

Volume changes of extremely large and giant intracranial aneurysms after treatment with flow diverter stents

Angelo Carneiro · Neil Rane · Wilhelm Küker ·
Martino Cellerini · Rufus Corkill · James V. Byrne

Received: 22 August 2013 / Accepted: 15 November 2013 / Published online: 7 December 2013
© Springer-Verlag Berlin Heidelberg 2013

Abstract

Introduction This study assessed volume changes of unruptured large and giant aneurysms (greatest diameter >20 mm) after treatment with flow diverter (FD) stents.

Methods Clinical audit of the cases treated in a single institution, over a 5-year period. Demographic and clinical data were retrospectively collected from the hospital records. Aneurysm volumes were measured by manual outlining at sequential slices using computerised tomography (CT) or magnetic resonance (MR) angiography data.

Results The audit included eight patients (seven females) with eight aneurysms. Four aneurysms involved the cavernous segment of the internal carotid artery (ICA), three the supraclinoid ICA and one the basilar artery. Seven patients presented with signs and symptoms of mass effect and one with seizures. All but one aneurysm was treated with a single FD stent; six aneurysms were also coiled (either before or simultaneously with FD placement). Minimum follow-up time was 6 months (mean 20 months). At follow-up, three aneurysms decreased in size, three were unchanged and two increased. Both aneurysms that increased in size showed persistent endosaccular flow at follow-up MR; in one case, failure was attributed to suboptimal position of the stent; in the other case, it was attributed to persistence of a side branch originating from the aneurysm (similar to the endoleak phenomenon of aortic aneurysms). At follow-up, five aneurysms were completely occluded; none of these increased in volume.

Conclusion Complete occlusion of the aneurysms leads, in most cases, to its shrinkage. In cases of late aneurysm growth or regrowth, consideration should be given to possible endoleak as the cause.

Keywords Endoleak · Flow diverters · Giant aneurysms · Thrombosis

Introduction

Flow diverters (FD) are woven stents constructed with high mesh density and low porosity, sufficiently flexible to allow navigation of tortuous cranial arteries. The intention is to effectively uncouple blood flow between aneurysm sac and parent artery by redirecting blood flow along the vessel axis and reconstructing the parent vessel lumen across the aneurysm neck, thus promoting aneurysm thrombosis [1–3].

Since their introduction over 5 years ago, FDs have been used to treat larger complex aneurysms and recurrences after endosaccular coil treatments. FDs are an elegant technical solution for such aneurysms but there remain unresolved problems associated with their use. Previous studies have described unfavourable clinical outcomes including ischaemic and compression syndromes caused by thrombosis of branch and parent arteries [4–9], transient aneurysm swelling after induced sac thrombosis [10] and delayed complications including bleeding of previously unruptured aneurysms [5, 8, 11, 12]. Failure to induce complete sac thrombosis is a cause of some delayed complications and aneurysm size may be a risk factor for failures [13, 14] though some authors consider the relationship unproven [15, 16].

These concerns have been addressed in several single and multicentre audit studies, which have suggested a range of possible causes [4, 9–11, 17–20]. Other reports have concentrated on the minority of cases in which aneurysms fail to

A. Carneiro · J. V. Byrne (✉)
Oxford Neurovascular and Neuroradiology Research Unit, Nuffield
Department of Surgical Sciences, West Wing, John Radcliffe
Hospital, Headington, Oxford OX3 9DU, UK
e-mail: james.byrne@nds.ox.ac.uk

N. Rane · W. Küker · M. Cellerini · R. Corkill
Department of Neuroradiology, John Radcliffe Hospital, Oxford, UK

thrombose completely [12, 21, 22]. Amongst these, large aneurysm size and thicker aneurysm walls have been proposed as possible adverse factors [14]. In order to assess the effect of FD treatments in very large aneurysms and the possible role of the greater amount of induced thrombosis in delayed complications and failure to regress after FD treatments, a single centre audit of this subgroup of aneurysms was performed.

Material and methods

Since their introduction into our clinical practice in 2008, eight patients treated with FDs for extremely large (defined as >20 mm in maximum dimension) or giant saccular unruptured aneurysms (diameter >25 mm) were identified from the institutional record. The study was designated a clinical audit, and no ethics committee review was required.

The patients were predominantly women (seven females; one male) with a mean age of 61.7 years.

The presenting symptoms were mass effect with headache or cranial nerve palsies in seven patients and seizures in one patient. The aneurysms involved the ICA in seven patients (four cavernous and three supraclinoid portions) and the basilar artery in one patient (see Table 1).

