Hong Kong J Radiol. 2019;22:56-63 | https://doi.org/10.12809/hkjr1916900

ORIGINAL ARTICLE

Transvenous Embolisation of Dural Arteriovenous Fistulas with Combination of Guglielmi Detachable Coils and Onyx: Preliminary Experience and Evaluation of the Clinical Outcomes

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ABSTRACT

Introduction: *The current mainstay treatment strategy for dural arteriovenous fistulas (DAVFs) is endovascular therapy. The use of transarterial Onyx 18 for treatment of DAVFs has been established, but there is limited literature on transvenous embolisation of DAVFs using Onyx 18 and Guglielmi detachable coils (GDCs). We herein present our preliminary experience of combined use of Onyx 18 and GDCs in treatment of DAVFs using a transvenous approach. Endovascular techniques, clinical and angiographic outcomes, and complications are discussed. We aimed to share our experiences to provide a foundation for future studies to improve patient care.*

Methods: *We retrospectively analysed all patients with DAVF (n=5, age 23-60 years) with endovascular treatment using Onyx 18 and GDCs performed in the same session, who were treated in our institution between 2014 and 2015. The double-catheter technique with transvenous approach was performed in all five cases. Treatment response and complications were evaluated clinically. We assessed the treatment outcomes with digital subtraction angiogram at 6 months and 18 months after embolisation, assessing the degree of residual arteriovenous shunting and presence of cortical venous reflux.*

Results: *Among the five reviewed cases, all achieved symptom alleviation. In two (40%) cases of DAVFs complete obliteration was achieved in the first session of embolisation; in one case significant reduction of arteriovenous shunting was achieved. In two (40%) cases, significant reduction of flow into DAVF was achieved after two separate sessions of embolisation. There were no reported cases of new neurological deficits after the procedures.*

Conclusion: *Onyx 18 in combination with GDCs using transvenous approach for DAVF treatment is a safe and feasible method, with a reasonably high success rate in a small sample. As DAVF is a spectrum of diseases with different severities and locations, treatment approaches should be highly individualised and a multidisciplinary approach should be adopted.*

Key Words: *Central nervous system vascular malformations; Dimethyl sulfoxide*

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Submitted: 14 Jun 2017; Accepted: 28 Sep 2017.

Disclosure of Conflicts of Interest: All authors have disclosed no conflicts of interest.

Funding/Support: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval: Requirement for patient consent was waived by the ethics board.

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中文摘要

結合Guglielmi分離式線圈和Onyx液體栓塞劑進行硬腦膜動靜脈瘺的經靜脈栓塞：臨床結果的初步經驗和評估

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引言：目前硬腦膜動靜脈瘺（DAVFs）的主要治療策略是血管內治療。雖然目前可使用經動脈Onyx 18液體栓塞劑治療DAVF，但有關結合Onyx 18和Guglielmi分離式線圈（GDC）進行DAVF的經靜脈栓塞的文獻有限。本文分享我們結合Onyx 18和GDCs進行經靜脈DAVF的初步經驗，並討論血管內技術、臨床和血管造影結果以及併發症。作者希望透過分享經驗，促使未來研究能改善患者診療。

方法：回顧分析5名DAVF患者（年齡介乎23-60歲）於2014年至2015年期間在我們醫院以Onyx 18和GDCs進行一次性血管內治療。在所有病例中都使用經靜脈入路雙導管技術。臨床評估包括治療效果和併發症。在栓塞後6個月和18個月用數字減影血管造影評估治療結果，並評估殘餘動靜脈分流的程度和是否存在皮質靜脈回流。

結果：在回顧的五宗病例中，所有症狀均有緩解。兩宗（40%）DAVF病例在首次栓塞中完成了閉合，當中一例的靜脈分流顯著減少。兩宗（40%）病例經過兩次獨立的栓塞治療後，靜脈分流顯著減少。沒有出現神經功能缺損的病例。

結論：Onyx 18結合GDCs進行經靜脈DAVF治療安全可行，在小樣本量的研究中具有相當高的成功率。由於DAVF是一種涉及不同嚴重程度和位置的疾病，因此治療方法應高度個性化，採用多學科方法對每位患者進行個體化治療。

INTRODUCTION

Dural arteriovenous fistulas (DAVFs) are abnormal vascular shunts between the dural arteries and dural venous sinuses. They can occur anywhere along the intracranial dura mater, although they are most frequently found involving the transverse, cavernous, and superior sagittal sinuses and the tentorium cerebelli.[1,2]

The exact incidence is unknown, but DAVFs account for 10% to 15% of intracranial arteriovenous malformations.[3] Whereas some cases of DAVFs may remain clinically silent, some may involute spontaneously[4] or present with different symptoms.

