

Transvenous embolization of arteriovenous malformations

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ABSTRACT

Embolization of arteriovenous malformations is characteristically used as part of a multimodal treatment approach, pre-operatively to facilitate microsurgical resection or as a preradiosurgical adjunct. The concept of AVM cure via embolization alone has gained popularity in recent years. Embolization of AVMs has been most commonly performed transarterially, with the transvenous route traditionally eschewed given concern over precipitating premature venous occlusion and consequent hemorrhage. However, the transvenous approach in treating AVMs offers several distinct advantages compared to the transarterial route and can be used in instances when the latter is not feasible, with several series having proven its efficacy and safety. Conceptually, AVM embolization performed via the transvenous route achieves complete obliteration by directly and facily targeting the nidus. Nidal embolisation penetration is facilitated by control of arterial inflow via systemic or local hypotension. Innovation in endovascular strategies has led to significantly improved obliteration rates. The experience with transvenous AVM embolization is reviewed and discussed.

1. Introduction

Transvenous embolization is routinely used in the treatment of carotid-cavernous fistulas, dural arteriovenous fistulas (dAVFs), and vein of Galen malformations [1–9]. This approach has widespread acceptance as safe and effective for the treatment of these lesions, in contrast to arteriovenous malformations (AVMs), as the risk of rupture for durally-supported vessels is low. Moreover, compromise of the venous outflow, without addressing the shunts, proves hazardous, with risk for AVM hemorrhage [10] and has tempered enthusiasm for use of transvenous curative AVM embolization.

Transvenous embolization as a curative standalone modality in the treatment of AVMs was first proposed by Mullan in 1994 [11], first performed in pigs in 1999 by Massoud and Hademenos [12,13] and clinically in patients in 2010 by Nguyen et al. [14]. Kessler et al. [15] later reported on a series of 5 patients with Spetzler-Martin grades III or IV AVMs treated via transvenous embolization, achieving complete obliteration in 80% of patients.

The development of slow-polymerizing non-adhesive embolic agents, such as Onyx, has facilitated the use of, and innovation in, transvenous embolization. Onyx permits a greater degree of control during injection compared to rapidly polymerizing cyanoacrylate-based

embolisates (isobutyl cyanoacrylate [IBCA], n-butyl cyanoacrylate [NBCA]), which were used previously, and has proven tremendously effective in AVM embolization, via both transarterial as well as transvenous routes [16–25]. The advent of Onyx for use as an embolisate has contemporaneously led to advancements in transvenous embolization [14,15,26–35]. Moreover, superselective catheterization of AVM and dAVF small pial veins has become possible with new microcatheter technology [5,6,8].

The clinical evidence basis for both safety and efficacy of transvenous embolization in the treatment of AVMs has continued to increase over the past decade [28,36–38,24,39–43,29–31,14,15,32,44,45,28,33–35]. A few series provide evidence for significant efficacy of transvenous embolization of AVMs with transarterial support, achieving angiographic obliteration in 95% of patients with a near 0% incidence of complications [26,28]. Combined transarterial and transvenous approaches may also be used together either contemporaneously or sequentially in the treatment of AVMs [26,46], as has been previously demonstrated in the management of dAVFs [47,48].

2. Methods

The PubMed database was searched for case series and cases

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Table 1

Transvenous embolization of arteriovenous malformations.

