CASE REPORT

Intermittent balloon occlusion to favor nidal penetration during embolization of arteriovenous malformations: a technique modification

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SUMMARY

Endovascular embolization of brain arteriovenous malformations (AVMs) has improved with liquid occlusive agents, but flow-related and anatomic restrictions limit endovascular capabilities. AVM compartments supplied by leptomeningeal networks and feeding arteries too small and/or tortuous for safe catheterization are rarely penetrated by liquid occlusive agents. A case with both impediments prompted a novel solution. A balloon was inflated across the supply to a lenticulostriate feeder, thereby favoring penetration of the liquid occlusive agent, injected from a different feeding territory, back into the AVM compartment supplied by the temporarily occluded feeder. This technique may reduce the number of embolization stages in large high-flow AVMs and increase the likelihood of achieving complete occlusion. This technique is highly complex and requires meticulous monitoring of multiple events.

BACKGROUND

A recent case with both anatomic and flow-related impediments to embolization prompted a novel solution. A balloon was inflated across the supply to a lenticulostriate feeder and to the leptomeningeal network beyond, thereby favoring penetration of the liquid occlusive agent, injected into a different feeding vascular territory, back into the AVM compartment supplied by the feeders temporarily occluded by that balloon.

CASE PRESENTATION

A 20-year-old normotensive woman presented with occipital headache, neck pain and inability to turn her head while driving.

INVESTIGATIONS

Initial brain CT showed a 'right frontal mass' and she was transferred to our institution for further evaluation and treatment. CT angiography and MR imaging/angiography showed a 44×36×50 mm (H×W×AP) Spetzler–Martin grade 3 right frontal cingulate AVM with a 17 mm frontal varix faintly calcified at its rim. There was no evidence that the AVM had ruptured, either remotely or acutely.

Cerebral angiography showed that the high-flow AVM was supplied primarily by branches of the right anterior cerebral artery (ACA). Additionally, well-established retrograde flow in the right ACA branches distal to the AVM nidus were fed by a leptomeningeal network supplied by distal branches of the right middle cerebral artery (MCA) and right

posterior cerebral artery. Further supply arose from a lateral striate branch of the M1 right MCA, the recurrent artery of Heubner, the marginal tentorial branch of the right meningohipophyseal trunk, dural branches of the right vertebral artery and falcine branches of both middle meningeal arteries (MMA). The 17 mm peripherally-calcified frontal varix drained to the inferior sagittal sinus and internal cerebral vein. Cortical venous congestion was striking in pathways to the superior sagittal sinus.

TREATMENT

Staged preoperative embolization with Onyx (ethylene-vinyl alcohol copolymer; ev3, Covidien Vascular Therapies, Irvine, California, USA) was requested (figure 1).

Embolization was initiated at the posterior aspect of the AVM and progressed anteriorly. During stage 1 embolization, 3.4 mL of Onyx was injected through a pericallosal branch of the right ACA. Subsequent embolizations were conducted after intervals of 2, 10, 4, 4 and 4 weeks, the timing of which was adjusted to avoid conflict with the patient's academic schedule. A total of 20.0 mL Onyx (6.4 mL Onyx-34 and 13.6 mL Onyx-18) was injected through six branches over the course of the first five stages of embolization. Treatment through the right MMA feeder did not reach the falcine branch that contributed to the nidus, isolating this supply to the left MMA feeder.

After completion of the first five stages of embolization the varix and the deep venous drainage had been obliterated and the cortical venous congestion had cleared. The remaining feeders included the falcine branch of the left MMA, the lateral striate branch of the M1 right MCA and four branches of the right ACA distal to the AVM nidus that were supplied by the leptomeningeal network fed by distal branches of the right MCA (figure 2). The only remaining feeder that appeared to be susceptible to catheterization was the left MMA.