Before the mid-1970s, DAVFs were thought to be congenital in origin. In the late 1970s, an acquired aetiology of DAVFs was proposed.[5] Since then, different aetiologies have been postulated, including venous sinus thrombosis, prior head trauma or transcranial surgery, hormonal influences (e.g. pregnancy, use of oral contraceptives) that may affect angiogenesis, tumours (causing obstructing of dural venous outflow), and prior ear infection.[6] In 1995, Cognard et al[7] and Borden et al[8] proposed that DAVFs are not benign lesions, and the clinical aggressiveness of DAVFs depends on the degree of cortical venous reflux (CVR). The Cognard and Borden classifications of DAVFs are the most commonly used, and both emphasise the importance of site of venous drainage and the presence of CVR.

As described by Borden and Cognard, the lack of cortical venous drainage (Borden Type I, Cognard types I, IIa) is a favourable feature for DAVFs and these cases are associated with a benign natural history.[7,8] There is a low risk of conversion of type I DAVFs into higher grade, and a low risk of intracranial haemorrhage or death.[9,10] In contrast, for DAVFs with CVR, the reported annual risks of non-haemorrhagic neurological deficits, intracranial haemorrhage, and mortality are up to 10%.[6,10]

The presence of CVR is considered the most important determinant for management.[11] These patients may present with aggressive symptoms such as intracranial haemorrhage or neurological deficits. Other intolerable symptoms that may warrant treatment such as bruit, severe headache, seizures, and neuropsychiatric

symptoms have also been reported.[2]

Weighing the risk of treatment and natural history of DAVFs, most studies advocate treatment for high-grade lesion to avoid risks of haemorrhage and non-haemorrhagic neurological defects. Low-grade lesions with debilitating symptoms (such as severe visual symptoms or tinnitus) may also be considered candidates for therapy.

In the past, DAVFs have been treated with many different approaches. Recent studies have shown an increased success rate of transcatheter embolisation, which can achieve a high occlusion rate, and is now considered as one of the primary treatment modality.

Onyx (ev3 Endovascular Inc., Plymouth [MN], US) is a non-adhesive liquid embolic agent comprised of ethylene vinyl alcohol copolymer dissolved in dimethyl sulfoxide (DMSO), and was approved by the US Food and Drug Administration in 2005 for presurgical embolisation of brain arteriovenous malformations.[12] Since then, transarterial Onyx has gained favour over n-butyl cyanoacrylate (NBCA) in the treatment of DAVFs, with promising results.[13-16] However, limited studies have been performed on the combined use of transvenous Onyx and Guglielmi detachable coils (GDCs) for treating DAVFs.

In this paper, we present our preliminary experience in combined use of transvenous Onyx 18 and GDCs in treatment of intracranial DAVFs. The techniques, angiographic and clinical outcomes are discussed. We aimed to share our experience and provide a foundation for future studies to be performed for improving patient care.

METHODS

Patients

We retrospectively analysed all patients with DAVFs treated with combined use of Onyx 18 and GDCs performed in the same embolisation session, who were treated in our institution between 2014 and 2015. Five consecutive patients (three men and two women; mean age 37.2 years, range 23-60 years) were recruited. All DAVF shunts were located along the venous sinus wall or tentorial bridging vein with non-direct and non-exclusive leptomeningeal venous reflux, and retained venous sinus antegrade flow (Cognard classification type IIb). Two of these patients received prior embolisation treatment (one with transarterial Onyx, one with transarterial NBCA) but they presented with persistent symptoms related to residual DAVFs. The main presenting symptoms included: headache (n=4, 80%), seizure (n=2, 40%), visual symptoms (n=2, 40%), altered mental status due to underlying intraventricular haemorrhage (n=1, 20%), and pulsatile scalp mass (n=1, 20%). The baseline characteristics of the patients are summarised in Table 1.

Pretreatment angiography, including selective bilateral external and internal carotid arteries digital subtraction angiographies, were performed for diagnosis and classification of the intracranial DAVFs.

Embolisation Procedures

Embolisation procedures were performed in our endovascular operating room with biplanar digital subtraction angiography facilities. Each patient was put

Table 1. Dural arteriovenous fistula patient characteristics.