| Authors | AVM angioarchitecture | Prior Rx Current Rx | Obliteration and Outcomes | Complications |
|---------|--|--|---|--|
| [49] | N = 60 Mean nidus size 2.6 cm Ruptured (88.3%), unruptured (11.7%) Deep (53.3%), superficial (46.7%) Venous drainage Singular 71.7% Multiple 16.7% Unspecified 11.7% Co-existing aneurysms 5% AICA thalamostriate vein AChA | Transvenous embolization 100% Prior treatment in 53.3% transarterial Onyx 38.3% transarterial NBCA 5% transarterial coiling 3.3% microsurgical resection 1.7% stereotactic radiosurgery 5% | Complete obliteration 93.3% Residual 6.7% Functional independence 90% (mRS < / = 2) | Permanent disability 8.3% Mortality 1.7% Retained microcatheter n = 10; procedure-related hemorrhage n = 3; brainstem edema n = 1 |
| [50] | N = 10 Mean nidus size 4.16 cm Supratentorial (100%) Cortical 30% Deep 50% Cortical and deep 20% Venous drainage Superficial 40% Deep 40% Superficial + Deep 20% Afferent arterial aneurysm n = 30%, venous stenosis 30%, localized venous ectasia 10% | Transvenous embolization 100% concurrent transarterial embolization 20% | Complete obliteration 90%, durable in 100% with long-term angio follow-up (n = 7); residual (2 mm) 10%, treated with post-emo SRS; functional outcomes good 100% | Seizure (n = 1) 3 months post- embo; IVH (n = 2) without disability |
| [26] | N = 12 Mean nidus size 1.9 cm Ruptured 75%, unruptured 25% Intranidal aneurysms 41.7% Location Eloquent location 50% Supratentorial 83.3% Infratentorial 16.7% Right-sided 91.7% Left-sided 8.3% Venous drainage Singular 83.3% Double 8.3% Triple 8.3% Superficial 75% Deep 16.7% Superficial and Deep 8.3% Arterial supply Anterior circulation 75% Posterior circulation 25% | Transvenous embolization 100% Final curative embo session Transvenously 66.7% Transarterially + transvenously 33.3% | Complete obliteration 91.7% immediately; spontaneous thrombosis in 8.3% with residual Functional independence 75% upon discharge, 91.7% long term follow-up | No complications |
| [51] | N = 40, 41 AVMs Mean nidus size 2.8 cm Ruptured 65.9% Unruptured 80.5% Venous drainage Single 75% Multiple 25% Intranidal aneurysms 17% High-flow shunt 2.5% Venous stenosis 2.5% | Transvenous embolization Concurrent transarterial embolization 17% Prior transarterial embolization 27% | Complete obliteration 92.6% | Procedure-related hemorrhage 2.5% |
| [33] | N = 5 Locations CPA 20% Subthalamus + midbrain 20% Paraventricular thalamic-pulvinar 40% Choroid fissure 20% Venous drainage Deep 80% Superficial 20% | Transvenous embolization 100% Concurrent transarterial embolization 60% | Complete obliteration 100% Functional outcomes stable or improved in all | No complications |
| [52] | Systematic review of 8 series with 66 patients | | Complete obliteration 96% Functional outcome good 89% Additional treatment required in 6% | Technical complications 8% Mortality 6% |

describing curative monotherapeutic transvenous embolization of arteriovenous malformations.

3. Results

3.1. Case series

Lv et al. [46] performed a systematic review of studies inclusive of 60 patients undergoing transvenous embolization of ruptured ($n = 53$) and unruptured ($n = 7$) AVMs (see Table 1 for summary of case series). Thirty-two AVMs were deep and 28 were superficial. Venous drainage was singular in 43, multiple in 10, and unspecified in 7 patients. Mean nidus size was 2.6 cm (range 0.4–8 cm). Spetzler-Martin grades were I in 2 patients, II in 12 patients, III in 30 patients, IV in 14 patients, and V in 2 patients. Three AVMs had co-existing aneurysms of the anterior inferior cerebellar artery, thalamostriate vein, and anterior choroidal artery, all of which underwent embolization prior to that of the AVM [46]. Thirty-two lesions were previously treated with various approaches (transarterial Onyx [$n = 23$], transarterial NBCA [$n = 3$], transarterial coiling [$n = 2$], microsurgical resection [$n = 1$], and stereotactic radiosurgery [$n = 3$]). Arterial control was achieved via systemic ($n = 6$) or local ($n = 5$) hypotension. Undetachable and detachable microcatheters were used in 26 and 48 procedures, respectively; there were 10 instances or retained microcatheter with the former and none with the latter.

Outcomes were generally excellent, with complete obliteration in 56/60 (93.3%) and residual in 4/60 (6.67%), with one patient undergoing SRS treatment of residual AVM [46]. On long-term follow-up, 54/60 (90%) patients were functionally independent (mRS $< / = 2$) and five patients had permanent disability. There was one death secondary to the patient's initial hemorrhage consequent to ruptured AVM. Predictors of a good outcome included history of rupture and low and intermediate Spetzler-Martin grades (I–III).

