keep the survey questionnaire simple and of limited detail allowed us to achieve a high response rate.

Conclusions

The results of this survey indicate that delayed aneurysm rupture after flow diversion treatment occurs in 1 % of all aneurysms treated and 2 % of those larger than 10 mm. Delayed ruptures affected aneurysms with a mean diameter of 24 mm, suggesting that very large to giant aneurysms are prone to this risk. Overall, the efficacy of flow diversion in preventing aneurysm from rupture appears to be 99 % in general and 98 % for aneurysms larger than 10 mm. The results concur with previous observations. Reducing the incidence of delayed rupture in very large and giant aneurysms where a reconstructive treatment is favoured and the value of additional intra-saccular aneurysm filling with coils should be further investigated.

Acknowledgments

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Conflict of interest

ZK is proctor for Silk FD implantation (Balt International). IS has consulted for Covidien/ev3.

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Annex 1 - List of participating centers in the RADAR survey

Nr	Reporting Physician	Center	Nr. of cases reported		
1	Lylyk, Pedro	ENERI, Buenos Aires, Argentina	369		Group 2
2	Cekirge, Saruhan - Saatci, Isil	Hacettepe University, Ankara, Turkey	208		
3	Boccardi, Edoardo	Niguarda Hospital, Milan, Italy	79	Group 1	
4	Szikora, Istvan	National Institute of Neurosurgery, Budapest, Hungary	69		
5	Passos, Paulo	Porto Alegre, Brasil	52		
6	Maimon, Shimon	Sourasky Medical Center, Tel Aviv, Israel	33		
7	Campos, Jorge	University of Lisbon, Hospital de Santa Maria, Lisbon, Portugal	33		
8	Wagner, Aase	Rigshospitalet, Copenhagen, Denmark	29		
9	Zeleňák, Kamil	Martin University Hospital, Martin, Slovakia	27		
10	Mangiafico, Salvatore	Careggi University Hospital, Florence, Italy	25		
11	McDougall, Cameron	Barrow Neurological Institute, Phoenix, USA	25		
12	Holtmannspötter, Markus	University of Munich, Germany	24		
13	Berge, Jerome and Barreau, Xavier	CHU Bordeaux, France	23		
14	Wanke, Isabel	University Hospital, Essen, Germany	21		
15	Ciceri, Elisa	Fondazione Besta, Milan, Italy	21		
16	Cognard, Christophe	CHU Toulouse, France	19		
17	Brew, Stefan	Queen Square, London, UK	19		
18	Weill, Alain	CHU, Montreal, Canada	15		
19	Orlov, Kirill	Novosibirsc State Institute of Blood Circulation Pathology, Novosibirsc, Russia	15		
20	Isokangas, Matti & Siniluoto, Topi	Oulu University Hospital, Oulu, Finland	12		
21	Wikholm, Gunnar	Sahlgrenska University Hospital, Gothenburg, Sweden	11		
22	Mitchell, Ken	Royal Brisbane Womans Hospital, Brisbane, Australia	10		
23	Weber,Werner	Klinikum Vest, Recklinghausen, Germany	10		
24	Madan, Anoop	Alfred Hospital, Melbourne, Australia	8		
25	Pierot, Laurent	Hôpital Maison-Blanche, Reims, France	8		
26	Bakke, Søren Jacob	Oslo University Hospital, Rikshospitalet, Oslo Norway	8		
27	Byrne, James	Oxford Radcliffe Hospitals, Oxford, UK	7		
28	Chong, Winston	Monash Medical Centre, Melborune, Australia	7		
29	Holt, Michael E.	Monash Medical Centre Melbourne Australia	6		
30	Hui, Francis	National Neuroscience Institute, Singapore	6		
31	de Miquel, Maria Angeles	Hospital de Bellvitge, Barcelona, Spain	6		
32	Defreyne, Luc	Ghent University Hospital, Belgium	6		
33	Tournade, Alain	Hôpitaux Civils de Colmar, Colmar, France	6		
34	Schumacher, Martin	University Hospital Freiburg, Freiburg, Germany	5		
35	Bajic, Radoslav and Gal Gyula	University Hospital Nord Norge, Tromsø, Norway	5		
36	Wickenhoefer, Ralph	BwZ-Krankenhaus, Koblenz, Germany	4		
37	Holt, Michael E.	St Vincent's Hospital Fitzroy, Melbourne Australia.	4		
38	Eshghi, Omid	University Medical center, Groningen, The Netherlands	4		
39	Shankar, Jai	QE II Hospital, Halifax, Canada	4		
40	Ghassan Abi Chedid	Sacre Coeur Hospital,,Baabda, Lebanon	4		
41	Firat, Mehmet Murat	Gaziosmanpasa University, Tokat, Turkey	4		
42	Lopes, Demetrius	Rush University Medical Center, Chicago, USA	3		
43	Voormolen, Maurits	University Hospital Antwerp, Edegem, Belgium	3		
44	Fesl, Gunther	University of Munich, Germany	3		
45	Engelhardt, Marc	Diakoniekrankenhaus Rotenburg, Rotenburg / Wümme, Germany	3		
46	Rajeev, Padmanahban	James Cook University Hospital, Middlesborough, UK	2		
47	Ricolfi, Frederic	CHU, Dijon France	2		
48	Royston, Duncan	Durban, South Africa	2		
49	Kirsch, Eberhard	Hirslanden Clinic. Aarau, Switzerland	1		
50	van Hasselt, B.A.A.M.	Isala klinieken, Zwolle, the Netherlands	1		
51	Cronqvist, Mats	Skane University Hospital, Lund, Sweden	1		
52	Brooks, Mark	Austin Health, Melbourne, Australia	1		
53	Arnold, Sebastian	Städt. Klinikum, Karlsruhe, Germany	1		
	Total		**1274**		

Tables

Table 1 - Percentage of treated aneurysms according to locations in Group 1. (N=720).

Aneurysm location	% of all aneurysms (N=668)*	% of aneurysms > 10mm (N=511)
ICA - cervical	2	3
ICA - petro-cavernous	23	29
ICA - paraopthalmic	33	30
ICA - C1 segment	15	15
MCA	4	3
ACA	2	2
VA	7	6
BA	11	11
PCA	2	1
Total	**100**	**100**

*One centre (N=52) omitted to submit the localisation of all aneurysms, but only for the aneurysms >10mm

Table 2 - Thromboembolic and haemorrhagic complications related to FD treatment.

	Procedural (Group 1)			Delayed (Group 1 and 2)		
	Thromboembolic	Parenchymal haemorrhage	SAH	Thromboembolic	Parenchymal haemorrhage	Aneurysm rupture
Numbers (%) according to available data	48/720 (6.7%)	7/720 (1%)	6/720 (0.8%)	33/720 (4.6%)	14/720 (1.9%)	14/1421 (1%)