Case No.	Sex	Age (y)	Symptoms	Location of DAVF	Cognard classification	Arterial supply
1	F	31	Headache, seizure (mRS 2)	Torcular herophili	IIb	Multiple branches from bilateral ECA and ICA, and left VA
2	M	42	Headache, visual blurring (mRS 1)	Superior sagittal sinus	IIb	Bilateral MMA, STA Angiogenesis from bilateral ACA
3	M	60	Headache, altered mental status (mRS 3)	Junction of vein of Galen and straight sinus	IIb	Bilateral ECA, bilateral ICA meningohypophyseal branches, posterior meningeal branch of left VA
4	F	23	Pulsatile occipital mass (mRS 1)	Occipital, transosseous venous drainage into intracranial bulge then to transverse sinus	IIb	Right occipital artery, right STA
5	M	30	Headache, seizure, visual blurring (mRS 3)	Superior sagittal sinus	IIb	Multiple branches from bilateral ECA and ICA

Abbreviations: ACA = anterior cerebral artery; DAVF = dural arteriovenous fistula; ECA = external carotid artery; ICA = internal carotid artery; MMA = middle meningeal artery; mRS = modified Rankin Scale; STA = superficial temporal artery; VA = vertebral artery.

under general anaesthesia. Intravenous heparin bolus followed by infusion was given to maintain an activated clotting time of between 200 and 300 s. The right common femoral artery was catheterised with a 5F arterial sheath. The artery supplying the DAVF was cannulated by a 5F diagnostic catheter for arterial control. The right common femoral vein was subsequently catheterised with a 6F venous sheath. A 6F Benchmark™ 071 guiding catheter (Penumbra Inc., Alameda [CA], US) was introduced into the internal jugular vein, and superselective canalisation of the target venous sinuses was achieved using an Excelsior® 1018® (Stryker Neurovascular, Fremont [CA], US) or an Excelsior® SL10® (Stryker Neurovascular) microcatheter aided by Transend® 0.014 (Stryker Neurovascular) guidewire. A Marathon™ flow directing microcatheter (ev3 Endovascular Inc.), with Mirage™ 0.008 (ev3 Endovascular Inc.) or Hybrid 0.007 (Balt Extrusion, Montmorency, France) microguidewires, was subsequently used to cannulate the target venous sinus using the parallel co-axial technique. The microcatheter tip was placed as close as possible to the DAVF site.

The DAVF outflow venous tracts and target venous sinuses were first embolised with GDCs (Target® 360 coils, Stryker Neurovascular). Onyx vials were pre-prepared by placing on an Onyx shaker for 20 minutes prior to use. Onyx was aspirated into the Onyx Delivery Syringe and attached to the Syringe Catheter Interface Adaptor (SCIFA; ev3 Endovascular Inc., Plymouth [MN], US). DMSO was used to fill the catheter dead space. The SCIFA was then attached to the catheter hub, and pre-prepared Onyx 18 (6% ethylene vinyl alcohol) was slowly injected into the DAVFs in a controlled manner, under biplane road-mapping technique with fluoroscopic guidance. This allows direct visualisation of filling of the venous side of the DAVF. The Onyx injection was stopped when the DAVFs venous outflow tract was completely embolised, or there was persistent Onyx reflux into the distal venous sinus.

A post-embolisation angiogram was performed via the artery control catheter. All catheters were removed, and heparin infusion was discontinued and reversed with protamine at the end of the procedure.

Follow-up digital subtraction angiography at 6 months postoperatively was performed to assess the degree of residual arteriovenous shunting and presence of CVR. Cases with residual DAVF were reassessed with digital subtraction angiography at 18 months postoperatively. Patients clinical outcomes were evaluated using the modified Rankin Scale.

RESULTS

Successful embolisation of the DAVF venous outflow and involved venous sinuses was achieved in all five cases. There was no non-target embolisation.

Among the five cases, complete obliteration (total disappearance of arteriovenous shunting) of the DAVFs were achieved in two (40%) cases, and near-total occlusion (significant reduction of arteriovenous shunting with a small residual shunt) in one (20%) case, in single-stage embolisation procedure. In the other two (40%) cases, near-total occlusion of the DAVF was achieved after two separate sessions of embolisation with slow flow residual arteriovenous shunt (Figures 1 and 2). All patients reported symptom alleviation after the procedure(s).