During stage 6 embolization the left MMA was catheterized and, when satisfactory positioning of the microcatheter was achieved, a 4.0×10 mm HyperGlide (Covidien Vascular Therapies) balloon was then advanced into the M1 right MCA. Its ability to occlude the origin of the lateral striate branch and flow to the leptomeningeal network was verified fluoroscopically and the balloon was then deflated.

To cite: Bank WO, Stemer AB, Armonda RA, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-010637

Figure 1 (A–D) Cerebral angiogram demonstrating supply to the high-flow arteriovenous malformation. (A) Right internal carotid artery (ICA) injection with filling through right anterior cerebral artery branches, a right lateral striate branch (from M1 right middle cerebral artery (MCA)) and retrograde filling from the leptomeningeal network supplied by right MCA branches. (B) Left ICA injection with supply from the recurrent artery of Heubner. (C, D) Right and left maxillary artery injections, respectively, with filling through the falcine branch of each middle meningeal artery. (E) CT angiogram (coronal image) showing the 44×36×50 mm (H×W×AP), Spetzler–Martin grade 3 right frontal cingulate AVM.

When Onyx-34 started to descend the falcine branch, Onyx-18 was substituted to promote distal penetration of the lower viscosity Onyx. As the Onyx started to fill the top of the AVM nidus, the balloon was inflated intermittently in the right MCA in the hope of ‘drawing’ the Onyx deep into the residual nidus to occlude the right lenticulostriate feeder and the right ACA branches distal to the residual nidus. During the injection of 6.7 mL Onyx-18 the balloon was inflated four times (3.5, 2.5, 2.5 and 1.75 min over the course of 22 min) with deflation intervals of 2.5, 4.5 and 5.75 min between inflations. Onyx injection was continued during deflation intervals, filling out more proximal areas of the residual nidus. Intermittent balloon inflation was stopped when retrograde filling of the distal right striate branch was achieved, and embolization was discontinued as filling of the right ACA branches distal to the AVM became

evident. Total injection time was 50 min. The volume of the total Onyx cast was 27.2 mL (6.9 mL Onyx-34 and 20.3 mL Onyx-18) (figures 3 and 4).

OUTCOME AND FOLLOW-UP

Immediate follow-up angiography confirmed occlusion of the recurrent artery of Heubner, the lateral striate branch of the right MCA and two of the four remaining ACA feeders that filled from the leptomeningeal network. No abnormal stasis was evident.

One month after embolization the preoperative angiogram confirmed durable obliteration of the striate and Heubner supply. No antegrade ACA supply remained proximal to the AVM nidus, and only two ACA feeders distal to the nidus continued to supply a small superior compartment of the AVM.

Figure 2 After stage 5 embolization: 20.0 mL Onyx. (A) Unsubtracted image of the Onyx cast. (B–D) Right internal carotid artery injection in early, mid and late arterial stages showing persistent filling of the arteriovenous malformation through the right lateral striate branch (from M1 right middle cerebral artery (MCA)) and the leptomeningeal network filling distal to the nidus through distal right MCA branches.

Figure 3 (A, C) Unsubtracted lateral and frontal images of the Onyx cast showing the position of the Marathon microcatheter in the left middle meningeal arteries (MMA) from which Onyx was injected (black arrows). (B, D) Lateral and frontal images of the left MMA injection showing filling of the superior aspect of the arteriovenous malformation (before stage 6 embolization).

The leptomeningeal network that supplied those feeders was significantly diminished in prominence. The residual superolateral component of the AVM was resected the next day in 4.5 h with 150 mL estimated blood loss. No residual arteriovenous shunting was present on postoperative angiography and the patient made a complete recovery.