He et al. [50] report on a series of 10 patients undergoing

transvenous embolization of AVMs with good outcomes (Fig. 1). Pre-operative Spetzler-Martin grades were I in 1 patient, II in 1 patient, III in 5 patients, IV in 1 patient, and V in 2 patients. AVMs localized to the supratentorial compartment in all patients, with 3 cortical, 1 deep white matter, 2 basal ganglia, 2 in corpus callosum, and 2 lesions with contemporaneous cortical and deep white matter involvement. Venous drainage was cortical in 4, deep in 4, and combined cortical and deep in 2 patients. Mean nidus size was 4.16 cm. Other AVM features included afferent arterial aneurysm in 3 cases, venous stenosis in 3 cases, and localized venous ectasia in 1 patient. Inclusion criteria for treatment with transvenous embolization included AVMs with a history of rupture, lesions for which resection or stereotactic radiosurgery (SRS) was not feasible or refused by the patient, lesions for which an intra-arterial approach was not feasible due to absence of arterial access, narrow arterial supplying vessels, or tortuosity of the arterial tree en route to the AVM nidus, angioarchitecture rendering a transvenous approach suitable, the presence of a single draining vein, and patient understanding of the risks [50].

Venous access was obtained via the straight sinus ($n = 5$), cortical veins via the SSS ($n = 4$), or venous tributaries via the vein of Labbe ($n = 1$) [50]. The authors employed the previously described transvenous ($n = 7$) or non-typical transvenous ($n = 3$) pressure cooker techniques to perform transvenous AVM embolization [27,44]. Hypotension to a MAP of 40–50 mmHg was established in all patients. Transarterial coil or Onyx AVM embolization was performed in an additional 2 patients. The former proves useful in the case of an extensive number of arterial feeders, while the latter is a reasonable approach in AVMs with a more limited number of arterial feeders. Balloon occlusion of the principal arterial feeder was used when several perforators derived from a dominant arterial feeder ($n = 2$).

Nine patients experienced complete obliteration post-procedurally and 1 patient had a residual 2 mm nidus, which required stereotactic radiosurgery post-embolization [50]. Among 7 patients with long-term angiographic follow-up, all had complete obliteration. The only post-