The angiographic findings of DAVF location, total volume of Onyx injected, coil volume, angiographic and clinical outcome were summarised in Table 2. In all five cases, no residual CVR was observed on follow-up angiogram. No fistula recanalisation was observed at 6- or 18-month follow-up angiography. There were no reported cases of new neurological deficits after the procedure(s). There were no reported cases of unintentional reflux of Onyx 18 into the normal vasculature. Results from other case series are summarised and compared with the those from the present study in Table 3.[15,17-19]

DISCUSSION

Many different approaches have been used to treat DAVFs, including conservative treatment,[4] gamma knife surgery,[20] surgery,[21] transarterial or transvenous catheter embolisation,[19] or a combination of these techniques. The main aim of current endovascular treatments for DAVFs is to completely obliterate the arteriovenous shunt, which can be achieved via a transarterial or transvenous approach.[20]

Oh et al[5] reported nine patients undergoing conservative treatment of cavernous sinus DAVF, in which three patients showed complete obliteration. The other six developed new or recurrent symptoms. Conservative treatment could be considered in cases with a benign natural history (Borden Type I, or Cognard types I or IIa), and when treatment risks outweigh the potential benefits. In these cases, the aim of treatment would be clinical improvement rather than complete angiographic

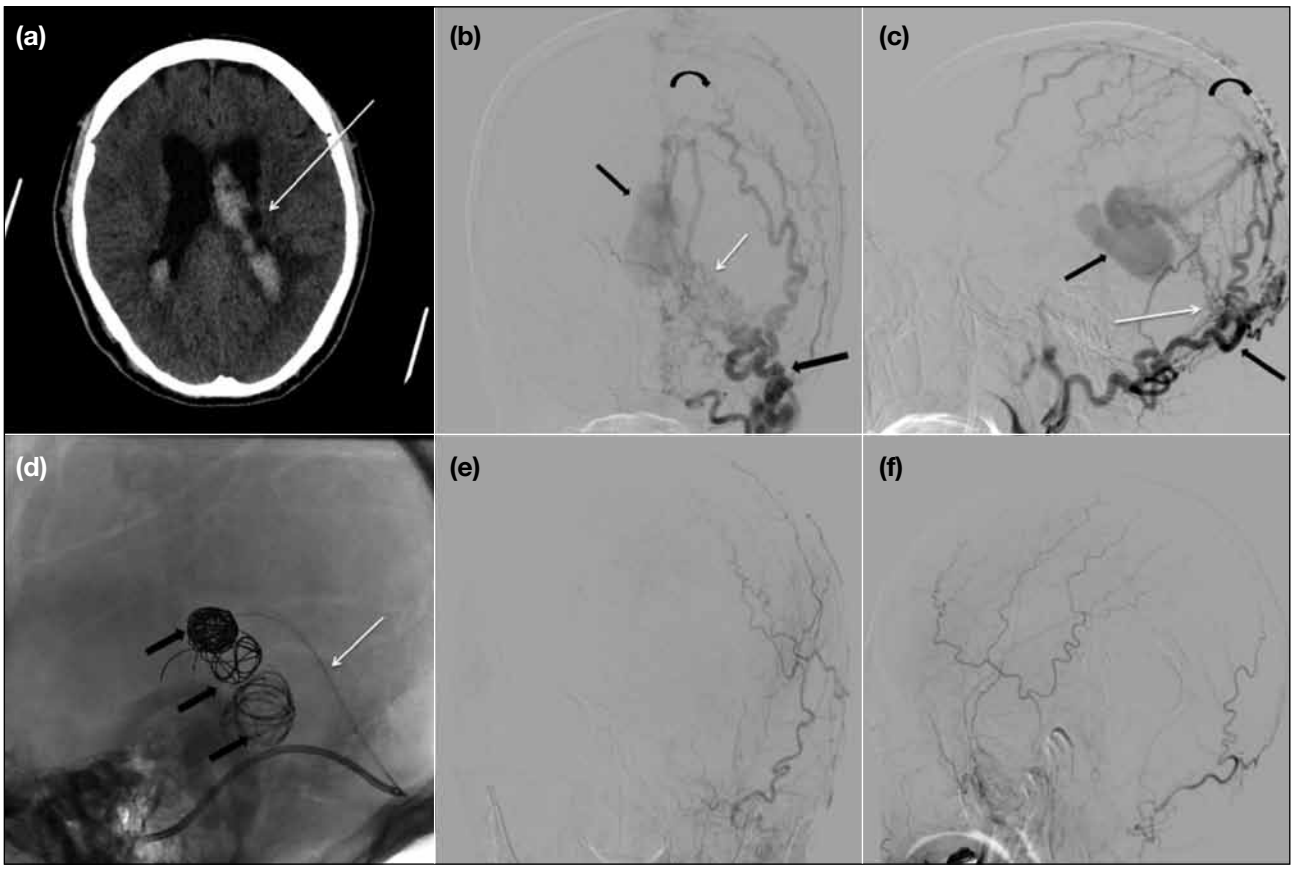

Figure 1. Case 3. Patient presented with headache and altered mental status. (a) Computed tomography of the brain showing intraventricular haemorrhage (arrow). Cranial AP (b) and lateral (c) views of left external carotid digital subtraction angiographies showing an extensive dural arteriovenous fistulas (white arrows) at junction of vein of Galen and straight sinus, with venous varices (straight black arrows) at vein of Galen. Cortical venous reflux (curved arrows) is identified. (d) Intra-operative image showing a Marathon microcatheter (white arrow) inserted through the Benchmark™ 071 guiding catheter. The pouch-like venous outflow is being embolised with multiple coils (black arrows), followed by Onyx 18. Cranial AP (e) and lateral (f) views of left external carotid digital subtraction angiographies performed 6 months after embolisation, showing complete occlusion of the dural arteriovenous fistulas, and no cortical venous reflux.

cure. However, if patients present with progressive neurological symptoms, more aggressive treatment should be considered even for benign types of DAVFs.

Studies have reported high success rate (up to 87%) of complete occlusion of DAVFs with the use of gamma knife surgery.[22] However, compared with endovascular treatment, which provides an immediate effect, gamma knife surgery has a latent period of up to 3 years before treatment effects are observed. Such delay is considered unacceptable in DAVFs with CVR, as these lesions are prone to bleeding during this latent period.

Wachter et al[21] reported different surgical approaches of treatment of DAVFs, with a high success rate for direct interrupting of the draining vein for non-sinus-type DAVFs. However, those authors also demonstrated comparable treatment outcomes between surgery and endovascular embolisation for sinus-type DAVFs, with the latter favoured for their lower complication rate.[21]

The transarterial approach requires superselective catheterisation of the arterial feeders. This method is particularly useful when the fistula site involves severely stenotic venous outflow or isolated venous sinus. However, many cases of DAVFs present with multiple small feeders derived from external and internal carotid arteries, which are mostly small fine and tortuous vessels. This makes the transarterial approach extremely difficult, if not impossible.[23,24] Even with successful superselective catheterisation of the small arterial feeders, there is still a high risk of retrograde reflux or migration of liquid embolic agent into arterialised draining veins, which may cause subsequent venous infarction. In these cases,