DISCUSSION

Since Kerber's calibrated leak microcatheter balloon was first manipulated past the carotid siphon and provided reliable access to intracerebral AVM feeders,¹ neurointerventionists have achieved steady progress in attempting to impregnate the nidus of AVMs completely with embolic material.^{2,3}

Large high-flow AVMs remain difficult. Embolization with liquid occlusive agents will occasionally penetrate the AVM nidus from treatable feeders and occlude compartments supplied by inaccessible sources (specifically, branches that are too small and/or too tortuous to adequately catheterize or branches distal to the AVM nidus that are supplied by leptomeningeal networks through normal brain tissue).^{4,5} The frequency with which supply from these compartments persists after endovascular treatment, however, has necessitated the frequent combination of endovascular treatment with gamma knife irradiation,

resection and/or intraoperative embolization of feeding arteries distal to the AVM nidus.⁶

Various technique modifications attempt to overcome these challenges, including dual arterial catheters (through which Onyx is injected) and dual-lumen microcatheter balloons.^{7,8} An isolated case report from 1989 describes temporary balloon occlusion during AVM embolization, but our case report is the first to use Onyx and intermittent temporary balloon occlusion to 'draw' flow into inaccessible AVM compartments.⁹

Inspiration came out of desperation: we had embolized the AVM five times but left the one feeder that would not be readily accessible during resection—that is, the lateral striate branch of the right MCA. One possibly accessible feeder remained, but entered the top of the residual AVM—namely, the falcine branch of the distal left MMA. The technique described takes advantage of intermittent temporary occlusion of the arterial supply to untreatable feeders to favor penetration of the embolic material into the nidal territory of the AVM directly subserved by those feeders. Our goal was achieved. Had we used this technique earlier it is conceivable that fewer stages would have led to complete obliteration with no need for resection.

Certain precautionary comments are important. Temporary occlusion of intracranial arteries that supply normal brain

Figure 4 (A) Right internal carotid artery injection (ICA) showing the origin of the right lateral striate branch over which the balloon was inflated. (B) During Onyx embolization: Onyx-18 filling from the left middle meningeal arteries to the superior compartment of the arteriovenous malformation (AVM) nidus while the 4x10 HyperGlide balloon is inflated in the M1 right middle cerebral artery (MCA). (C) Onyx penetration into areas of the AVM nidus supplied by the lateral striate branch of the right MCA. (D) Right ICA injection after stage 6 embolization showing occlusion of the portion of the AVM nidus supplied by the lateral striate branch.

should follow the guidelines worked out by vascular neurosurgeons who clip intracranial aneurysms (ie, <5 min for each occlusion with 5 min intervals between each occlusion and no more than four occlusive episodes).¹⁰ When the need for temporary occlusion of normal territories is anticipated, arrangements should be made for burst suppression and brain monitoring of those territories. This can be done with little degradation of image quality, especially when planned in advance. (In the reported case, no arrangements had been made for neuromonitoring or burst suppression; the intended 5 min occlusion intervals were adjusted to the achieved penetration of liquid occlusive agents towards the targeted period). The timing of each occlusion period and the deflation interval between each episode of occlusion must be accurate; this complicates the procedure. Rinsing of two guiding catheters in different vascular territories and two microcatheter systems, each serving distinctly different functions, further complicates the procedure. Close attention must be paid during retrograde penetration into feeding pedicles at the edge of the nidus to avoid occlusion of normal branches proximal to the AVM nidus. This is not a technique to be attempted by an inexperienced team or one that only occasionally treats AVMs.

Learning points

- ▶ Intermittent temporary occlusion of the arterial supply to untreatable AVM feeders can favor penetration of Onyx into the nidal territory of the AVM directly subserved by those feeders.
- ▶ Early use of this technique during staged embolisations of large high-flow AVMs may reduce the number of embolization stages and may increase the likelihood of achieving complete occlusion of the AVM.
- ▶ This technique, however, is highly complex and requires meticulous monitoring of multiple events, some of which occur simultaneously. It is not one to be attempted by a team that only occasionally treats AVMs.

Contributors All authors contributed substantially to the treatment of this patient as well as the composition and review of the manuscript.

Competing interests None.

Patient consent Obtained.

Ethics approval Institutional Review Board.

Provenance and peer review Not commissioned; externally peer reviewed.

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