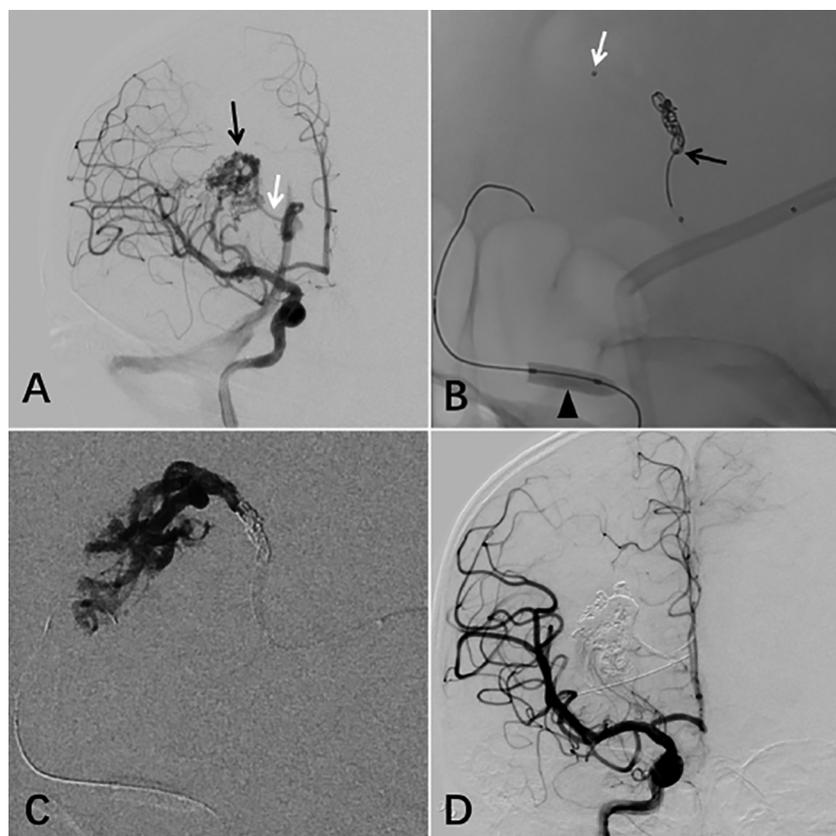


Fig. 1. Transvenous embolization of basal ganglionic AVM. (A) Right ICA digital subtraction angiogram (anteroposterior), demonstrating right basal ganglia AVM (black arrow). The AVM was supplied by perforators from the right MCA and venous drainage was via the internal cerebral vein (white arrow). (B) Unsubtracted image demonstrating transvenous pressure cooker technique using double microcatheters. Coils were deployed in the nidus-adjacent draining vein via the first microcatheter (black arrow) and Onyx was injected via the second microcatheter (white arrow). (C) Roadmap of the embolization demonstrating Onyx injection. (D) Right ICA angiogram demonstrating complete angiographic AVM obliteration. Modified with permission from Fig. 1 of [50].

procedural complication was seizure experienced in 1 patient 3 months post-embolization. All other patients remained clinically stable. Functional outcomes were good in all patients, with modified Rankin Scale scores of ≤ 1 in the entire cohort. Two patients experienced intraventricular hemorrhage without disability. These were attributable to perforation of the draining vein in a nidus-adjacent location by the microguidewire. The absence of parenchymal support around intraventricular vessels, along with high arterial input pressure and restriction of venous outflow renders them more prone to rupture [53,54]. This is in comparison to only 3 instances of procedure-related hemorrhage (and 1 case of brainstem edema) occurring in Lv et al. [46] series of 60 patients (see above). Thus, procedure-related morbidity is likely lower than presented by He et al. [50], though the two cases of IVH did not result in neurologic morbidity.

Viana et al. [26] report on a series of 12 patients with ruptured ($n = 9$) and unruptured ($n = 3$) AVMs treated with transvenous embolization (Fig. 2). Among ruptured AVMs, presentations included intraparenchymal hemorrhage (IPH) ($n = 8$), subarachnoid hemorrhage (SAH) ($n = 1$), and IVH ($n = 1$). Mean nidus size was 1.9 cm and intranidal aneurysms were present in 5 patients. Six lesions were located in eloquent parenchyma. There were 10 supratentorial and 2 infratentorial lesions, with 11 AVMs right-sided and 1 AVM left-sided. There was a single draining vein in 10 patients, two draining veins in 1 patient, and three draining veins in 1 patient. Venous drainage patterns included exclusively superficial ($n = 9$), deep ($n = 2$), and combined deep and superficial ($n = 1$). Arterial supply was from the anterior or posterior circulation in 9 and 3 patients respectively, with two lesions recruiting perforator supply. Median Spetzler-Martin grade was II.

Transvenous access was obtained via the transjugular route [26]. An

average of 2 embolization sessions were required per patient, with mean Onyx injected of 3.2 ml per patient. In the final curative embolization session, 8 were performed exclusively transvenously and 4 were performed via both transarterial and transvenous approaches (1 in which transarterial and transvenous access were performed simultaneously). Arterial catheterization was not possible in 3 patients due to narrow, tortuous arterial pedicles [26]. Angiographic obliteration was achieved in 11 patients and 1 patient had spontaneous occlusion upon 6 month follow-up. There were no complications. Mean and median modified Rankin scale (mRS) scores were 1.7 and 2 upon discharge, and 1.6 and 2 upon 6 month follow-up, respectively. Functionally independent outcome was achieved in 9 patients upon discharge and 11 patients upon 6 month follow-up.

Mendes et al. [51] report on a prospective series of 40 patients with 41 AVMs undergoing transvenous embolization. One patient was treated over two sessions and one patient harbored two lesions treated in separate sessions (42 embolization procedures). Mean age of patients was 37.7 years with 55% female. Twenty-seven patients (67.5%) presented with hemorrhage. Among patients with unruptured AVMs, presentations included seizures (22%), headache (7%), and focal neurologic deficits (2.5%). Mean nidus size was 2.8 cm. Spetzler-Martin grades were I or II in 41.5%, III in 36.5%, and IV or V in 22%. AVMs had a single draining vein in 75% of cases, with intranidal aneurysms in 17% of lesions, and high-flow shunt and venous stenosis in 2.5% each. Transarterial embolization was performed prior to transvenous embolization in 27% of patients and contemporaneous transarterial and transvenous AVM embolization were performed in 17% of patients. Onyx (85.5%), Phil (9.5%), or combined Onyx and Phil (5%) were used as embolizate. Mean follow-up was 28.4 months.