Endovascular treatments were performed with Silk (Balt Extrusion, Montmorency, France) ($n=3$) and Pipeline (ev3 Neurovascular, Irvine, California, USA) ($n=5$) FD devices. Seven aneurysms were treated with a single FD and one with 2FDs. Two aneurysms were treated with FDs alone because of

recurrence after previous treatments by endosaccular coil embolization. In four treatments, endosaccular coils were placed in addition to FDs. All patients were treated with aspirin and clopidogrel (75 mg daily each) for a minimum of 3 months post treatment.

Aneurysms' volumes were measured on pre-treatment CT angiography (CTA) examinations, which were performed in all patients. Intravenous pump injections of Iopamidol (50 mls Niopam 370, Bracco) were monitored, and data acquisition triggered when contrast first appeared in the cervical carotid arteries to obtain maximum arterial phase filling of intracranial vessels. Images were reconstructed in the axial plane with 2-mm slice thickness, without intervals. Segmentation of the outer and inner (contrast filled) areas of each aneurysm was manually drawn from each slice. When the aneurysm was uniformly opacified with contrast, just one measurement was performed. When there was a difference between the outer and opacified contour (indicating the presence of endosaccular thrombus), differential measurements were made. By summing the areas of all the sections and factoring the thickness of the slices (2 mm), the overall and contrast filled volumes of each aneurysm was determined.

Symptomatic evolution was retrospectively evaluated from patients' hospital notes.

Follow-up imaging was by magnetic resonance imaging (MRI) and MR angiography (MRA). All the patients were first scanned at 3–6 months, and then repeated at various time points thereafter. The minimum follow-up interval was 6 months and the longest was 48 months; the mean being 20 months (see Fig. 4). During the follow-up period, digital

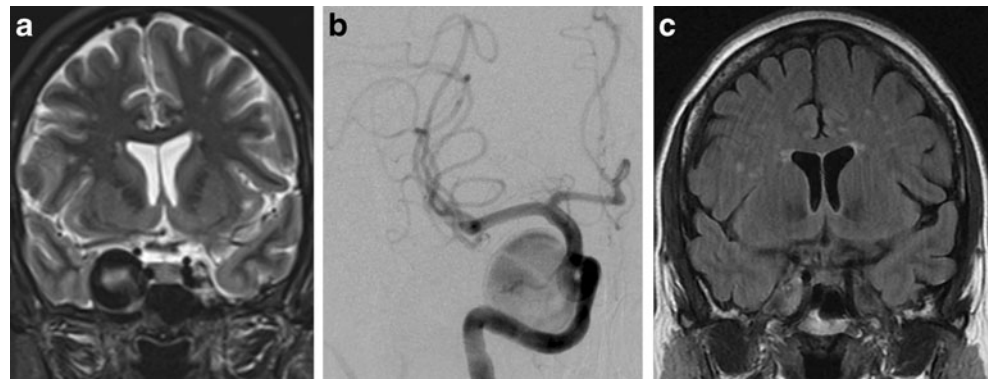
Table 1 Characteristics of patients and aneurysms

No.	Age; gender	An. location	Presentation	Pre-treat volume (mm ³)	Treatment details	3–6 months volume (mm ³)	Last FU volume (mm ³)	Clinical outcome
1	60; F	Cav ICA	Mass effect	O—6754 L—1790	Silk plus coils	O—6024 L—0	O—4088 L—0	Improved (trans. worse)
2	66; F	BA	Mass effect	O—6684 L—2568	Pipeline x2 plus coils	O—7144 L—0	O—6518 L—0	Improved (trans. worse)
3	60; F	Cav ICA	Mass effect	O/L—2628	Silk plus coils	O—3550 L—726	O—4444 L—428	Unchanged
4	46; F	Cav ICA	Mass effect	O/L—6688	Pipeline	O—2942 L—0	O—492 L—0	Cure (trans. worse)
5	69; M	Supraclin ICA	Seizures	O—15194 L—3816	Pipeline plus coils	O—15671 L—0	NA	No further seizures
6	67; F	Supraclin ICA	Mass effect	O—2924 L—2534	Silk	O—3294 L—112	O—7470 L—53	Unchanged
7 *	63; F	Supraclin ICA	Mass effect	O—5154 L—166	Pipeline	O—4380 L—0	NA	Unchanged
8 *	63; F	Cav ICA	Mass effect	O—5116 L—678	Pipeline	O—5190 L—34	NA	Unchanged

BA basilar artery, Cav cavernous, F female, L luminal volume, M male, N.A. non-applicable, O outer volume, Supraclin Supraclinoid, treat. treatment

*Aneurysm previously treated with coils

Fig. 1 a–c (patient 4). Image **a**, demonstrates a large, fully patent, right cavernous ICA aneurysm, which was treated with a Pipeline FD. DSA immediately after deployment of the stent [image **b**], showed a reduction in the contrast density inside the sac. MRI after 12 months (image **c**), the aneurysm was completely thrombosed and its dimensions had significantly reduced



subtraction angiography (DSA) was performed in five patients. Measurements of aneurysm volumes on follow-up were made on MRI/MRA performed using 3D-T1W fast scans timed to image an injected bolus of gadolinium in the arterial phase on either 1.5 or 3Tesla scanners. The segmentation method described above was used. Difference of <10 % in measured volumes was ignored.