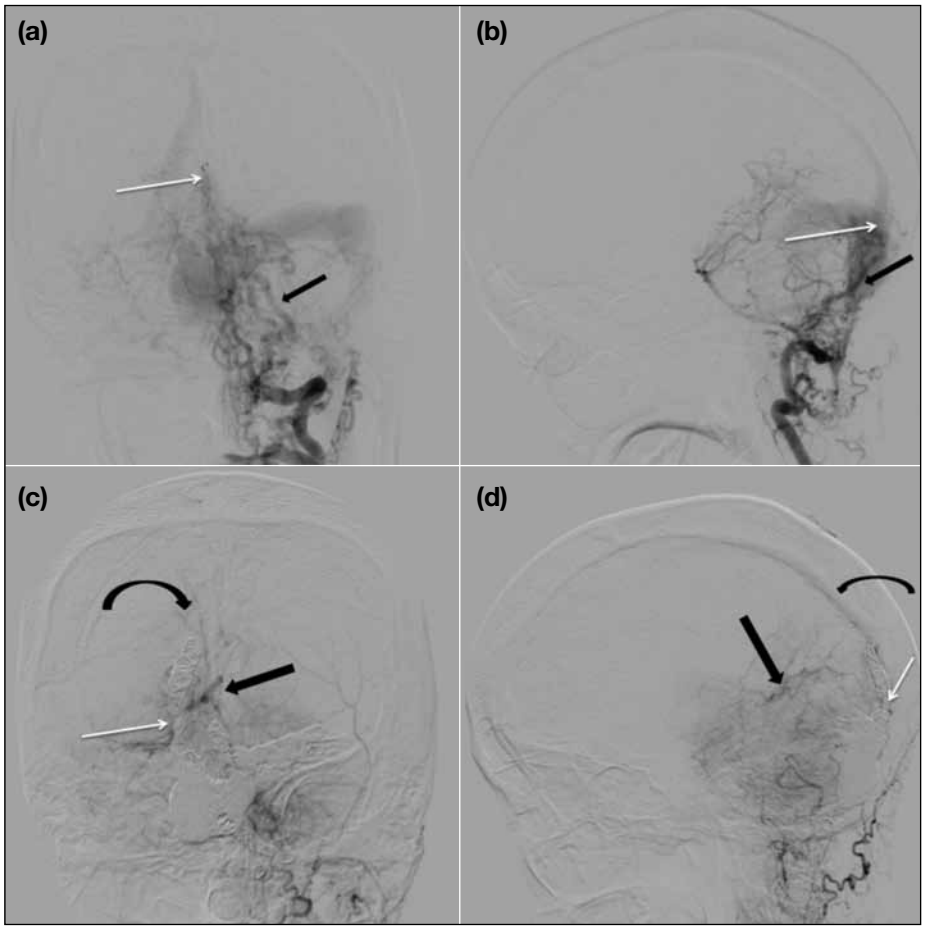

Figure 2. Case 1. (a and b) Left vertebral angiogram showing extensive dural arteriovenous fistulas (white arrow) along the tentorium, torcular herophili and posterior cranial fossa with multiple arterial supply (black arrow). Drainage into distal superior sagittal sinus, torcular and left sigmoid sinus is noted. Cranial AP (c) and lateral (d) views of left vertebral angiogram performed 6 months after embolisation. Coil mass and Onyx cast is noted in the posterior fossa at the region of the torcular herophili and left transverse sinus. Tiny residual dural arteriovenous fistulas (white arrows) is noted at distal superior sagittal sinus, supplied by the posterior meningeal artery. Significant reduction in arteriovenous shunting and cortical venous reflux (black arrows) is achieved. The superior sagittal sinus (curved arrows) remains patent.

the transvenous approach is considered safer when the diseased sinus segment can be completely occluded, as long as the diseased segment has minimal contributions to the normal venous drainage.

Transvenous embolisation involves retrograde catheterisation of the involved dural venous sinus or the cortical veins, followed by obliteration of the arteriovenous shunt by use of liquid embolic agents and/or coils. Previous studies have suggested obliteration of the fistula site solely with coils; however, complications such as postoperative cranial nerve palsy have been reported.[25] Furthermore, despite dense packing of coils, residual fistulas may still occur. Therefore, in our cases, we first deployed detachable coils into the venous outflow, and followed by slow injection of Onyx 18.

By first employing coils and then Onyx injection, the coils achieved initial flow reduction within the venous channels and form a scaffold for Onyx cast anchorage. The use of Onyx enables infiltration into the fistula site and smaller total volume of coils required.[26] Long et al[26] described their initial experience in the combined use of transvenous Onyx 18 together with coils in treatment of cavernous sinus DAVFs in eight cases. That study demonstrated promising results for the combined transvenous use of Onyx 18 and detachable coils in the management of cavernous sinus DAVFs; however, that technique for management of non-cavernous sinuses DAVFs has not been reported.

The use of Onyx involves some risks. The slow injection rate of Onyx implies a prolonged fluoroscopic time, where potential radiation-induced complications may occur.[27,28] Other complications have also been reported, including cranial nerve injury, catheter entrapment, rupture, and vasospasm due to angiotoxicity caused by both Onyx 18 and DMSO.[27-30] The risk of catheter entrapment can be avoided by positioning the catheter tip in a relatively straight vessel segment to avoid reflux around the catheter tip, although this is not always technically feasible. DMSO-induced angiotoxicity and vasospasm

Table 2. Treatment summary and outcomes.

Case No.	Location of Onyx injection and coil deployment	Total volume of Onyx injected (ml)	Total volume of coils deployed (mm^3)	Angiographic outcome	Clinical outcome
1	Distal end of SSS, torcular, occipital sinus; left transverse sinus	13.5	235.27	Near-complete occlusion (with small slow flow residual shunt)	No symptoms (mRS 0)
2	SSS	5.5	24.52	Complete occlusion	No symptoms (mRS 0)
3	Junction between the vein of Galen and straight sinus	18	646.97	Complete occlusion	No symptoms (mRS 0)
4	Venous bulge draining into transverse sinus	15	145.33	Near-complete occlusion (with small slow flow residual shunt)	Decreased occipital fullness (mRS 1)
5	Falcine sinus, SSS	34	171.93	Near-complete occlusion (with small slow flow residual shunt)	Occasional breakthrough seizures (mRS 2)

Abbreviations: mRS = modified Rankin Scale; SSS = superior sagittal sinus.

Table 3. Review of literature and comparison of treatment results.