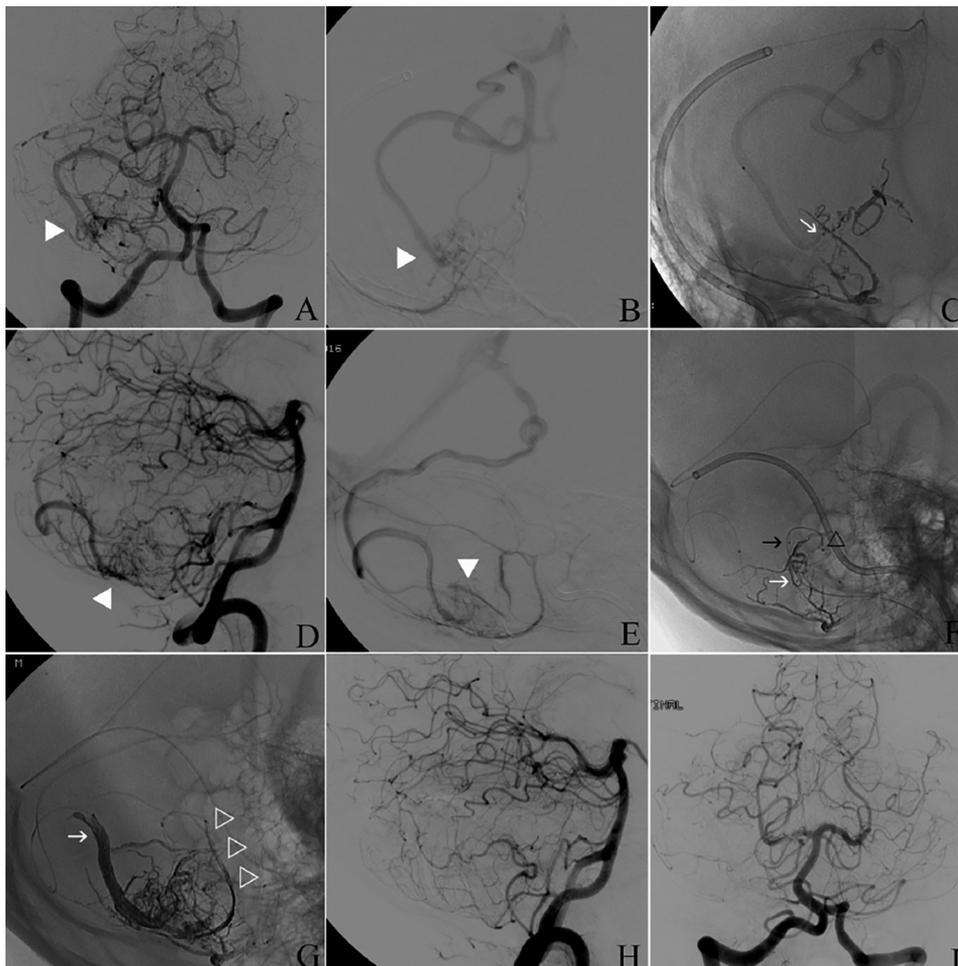


Fig. 2. Transvenous embolization, second session. (A, D) Right vertebral artery digital subtraction angiogram, anteroposterior (A) and lateral (D) views, demonstrating right cerebellar AVM (white arrowhead). (B, E) superselective DSA via microcatheter in dural arterial branch of right occipital artery, frontal (B) and lateral (E) views, demonstrating residual nidus (white arrowhead) following initial embolization session. (C) Anteroposterior skull X-ray demonstrating detachable-tip microcatheter in the AVM draining vein close to the nidus (white arrow). (F) Lateral skull X-ray demonstrating three microcatheters near the residual nidus. A detachable-tip microcatheter is located in the draining vein proximate to the nidus (white arrow), along with a detachable-tip microcatheter in a dural arterial branch of the right occipital artery (black arrow), and a microcatheter (black arrowhead) in the dural branch of the right occipital artery between the distal and detachable points of the microcatheter, ready for embolization utilizing the modified pressure cooker technique and transvenous embolization. (G) Lateral skull X-ray demonstrating the final Onyx cast following arterial embolization by arterial (white arrowheads) and venous access (white arrow). (H, I) Final right vertebral artery digital subtraction angiogram, lateral (H) and anteroposterior (I) views, demonstrating complete cerebellar AVM occlusion. Modified with permission from Fig. 3 of [26].

Complete obliteration was achieved in 92.6% of patients [51]. Complications included one procedure-related hemorrhage (2.5%) secondary to microcatheter vessel perforation and one venous infarct without clinical sequelae. There was no procedure-related mortality. On 6 month follow-up, there were no AVM recurrences, 1 patient had significant disability, and one patient died. There were no instances of venous occlusion or hemorrhagic complications with transvenous embolization.

Consoli et al. [33] report on a series of 5 patients undergoing transvenous AVM embolization, with 3 patients concurrently treated with transarterial embolization. Lesion locations included the cerebellopontine angle (n = 1), subthalamus and midbrain (n = 1), paraventricular thalamic-pulvinar (n = 2), and choroid fissure (n = 1). A single deep draining vein was present in 4 patients and 1 AVM exhibited superficial drainage. Complete obliteration was achieved in all patients without complication and functional outcomes improved or remained stable in all.

Fang et al. [52] conducted a systematic review of efficacy of transvenous embolization of brain AVMs, including 8 series with 66 patients. Complete obliteration was achieved in 96% of patients and good functional outcomes in 89%. Technical complications occurred in 8%, with additional treatment required in 6%, and mortality of 6%.