Results

The mean size of aneurysms before FD treatment was 6390 mm³ (ranging from 2628 to 15194 mm³). Prior to any treatment, six aneurysms contained endosaccular thrombus, defined as >10 % difference between outer and luminal volumes, and two aneurysms had thin walls. The proportion

of thrombosis in the thick-walled, i.e. partially thrombosed aneurysm, varied from 13 % to 77 %, being <50 % in one case and >50 % in five cases (see Fig. 4).

At last follow-up after FD deployment, two aneurysms increased in size, three were unchanged and three decreased in volume. In terms of aneurysm location, of the four extradural (i.e. cavernous) ICA aneurysms, two decreased in size, one was unchanged and one enlarged in size. Of the four intradural aneurysms (three supraclinoid ICA and one BA), one decreased in size, two were unchanged and one increased in size.

Only two aneurysms were not treated with endosaccular coils. One of them (patient 4) resolved completely and showed the most significant size reduction at follow-up (see “Case A”). The other (patient 6) enlarged over time with some persistent luminal flow evident on follow-up angiography

Fig. 2 a–e (patient 3). Image **a** shows a large, wide-necked (arrow), cavernous ICA aneurysm, as well as an associated small supraclinoid aneurysm. During treatment with a Silk FD and coils, the stent failed to expand completely (image **b**). Follow-up DSA after 24 months (image **c**) showed the sac was still patent. Images **d** pre-treatment MRI and **e** 48-month MRI show enlargement of the aneurysm