Series	No. of cases	Modality of treatment	Complete/near-complete occlusion
Lv et al[17]	31	Transarterial Onyx embolisation	61% Complete, 39% partial
Chew et al[15]	12	Transarterial Onyx embolisation	75% Complete
Kirsch et al[19]	33	Transarterial NBCA embolisation	30% Complete
	96	Combined transarterial NBCA and transvenous coil embolisation	54% Complete
Choi et al[18]	5	Transvenous coil embolisation	80% Complete, 20% partial with residual symptoms
Our study	5	Transvenous coil plus Onyx embolisation	40% Complete, 60% near complete with no recurrence

Abbreviation: NBCA = n-butyl cyanoacrylate.

can also be prevented by slow Onyx injection,[27] at a cost of prolonged operation time. One possible limitation of Onyx is that there are limited numbers of DMSO-compatible delivery systems available for clinical use.[30] Onyx-related complications may also affect the success of this approach. For instance, large amounts of Onyx and DMSO may be required and complications related to this are possible. Furthermore, reflux of Onyx distally into normal venous sinuses will affect the treatment end-point.

Our study has some limitations. We performed our new approach in only five patients; generalisation of this treatment approach requires further evaluation. Another limitation is that our approach was performed in only selected patients where the transarterial approach was considered extremely difficult or not feasible; thus, exact comparison of the current approach with the transarterial approach is impossible.

Compared with our prior experience of transarterial embolisation, we noticed an increased rate of complete or near-complete occlusion of the shunting using our new approach. Between 2008 and 2013 we performed 12 embolisation operations for DAVFs using the transarterial approach (4 with NBCA and 8 with Onyx 18). Of them, only seven (58%) cases were able to achieve complete or near-complete occlusion, owing to the presence of multiple small and tortuous arterial feeders not-accessible by microcatheters and microguidewires.

CONCLUSION

Onyx 18 in combination with GDCs using a transvenous approach is a safe and feasible method for treating DAVFs, with a reasonably high success rate in a small sample. With our preliminary experience, we aim to improve outcome for patients with DAVF. As DAVF is a spectrum of diseases with different severities and locations, treatment approaches should be highly individualised, and a multidisciplinary approach should be adopted for treatment for each patient. Future study is recommended for selected cases where both transarterial and transvenous approaches are considered technically feasible, to compare the success rate of each modality.