3.2. Case examples

Martinez-Galdamez et al. [55] report on a 45 year old lady with a subependymal AVM who developed recurrent IVH, initially treated with stereotactic radiosurgery and transarterial embolization, who upon presenting with recurrent IVH was successfully treated with transvenous embolization.

Benabu et al. [56] report on successful complete thrombosis of a basal ganglia/thalamic-associated venous aneurysm using transvenous embolization, following initial attempted transarterial embolization not achieving AVM occlusion. The presence of venous aneurysms confers a high risk of hemorrhage to AVMs and may cause symptoms attributable to mass effect [57–59].

Konan et al. [60] report on a 35 year old male with right-sided hemifacial spasm which resolved with transvenous embolization of a left temporo-occipital AVM. A dilated right lateral mesencephalic vein was found near the facial nerve. The lateral mesencephalic vein was approached through the superior petrosal sinus via the jugular vein transfemorally and embolized successfully, effecting symptomatic resolution.

4. Discussion

4.1. Transvenous embolization indications

Features rendering transvenous embolization a reasonable approach for AVMs include deep location, small nidus size, presence of a single draining vein, inaccessibility via the arterial route, and infeasibility of other treatment options [14,15,28–35]. AVMs not amenable to transarterial embolization include those with distal, narrow, or tortuous arterial feeders, as well as lesions in which there is arterial supply to the AVM from en passage feeders supplying normal parenchyma [61], representing excellent candidates for transvenous embolization. Transvenous embolization may also be used in the treatment of transarterially inaccessible AVM nidus remnants.

The transvenous approach may potentially and selectively be used for ruptured AVMs, lesions of high Spetzler-Martin grade, and high-risk lesions with aneurysms located intranidally, venous stenosis, high flow arteriovenous shunting, and high-flow AVMs resulting in perilesional steal [51]. Transvenous embolization is not a favored approach for plexiform pial AVMs, as early venous occlusion without securing of the multiple arterial feeders results in an exceedingly high risk for venous hypertension resulting in cerebral edema and hemorrhage [5,62,69].

4.2. Treatment strategy

The principal challenge in transvenous embolization is to fill the nidus prior to compromise of venous drainage and to control arterial inflow in order to allow steady intranidal polymerization of Onyx. Obliteration of arterial feeders, without addressing the nidus or draining vein(s) results in AVM recurrence. Thus, a complete and durable obliteration of AVMs requires nidal and draining vein occlusion [29–35].