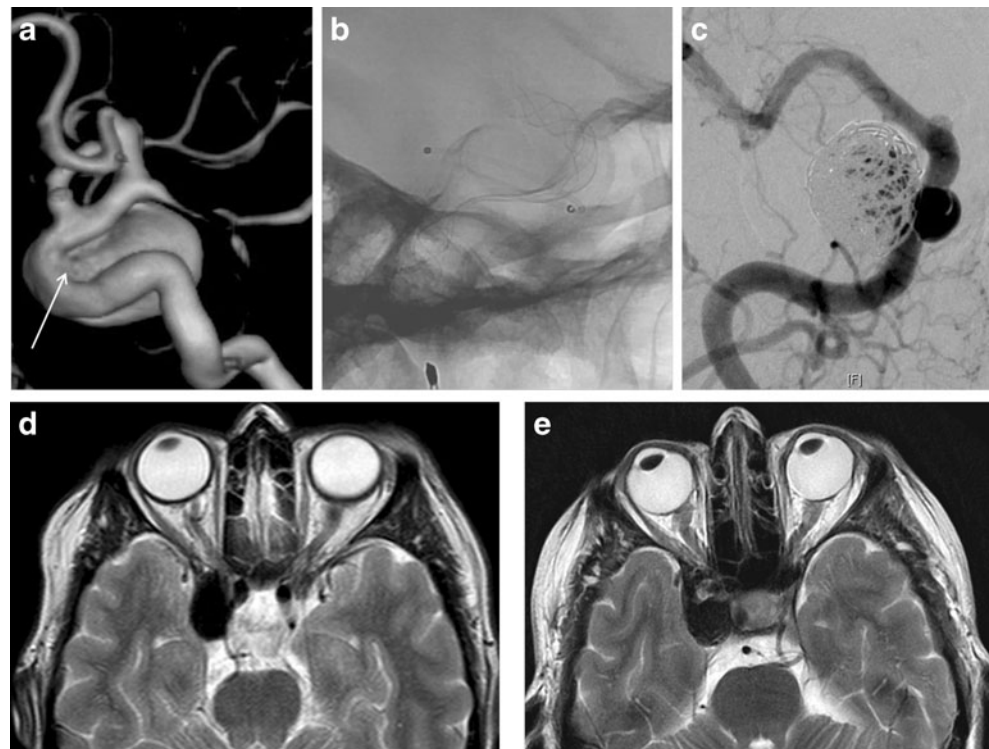
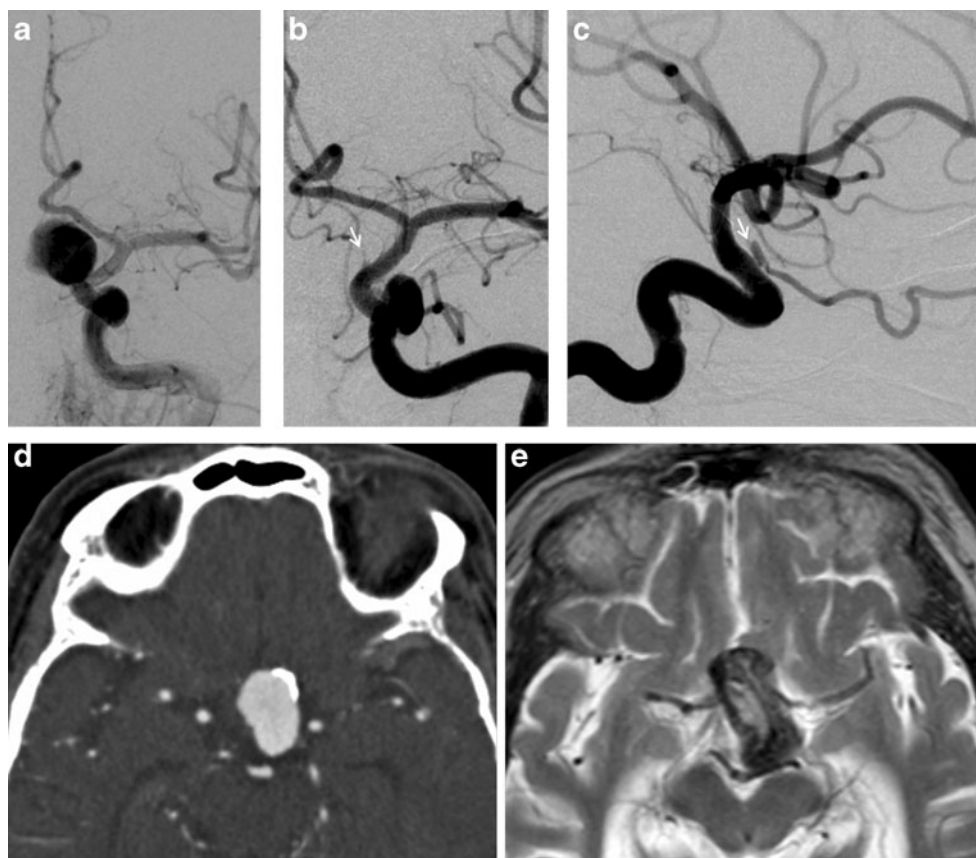


Fig. 3 a–e (patient 6). Image **a** shows a carotid-ophthalmic ICA aneurysm, immediately after deployment of a Silk FD with some saccular stasis. Images **b** and **c** are from DSA obtained 42 months later. They show the sac to be substantially occluded but a minor residuum (*arrows*), arising from the proximal portion of the patent ophthalmic artery, which originates from the aneurysm sac. Images **d** (pre-treatment CTA) and **e** (40-month follow-up MRI) show considerable aneurysm expansion, despite the substantial sac thrombosis



(see “Case C”). Six aneurysms were also treated with coils, either before or simultaneously with FD treatment. One of them (patient 3) enlarged, three were stable (patients 2, 5 and 8) and two decreased in volume (patients 1 and 7).

Of the two aneurysms that increased in size, enlargement was delayed and observed at intervals greater than 3 years after treatment. One arose from the cavernous and the other from the supraclinoid ICA in both instances, persistent endosaccular flow was evident on follow-up MRA. Persistent endosaccular flow was only evident on the last follow-up MRA in three aneurysms (patients 3, 6 and 8). Significantly, none of the five completely occluded aneurysms showed volume increases greater than 10 %.