REFERENCES

1. Chaudhary MY, Sachdev VP, Cho SH, Weitzner I Jr, Puljic S, Huang YP. Dural arteriovenous malformation of the major venous sinuses: an acquired lesion. AJNR Am J Neuroradiol. 1982;3:13-9.
2. Lasjaunias P, Chiu M, ter Brugge K, Tolia A, Hurth M, Berenstein M. Neurological manifestations of intracranial dural arteriovenous malformation. J Neurosurg. 1986;4:724-30. Crossref
3. Newton TH, Cronqvist S. Involvement of dural arteries in intracranial arteriovenous malformations. Radiology. 1969;93:1071-8. Crossref
4. Luciani A, Houdart E, Mounayer C, Saint Maurice JP, Merland JJ. Spontaneous closure of dural arteriovenous fistulas: report of three cases and review of the literature. AJNR Am J Neuroradiol. 2001;22:992-6.
5. Oh JT, Chung SY, Lanzino G, Park KS, Kim SM, Park MS, et al. Intracranial dural arteriovenous fistulas: clinical characteristics and management based on location and hemodynamics. J Cerebrovasc Endovasc Neurosurg. 2012;14:192-202. Crossref
6. Berenstein A, Lasjaunias P, ter Brugge KG. Surgical Neuroangiography. Berlin: Springer; 2004. p 565-607. Crossref
7. Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, et al. Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. Radiology. 1995;194:671-80. Crossref
8. Borden JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. J Neurosurg. 1995;82:166-79. Crossref
9. Shah MN, Botros JA, Pilgram TK, Moran CJ, Cross DT 3rd, Chicoine MR, et al. Borden-Shucart Type I dural arteriovenous fistulas: clinical course including risk of conversion to higher-grade fistulas. J Neurosurg. 2012;117:539-45. Crossref
10. Gross BA, Du R. The natural history of cerebral dural arteriovenous fistulae. Neurosurgery. 2012;71:594-602. Crossref
11. Davies MA, TerBrugge K, Willinsky R, Coyne T, Saleh J, Wallace MC. The validity of classification for the clinical presentation of intracranial dural arteriovenous fistula. J Neurosurg. 1996;85:830-7. Crossref
12. Onyx Liquid Embolic System (LES). Premarket Approval, The Centre for Devices and Radiological Health, Food and Drug Administration (FDA); 2005. Available from: https://www.accessdata.fda.gov/cdrh_docs/pdf3/P030004c.pdf. Accessed 13 Jun 2017.
13. Macdonald JH, Millar JS, Barker CS. Endovascular treatment of cranial dural arteriovenous fistulae: a single-centre, 14-year experience and the impact of Onyx on local practise. Neuroradiology. 2010;52:387-95. Crossref
14. Panagiotopoulos V, Möller-Hartmann W, Asgari S, Sandalcioglu IE, Forsting M, Wanke I. Onyx embolization as a first line treatment for intracranial dural arteriovenous fistulas with cortical venous reflux. Rofo. 2009;181:129-38. Crossref
15. Chew J, Weill A, Guilbert F, Raymond J, Audet ME, Roy D. Arterial Onyx embolisation of intracranial DAVFs with cortical venous drainage. Can J Neurol Sci. 2009;36:168-75. Crossref
16. Cognard C, Januel AC, Silva NA Jr, Tall P. Endovascular treatment of intracranial dural arteriovenous fistulas with cortical venous drainage: new management using Onyx. AJNR Am J Neuroradiol. 2008;29:235-41. Crossref
