CASE REPORT

Pipeline embolization device induced collateral channels in elective flow diversion treatment

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SUMMARY

The pipeline embolization device (PED) is a well recognized treatment for intracranial aneurysms. However, uncertainty remains regarding its effects on flow alteration, which is particularly highlighted by persistently perfused aneurysmal remnants and non-regressing, non-perfused aneurysmal masses. Here we present a 68-year-old woman with an incidental giant fusiform right paraophthalmic aneurysm electively treated with a PED. After lowering her antiplatelet therapy to promote aneurysm thrombosis, she was found to have a progressively enlarging perfused aneurysmal remnant. Angiography revealed PED occlusion, but curiously the development of a peri-construct collateral channel which feeds the aneurysmal remnant, and gives rise to distal branches and contributes to middle cerebral artery flow. The large 'thrombosed' aneurysmal mass showed tiny internal vessels on cone beam CT angiography as well as florid enhancement on MRI, further confirming that apparently thrombosed remnants are biologically active and may be remodeled depending on flow demand.

BACKGROUND

The pipeline embolization device (PED; Medtronic, Minneapolis, Minnesota, USA) has become an accepted option for the treatment of complex intracranial aneurysms.^{1 2} The PED bridges the aneurysm neck, inducing intra-aneurysmal flow stagnation, thrombosis, and eventual occlusion.³ However, reports of non-efficacy and complications have since emerged, including persistent aneurysm filling, in-stent stenosis, and delayed rupture.⁴⁻⁶ We previously reported, to our knowledge, the first case of delayed recanalization of a 'thrombosed' aneurysmal remnant after acute treatment of a ruptured dissecting anterior cerebral artery aneurysm.⁷ Here we present a second case describing a similar phenomenon but in an electively treated aneurysm with clearer pathophysiologic delineation, which we hope will provide greater clarity of this enigmatic process.

CASE PRESENTATION

A 68-year-old right-handed female hypertensive smoker presented with a 3 month history of frontal headaches. On CT angiogram, a partially thrombosed giant fusiform right paraophthalmic aneurysm causing indentation of the adjacent frontotemporal lobe was discovered, as well as a second smaller wide necked left anterior communicating artery aneurysm (figure 1). After loading with aspirin 650 mg and clopidogrel 600 mg, the

right paraophthalmic aneurysm was treated with flow diversion using a single 5×20 mm PED. The entire PED was well opposed but was narrowed distal to the aneurysmal neck due to the aneurysmal mass effect. Post-deployment angiogram demonstrated immediate stasis within the aneurysm sac (figure 1). Postoperative day 1 CT of the head showed a small asymptomatic subarachnoid hemorrhage within the right Sylvian fissure but no other complications. The patient was discharged in a stable condition on daily aspirin 325 mg and clopidogrel 75 mg.

The patient remained asymptomatic during subsequent follow-up. Multiple follow-up CT and MR angiograms showed a mild decrease in overall aneurysm volume, but persistent and slowly enlarging interstitial filling into an aneurysm neck remnant (figure 2). This occurred despite reductions in antiplatelet therapy—clopidogrel was stopped at 5 months post-treatment with aspirin continued daily at 81 mg, and the aspirin dose was further decreased to 81 mg every other day at 10 months in an effort to promote thrombosis within the remnant. CT angiography at 13 months post-treatment showed progressive reduction in the caliber of the right cervical internal cerebral artery, concerning for in-stent stenosis limiting outflow.

A catheter angiogram was performed 15 months post-treatment on a Siemens Axiom Artis Biplane angiography unit (Siemens Healthcare, Erlangen, Germany) for further evaluation. This showed occlusion of the narrowed distal portion of the PED, at the level of the perfused aneurysmal remnant seen on the previous CT angiograms. High frame rate oblique planar angiographic images and three-dimensional reconstructions of cone beam CT angiography (figure 3) delineated a single large peri-construct collateral channel arising through the struts of the PED below the angiographic clinoid ring, which gives rise to the right ophthalmic artery, and then continues cranially to feed the dorsal aneurysmal neck remnant. The channel then spirals around the outside of the occluded PED, giving rise to the anterior choroidal artery then connecting to the right M1 segment contributing to flow in the right middle cerebral artery. Cone beam CT angiography reconstructions additionally gave the impression of tiny branch vessels within the thrombosed part of the aneurysm sac, with indeterminate origins.

The patient underwent a MRI vessel wall study following the catheter angiogram, which showed prominent enhancement of the 'thrombosed' part of the aneurysm (figure 4).

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Figure 1 Pre (A–D) and post (E–H) pipeline embolization device images of the giant right paraophthalmic aneurysm, including axial non-contrast CT of the head (A, F), axial (B, G), and coronal MIP (C, H) CT angiogram, and oblique (D, E) subtracted angiogram. There is a second left anterior communicating artery aneurysm which was not treated (C, H). Immediate post-deployment angiogram (E) and subsequent CT angiogram on postoperative day 1 (F, G) demonstrated immediate stasis within the aneurysm, with preserved distal middle cerebral artery filling. The distal end of the stent was tapered (E) due to the narrow caliber of the parent terminal internal cerebral artery from adjacent aneurysm compression (D, arrow). Postoperative day 1 CT of the head (F) showed a small amount of subarachnoid hemorrhage in the right Sylvian fissure.