Illustrative cases

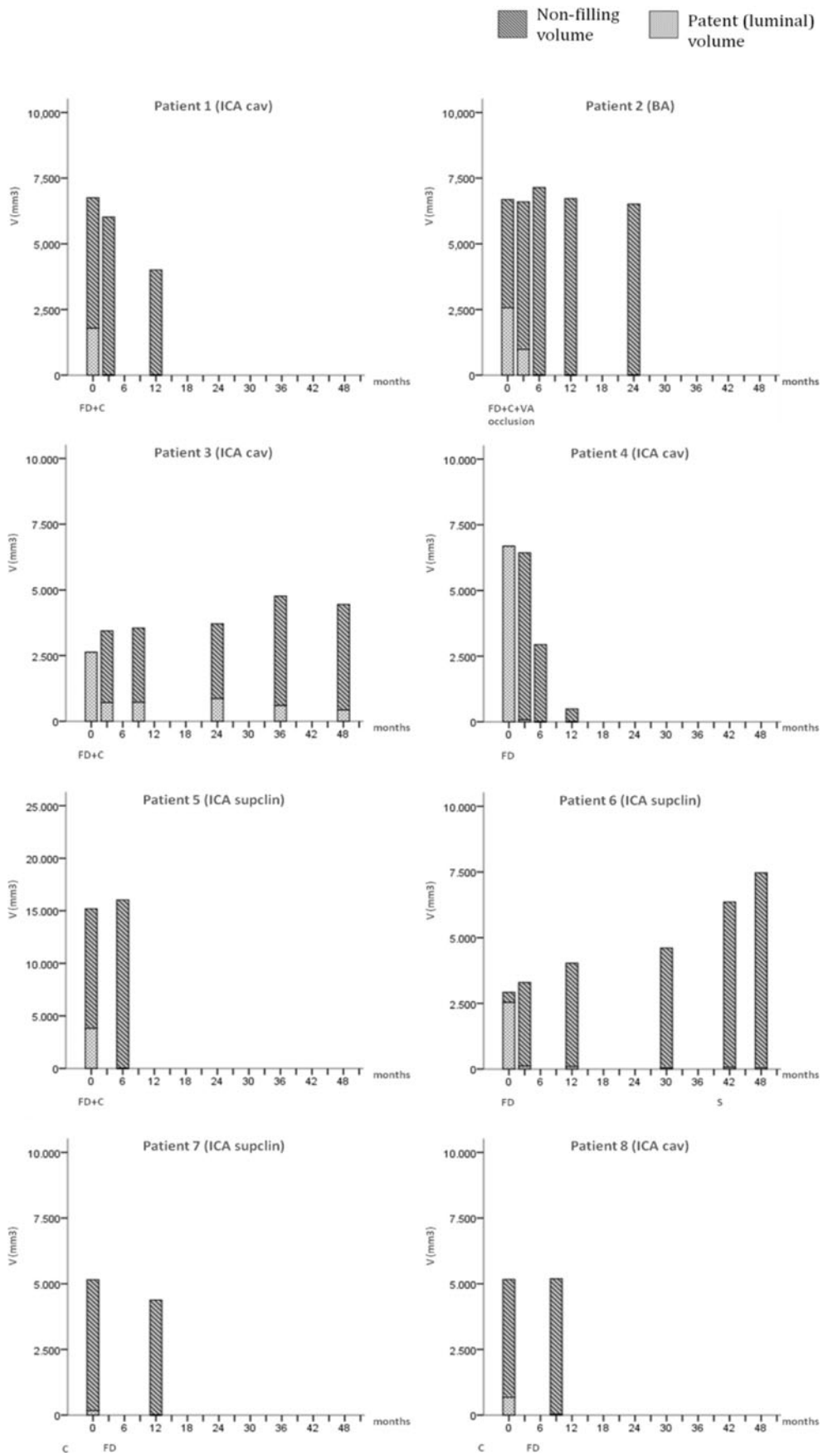
Case A (patient 4, Table 1)

This 46 year-old woman presented with a 3-month history of visual disturbance, which progressively evolved to cause continuous diplopia. She also complained of paroxysmal pain behind the right eye and in the right side of the cranium. Physical examination revealed palsy of the fourth and sixth right cranial nerves. As part of her investigations, an MRI demonstrated a

right cavernous ICA aneurysm (largest diameter 25 mm) (see Fig. 1). Endovascular treatment was performed with a single Pipeline FD (4.0×25 mm), which was deployed across the neck of the aneurysm; no endosaccular coils were used.

Three days after the procedure, the patient’s symptoms worsened with an increase in retro-ocular pain. MRI/MRA did not reveal a complication in the brain, and the ICA was patent. She was treated with corticosteroids for 4 weeks, and symptoms improved, so that after 6 months, they had completely disappeared. Follow-up MRI/MRA (performed at 12 months) showed aneurysm thrombosis and shrinkage of the sac. Subsequent DSA demonstrated no endosaccular blood flow.