17. Lv X, Jiang C, Li Y, Yang X, Wu Z. Intraarterial and intravenous treatment of transverse/sigmoid sinus dural arteriovenous fistulas. Interv Neuroradiol. 2009;15:291-300. Crossref
18. Choi HS, Kim DI, Kim BM, Kim DJ, Ahn SS. Endovascular treatment of dural arteriovenous fistula involving marginal sinus with emphasis on the routes of transvenous embolization. Neuroradiology. 2012;54:163-9. Crossref
19. Kirsch M, Liebig T, Kühne D, Henkes H. Endovascular management of dural arteriovenous fistulas of the transverse and sigmoid sinus in 150 patients. Neuroradiology. 2009;51:477-83. Crossref
20. Söderman M, Edner G, Ericson K, Karlsson B, Rähn T, Ulfarsson E, et al. Gamma knife surgery for dural arteriovenous shunts: 25 years of experience. J Neurosurg. 2006;104:867-75. Crossref
21. Wachter D, Hans F, Psychogios MN, Knauth M, Rohde V. Microsurgery can cure most intracranial dural arteriovenous fistulae of the sinus and non-sinus type. Neurosurg Rev. 2011;34:337-45. Crossref
22. Guo WY, Pan DH, Wu HM, Chung WY, Shiau CY, Wang LW, et al. Radiosurgery as a treatment alternative for dural arteriovenous fistulas of the cavernous sinus. AJNR Am J Neuroradiol. 1998;19:1081-7.
23. Wakhloo AK, Perlow A, Linfante I, Sandhu JS, Cameron J, Troffkin N, et al. Transvenous n-butyl-cyanoacrylate infusion for complex dural carotid cavernous fistulas: technical considerations and clinical outcome. AJNR Am J Neuroradiol. 2005;26:1888-97.
24. Kiyosue H, Hori Y, Okahara M, Tanoue S, Sagara Y, Matsumoto S, et al. Treatment of intracranial dural arteriovenous fistulas: current strategies based on location and hemodynamics, and alternative techniques of transcatheter embolization. Radiographics. 2004;24:1637-53. Crossref
25. Nishino K, Ito Y, Hasegawa H, Kikuchi B, Shimbo J, Kitazawa K, et al. Cranial nerve palsy following transvenous embolization for a cavernous sinus dural arteriovenous fistula: association with the volume and location of detachable coils. J Neurosurg. 2008;109:208-14. Crossref
26. Long XA, Karuna T, Zhang X, Luo B, Duan CZ. Onyx 18 embolisation of dural arteriovenous fistula via arterial and venous pathways: preliminary experience and evaluation of the short-term outcomes. Br J Radiol. 2012;85:e395-403. Crossref
27. Nogueira RG, Dabus G, Rabinov JD, Eskey CJ, Ogilvy CS, Hirsch JA, et al. Preliminary experience with Onyx embolization for the treatment of intracranial dural arteriovenous fistulas. AJNR Am J Neuroradiol. 2008;29:91-7. Crossref
28. Stiefel MF, Albuquerque FC, Park MS, Dashti SR, McDougall CG. Endovascular treatment of intracranial dural arteriovenous fistulae using Onyx: a case series. Neurosurgery. 2009;65:132-9.
29. Toulgoat F, Mounayer C, Túlio Salles Rezende M, Piotin M, Spelle L, Lazzarotti G, et al. Transarterial embolisation of intracranial dural arteriovenous malformations with ethylene vinyl alcohol copolymer (Onyx18) [in French]. J Neuroradiol. 2006;33:105-14. Crossref
30. Jahan R, Murayama Y, Gobin YP, Duckwiler GR, Vinters HV, Viñuela F. Embolization of arteriovenous malformations with Onyx: clinicopathological experience in 23 patients. Neurosurgery. 2001;48:984-95.