Given that the patient was asymptomatic, treatment was deferred and a repeat angiogram was planned in 6 months. Aspirin dose was increased to 81 mg daily to prevent thrombosis of the peri-construct channel.

DISCUSSION

We have previously reported a case of collateral channel formation around a thrombosed PED post-treatment of a ruptured anterior cerebral artery aneurysm.⁷ Our current case of peri-construct collateral channel formation in the post-treatment of

an unruptured internal cerebral artery aneurysm illustrates that the phenomenon is not unique to ruptured aneurysms.

This case demonstrates a single peri-construct channel, which does not have the typical appearance of hypertrophied vasa vasorum due to its smooth nature. This channel has likely been formed or promoted by distal flow demand from the ophthalmic, anterior choroidal, and middle cerebral arteries. Flow demand through the ophthalmic artery has previously been shown to cause lower rates of complete obliteration with PED in the treatment of paraophthalmic and ophthalmic aneurysms.⁸

Figure 2 Subsequent follow-up CT angiograms from 7 months (A), 10 months (B), and 13 months (C, D) post-treatment showed progressively enlarging filling into an aneurysm remnant along the anteromedial aspect of the stent (arrows). Oblique MIP CT angiogram (D) illustrates the level of the aneurysm remnant relative to the pipeline embolization device. There remained stable narrowing of the distal stent.

Figure 3 Magnified oblique subtracted angiograms (A, B) and dual volume cone beam CT angiogram (C) images show occlusion of the narrowed distal portion of the pipeline construct, at the level of the perfused aneurysmal remnant. There is a single large peri-construct collateral channel arising through the struts of the pipeline embolization device (PED) below the angiographic clinoid ring, which gives rise to the right ophthalmic artery, and then continues cranially to feed the dorsal aneurysmal remnant. From this point, the channel spirals around the outside of the occluded PED, gives rise to the anterior choroidal artery, and connects to the right M1 segment contributing to flow in the right middle cerebral artery. Videos delineating the course of the peri-construct channel are available online (see online supplementary material).

Figure 4 Axial T2 weighted (A) and axial T1 weighted (B) MRI images show a T1 intermediate, T2 hyperintense right paraophthalmic aneurysm mass. Axial (C) and coronal (D) T1 weighted post-gadolinium contrast images show prominent enhancement within the aneurysm mass.

Reduction of antiplatelet therapy to encourage aneurysm remnant closure may have promoted PED luminal thrombosis, especially in the presence of distal construct stenosis, thus elevating pressure within the aneurysmal remnant and causing subsequent enlargement of the peri-aneurysmal channel. The relatively large size of the peri-construct channel suggests that it may represent the expansion of a flow channel, proximally through intra-aneurysmal ‘thrombus’ and distally in part due to re-expansion of the native vessel outside the PED. This expansion may have also occurred in part due to reduced external compression as the adjacent aneurysm regressed in size.

The tiny branch vessels seen on cone beam CT angiography and the intra-aneurysmal enhancement on MRI in this patient’s thrombosed aneurysmal remnant are also much more evident compared with our previous case, and provide further evidence of florid intra-aneurysmal biological activity. A histological study on thrombosed giant fusiform aneurysms by Szikora *et al*⁹ demonstrated the lack of thrombus organization (ie, no smooth muscle cell invasion or connective tissue formation within the thrombus) inside aneurysms up to 13 months post-flow diversion, despite non-filling on angiography. Previous articles have theorized that this non-organized, unstable thrombus may induce degeneration of the aneurysm wall via release of thrombocyte derived growth factors, peroxidases, and matrix metalloproteinases, leading to aneurysm rupture post-flow diversion.^{6 9 10} It is not known whether a similar biochemical process can induce channel formation within an active thrombus in our case. However, the MRI enhancement in this case suggests that the lumen of the treated aneurysm contains more than non-organized thrombus, which should not enhance so avidly. More robust histological data are needed to help us understand the thrombotic process post-flow diversion.

Learning points

- ▶ Multiple previous studies have suggested non-perfused aneurysmal remnant can be biologically active. This case of a single peri-construct collateral channel arising through an occluded pipeline embolization device (PED) highlights this phenomenon, and show that new peri-construct channels can form through previously thrombosed aneurysm as a response to flow demand.
- ▶ This case also confirms that the phenomenon is not isolated to acutely treated aneurysms.
- ▶ Antiplatelet reduction can induce enlargement of these remnants. As such we suggest a more considered approach to antiplatelet cessation as a method of promoting aneurysmal thrombosis, especially in the case of small PED devices and constructs with stenoses.
- ▶ If a persistent perfused aneurysmal remnant is encountered, an up to date catheter angiogram must be performed prior to antiplatelet therapy reduction, as CT and MR angiograms are limited in their ability to detect these peri-construct channels and luminal occlusion.
- ▶ The rupture risk of these channels and remnants through an aneurysm which is mostly ‘thrombosed’ is not yet known. Further analysis of this phenomenon is required.

Contributors AHYC conceived the paper and curated the images. YAC and BJD drafted the article. All authors revised the paper for content and gave final approval for the version to be published.

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