Fig. 4 Volume changes on follow-up MR, before and after treatment(s). Bar heights show overall aneurysms volumes, with filling (luminal) and non-filling volumes. Time 0 corresponds to the volume prior to FD treatment. Patients 1, 2, 3 and 5 were treated with coils at the time of FD treatment (in patient 2, coiling was performed 4 weeks prior to planned FD treatment). Patients 7 and 8 were treated for recurrent aneurysms after previous endosaccular coil embolisation (i.e. before time 0) and without additional coils. Patients 3 and 4 had thin-walled aneurysms, the remainder, including patients 7 and 8, were classified thick-walled, i.e. partially thrombosed. Please note that the graph for patient 5 has a different Y scale, because the aneurysm was much bigger than the rest. Abbreviations: C coiling, FD flow diverter, S non FD stenting and as Table 1



Case B (patient 3, Table 1)

This 60 year-old woman presented with a history of several months of diplopia. Examination found partial right third cranial nerve palsy, and MRI showed a cavernous ICA aneurysm, measuring 22 mm in the largest diameter (see Fig. 2). Endovascular treatment was performed by placement of a Silk FD (4.5×20 mm) followed by endosaccular coiling. However, during deployment, the stent became stretched and its proximal segment, which was partially covering the aneurysm neck, failed to expand properly. No procedural or delayed complications occurred, and the patient's symptoms remained stable. Follow-up DSA performed at 24 months showed persistent residual filling at the aneurysm neck and within the body of coils. Subsequent MRA (performed up to 48 months) showed persistent endosaccular blood flow and fluctuations in size with an overall increase relative to the pre-treatment volume, without symptomatic worsening (see Fig. 4).

Case C (patient 6, Table 1)

A 67 year-old woman presented with visual obscuration of the left eye. She gave a history of right-sided glaucoma and was an ex-smoker. She was initially diagnosed with blepharitis, but subsequent ophthalmology review discovered a monocular left-sided visual defect, not related to glaucoma. CT demonstrated a partly calcified left carotid-ophthalmic artery aneurysm measuring 24 mm. DSA confirmed a solitary aneurysm with a large patent sac. The ophthalmic artery origin was absorbed in the aneurysm neck region (see Figs. 3 and 4).

Endovascular treatment was performed with a single Silk FD (4.5×20 mm) deployed without complication across the aneurysm neck and covering the ophthalmic artery origin. Although progressive thrombosis ensued over the following 2 years, there was associated increasing sac size and prominent residual contrast enhancement. The patient's symptoms were unchanged during this period although the findings were worrisome. DSA was performed 42 months after FD treatment and showed a small element of residual sac filling via the covered ophthalmic artery. At this stage, there was no change in the patient's vision. To prevent further enlargement, an LVIS stent (Microvention, Tustin CA) was deployed (stent-in-stent) across the origin of the ophthalmic artery without complication. The patient remains clinically stable, and follow-up continues.

Discussion

Flow diverter stents are designed to combine the properties of covered and porous stents, the former to restrict blood flow within the parent artery and the latter to maintain antegrade blood flow in branch arteries. This compromise is based on

flow studies defining an optimum stent porosity of about 70 % [2]. Their use in recent clinical trials supports this theory with reports of successful thrombosis in the majority of treated aneurysms. However, failure has been reported in a minority of aneurysms resulting in either aneurysm persistence, growth or bleeding [23]. The cause of these failures has been the subject of considerable speculation.

In this small series of extremely large aneurysms, it was surprising how few aneurysms completely collapsed after induced thrombosis and the length of time the process took. The presence of substantial prior thrombosis appears to reduce the rate of volume reduction after FD treatments, but this conclusion needs to be seen in the context of small numbers, the presence of additional coils in the majority of aneurysms and no standardised post-treatment antiplatelet regimen. Endosaccular coils tended to stabilise size changes during follow-up. Incomplete shrinkage of aneurysm sacs containing coils is not surprising, though in one such aneurysm, the volume reduced substantially. The most rapid and complete resolution occurred in an aneurysm without prior thrombus or endosaccular coils. This observation suggests, but by no means proves, that prior organised thrombus and coils are negative factors for sac regression.

It was reassuring that delayed increases in aneurysm size were associated with residual sac patency, albeit modest in patient 6. Incomplete thrombosis may be the result of a technical failure in deployment. This is illustrated by our patient 3 (illustrative case B) and reflects the plasticity of the woven design, which allows the FD to conform to the walls despite variations in lumen diameter. This feature requires the operator to size FDs carefully and to anticipate changes in deployed length and the effects of curvatures on the width and functional porosity of the deployed device. Failure of the device to deploy fully and conform to the arterial wall will negate its purpose and ability to restrict blood flow at the aneurysm neck and around the stent.

More difficult to understand are failures when the device conforms evenly to the target artery and achieves its optimum effective porosity after deployment. In these cases, several explanations have been proposed. These include variability in individual patients response to or compliance with prescribed antiplatelet drugs administered to prevent in-stent thrombosis, anomalies of local blood flow conditions, which prevent sufficient endosaccular flow reduction to induce thrombosis, and instability of thrombus within the aneurysm. No definitive regime for prophylactic antiplatelet drugs has been established, but it is generally assumed that their continued use may reduce the speed and completeness of induced thrombosis. Resistance and individual variations in the effects of properly administered drugs is likely to be a factor in some failures; otherwise, a standard dose regime would probably have been established by now. Blood flow into the aneurysm is dictated by various factors, of which the anatomy of parent

artery, neck and branch arteries are important. Computational flow dynamic studies have identified patterns, dictated by the local anatomy that predicted poor outcomes of FD treatments [14, 24, 25]. Though patterns of flow interruption after FD deployment correlate with failures of induced thrombosis, this effect is likely to be only relevant at deployment or soon afterwards since any substantial thrombosis will change the anatomic conditions. So pre-treatment haemodynamic patterns are probably not as important in delayed aneurysm growth or bleeding as the absence of a complete “healed” endothelium. Lack of such a barrier at the aneurysm neck is the probable reason for late instability of the induced thrombosis. The exact mechanism remains undefined, and combinations of haemodynamic and biological factors including an exchange of endogenous thrombolytic agents have been proposed to explain the phenomenon.

Transient increases in sac size after induced thrombosis are well recognised [23]. This is probably part of the normal evolution of the blood clot and more likely to cause symptoms in aneurysms at eloquent locations, e.g. internal carotid aneurysms causing ophthalmoplegia and visual loss due to cranial nerve compression. Subsequent total resolution of mass effect may also depend on location, e.g. more likely to be complete for extra-dural aneurysms, and on the chronicity of mural thickening and calcification prior to treatment. However, relief of symptoms does not depend on complete aneurysm collapse, since combining FD treatment with endosaccular coils is effective. The issues raised by the small proportion of aneurysms that do not resolve in the medium to long term are their continued risk of bleeding and growing with worsening local compression.

To this controversy, we would like to introduce the endoleak concept because of similarities in the situation in our illustrative case C (patient 6) with a carotid-ophthalmic artery aneurysm and that reported for abdominal aortic aneurysms. The term endoleak was first used in the context of endovascular aortic replacements (EVAR). The use of stent grafts within the aorta can lead to reversed flow within segmental aortic collaterals and delayed enlargement of the sac due to secondary endoleak [26]. Recurrences of carotid-ophthalmic artery aneurysms after proximal carotid artery occlusion or stenting can occur because of flow reversal in the carotid-ophthalmic artery, similar to that described in lumbar collateral arteries. In this situation and after stenting for vertebrobasilar aneurysms, preventative occlusions of the ophthalmic artery or the contralateral vertebral artery, respectively, are recommended to prevent acute and delayed aneurysm enlargement. However, in our patient, no flow reversal in the branch artery was demonstrated and the endoleak arose from patency of the branch artery. This we attribute to the incorporation of the proximal portion of the carotid-ophthalmic artery in the aneurysm sac and continued antegrade blood flow preventing uncoupling of flow between

parent artery and aneurysm sac. It is similar to that illustrated by Klisch et al. [12] in a fusiform basilar artery aneurysm. This mechanism would explain the observed increase in sac thrombosis over time and the need to consider strategies such as telescopic stenting or preventative branch artery occlusion when using FDs since retreatment of aneurysms complicated by this type of endoleak poses difficulties. There are reports of successful retreatment with FDs with a stent-in-stent configuration [12], and here, we describe retreatment with an LVIS braided stent, which is regarded as having intermediate flow diverting properties.

Conflict of interest JVB's Department provides independent image reading services to Balt International under an institutional contract.

References

1. Augsburger L, Farhat M, Reymond P et al (2009) Effect of flow diverter porosity on intraaneurysmal blood flow. *Klin Neuroradiol* 19(3):204–214
2. Byrne JV, Szikora I (2012) Flow diverters in the management of intracranial aneurysms: a review. *EJMINT* 2012:1225000057 ejmint.org
3. Szikora I, Nelson PK, Berentei Z et al (2008) The potential of flow modification in the treatment of intracranial aneurysms. *Interv Neuroradiol* 14(Suppl 1):77–80
4. Szikora I, Berentei Z, Kulcsar Z et al (2010) Treatment of intracranial aneurysms by functional reconstruction of the parent artery: the Budapest experience with the pipeline embolization device. *AJNR Am J Neuroradiol* 31(6):1139–1147
5. Byrne JV, Beltechi R, Yamold JA et al (2010) Early experience in the treatment of intra-cranial aneurysms by endovascular flow diversion: a multicentre prospective study. *PLoS One* 5(9)
6. Lubicz B, Collignon L, Raphaeli G et al (2010) Flow-diverter stent for the endovascular treatment of intracranial aneurysms: a prospective study in 29 patients with 34 aneurysms. *Stroke* 41(10):2247–2253
7. Wagner A, Cortsen M, Hauberberg J et al (2012) Treatment of intracranial aneurysms. Reconstruction of the parent artery with flow-diverting (Silk) stent. *Neuroradiology* 54(7):709–718
8. Nelson PK, Lylyk P, Szikora I et al (2011) The pipeline embolization device for the intracranial treatment of aneurysms trial. *AJNR Am J Neuroradiol* 32(1):34–40
9. Berge J, Biondi A, Machi P et al (2012) Flow-diverter silk stent for the treatment of intracranial aneurysms: 1-year follow-up in a multicenter study. *AJNR Am J Neuroradiol* 33(6):1150–1155
10. Lylyk P, Miranda C, Ceratto R et al (2009) Curative endovascular reconstruction of cerebral aneurysms with the pipeline embolization device: the Buenos Aires experience. *Neurosurgery* 64(4):632–642, discussion 642–3; quiz N6
11. Beckske T, Kallmes DF, Saatci I et al (2013) Pipeline for uncoilable or failed aneurysms: results from a multicenter clinical trial. *Radiology* 267(3):858–868
12. Klisch J, Turk A, Turner R et al (2011) Very late thrombosis of flow-diverting constructs after the treatment of large fusiform posterior circulation aneurysms. *AJNR Am J Neuroradiol* 32(4):627–632
13. Briganti F, Napoli M, Tortora F et al (2012) Italian multicenter experience with flow-diverter devices for intracranial unruptured

- aneurysm treatment with periprocedural complications—a retrospective data analysis. *Neuroradiology* 54(10):1145–1152
14. Larrabide I, Aguilar ML, Morales HG et al (2013) Intra-aneurysmal pressure and flow changes induced by flow diverters: relation to aneurysm size and shape. *AJNR Am J Neuroradiol* 34(4):816–822
 15. Saatci I, Yavuz K, Ozer C et al (2012) Treatment of intracranial aneurysms using the pipeline flow-diverter embolization device: a single-center experience with long-term follow-up results. *AJNR Am J Neuroradiol* 33(8):1436–1446
 16. Leonardi M, Cirillo L, Toni F et al (2011) Treatment of intracranial aneurysms using flow-diverting silk stents (BALT): a single centre experience. *Interv Neuroradiol* 17(3):306–315
 17. Pierot L (2011) Flow diverter stents in the treatment of intracranial aneurysms: where are we? *J Neuroradiol* 38(1):40–46
 18. Maimon S, Gonen L, Nossek E et al (2012) Treatment of intra-cranial aneurysms with the SILK flow diverter: 2 years' experience with 28 patients at a single center. *Acta Neurochir (Wien)* 154(6):979–987
 19. Fischer S, Vajda Z, Perez MA et al (2012) Pipeline embolization device (PED) for neurovascular reconstruction: initial experience in the treatment of 101 intracranial aneurysms and dissections. *Neuroradiology* 54(4):369–382
 20. Yu SC, Kwok CK, Cheng PW et al (2012) Intracranial aneurysms: midterm outcome of pipeline embolization device—a prospective study in 143 patients with 178 aneurysms. *Radiology* 265(3):893–901
 21. Turowski B, Macht S, Kulcsar Z et al (2011) Early fatal hemorrhage after endovascular cerebral aneurysm treatment with a flow diverter (SILK-Stent): do we need to rethink our concepts? *Neuroradiology* 53(1):37–41
 22. Kulcsar Z, Houdalt E, Bonafe A et al (2011) Intra-aneurysmal thrombosis as a possible cause of delayed aneurysm rupture after flow-diversion treatment. *AJNR Am J Neuroradiol* 32(1):20–25
 23. Szikora I, Marosfői M, Salomváry B et al (2013) Resolution of mass effect and compression symptoms following endoluminal flow diversion for the treatment of intracranial aneurysms. *AJNR Am J Neuroradiol* 34(5):935–939
 24. Cebral JR, Mut F, Raschi M et al (2011) Aneurysm rupture following treatment with flow-diverting stents: computational hemodynamics analysis of treatment. *AJNR Am J Neuroradiol* 32(1):27–33
 25. Kulcsar Z, Augsburger L, Reymond P et al (2012) Flow diversion treatment: intra-aneurysmal blood flow velocity and WSS reduction are parameters to predict aneurysm thrombosis. *Acta Neurochir (Wien)* 154(10):1827–1834
 26. White GH, Yu W, May J et al (1997) Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysm: classification, incidence, diagnosis, and management. *J Endovasc Surg* 4:152–168