It is prudent to achieve transarterial prior to transvenous access, in order to facilitate good angiographic AVM visualization in order to maximize the probability of nidal occlusion, as well as to have a microcatheter in place in the event of hemorrhage during transvenous embolization [26]. Nidal embolisation penetration and stasis are facilitated by strategies to reduce arterial inflow (systemic hypotension, embolization or temporary balloon occlusion of arterial feeders), allowing polymerization of transvenously-delivered embolisation within the AVM nidus [13,15,27,28,33,35,42,49,63,27,67,43].

Systemic hypotension typically targets a mean arterial pressure (MAP) 20% below baseline and can be achieved using nitroprusside, esmolol, or potent inhaled anesthetic (isoflurane), taking care to ensure somatosensory evoked potentials are well-maintained [27,33,34]. Local hypotension can be achieved via transarterial embolization (Onyx, NBCA) or temporary balloon occlusion of arterial feeders. Occlusion of AVM feeders typically reduces feeding artery pressure from 18 to 10 mmHg for at least 5 s and causes loss of the pulsatile pressure pattern. If all arterial input is shutdown, the venous pressure approaches 0 mmHg [64]. Furthermore, use of transarterial embolization contemporaneously with transvenous embolization reduces intravenous resistance and facilitates intranidal permeation of liquid embolisation [14,15,28–35].

The transvenous pressure cooker technique has been described and used as a strategy to promote intranidal permeation of Onyx embolisation [27,44,50]. In the former, two microcatheters are placed transvenously, with one nidus-adjacent in the draining vein and the other in the nidus proper. Coiling of the draining vein precedes Onyx embolization of the nidus, which increases nidal accumulation. Coil diameter should be twice that of the draining vein. The non-typical variant of the transvenous pressure cooker technique is performed when space only permits for passage of a single microcatheter, through which transvenous coil embolization of the draining vein immediately nidus-adjacent is followed by forcing the microcatheter through the coils into the nidus to then deliver Onyx. In either modification of the approach, transarterial control is achieved prior to transvenous embolization.

Efficacy of accessing the arterial side of a vascular malformation via the transvenous route was demonstrated by Albuquerque et al. [1] in a series of 4 patients with dAVFs. In these patients, transvenous to arterial Onyx embolization was performed. Lesions included two transverse-sigmoid dAVFs, an ethmoidal AVF, and a vein of Galen malformation. All dAVFs were cured and the patient with a vein of Galen malformation experienced symptomatic improvement. There were no procedure-related complications.

4.3. Complications

The major risks of transvenous embolization of AVMs include cerebral edema, venous infarct, and/or catastrophic hemorrhage consequent to occlusion of the draining vein or perforation by microcatheter or microguidewire [68,62]. The risk for hemorrhage is increased by premature draining vein occlusion, use of a high volume of Onyx, and vessel perforation by microwire or microcatheter [62]. Reflux of Onyx embolisation into arterial feeders supplying normal parenchyma may result in ischemic infarction and focal neurologic deficits.

Hemorrhage may also occur during withdrawal of an embolisation-encased microcatheter [65,66]. Thus, jugular access is preferred, as in the event of microcatheter retention within the Onyx embolisation case,

it can be cut in the neck [50]. The advent of detachable microcatheters diminishes the risk of intravenous retention and the attendant hemorrhage risk [65,66]. The security catheter technique utilizes a second microcatheter placed in an arterial feeder prior to withdrawing the transvenous microcatheter [40], which may be encased in Onyx [17–20], in order to obtain control in the event of hemorrhage.

In He et al. [50] series, the microcatheter could be safely removed in only 1 patient and was cut at the level of the jugular sheath in the remaining patients. In Lv et al. [46] series, microcatheter retentions occurred in 10 instances across 60 patients, with 10 of 26 undetachable microcatheters, and none of 48 detachable microcatheters, retained. Antiplatelet agents may be used to prevent thromboembolic complications in the event of microcatheter retention. Transvenous embolization has not been used extensively enough to provide an accurate estimate of AVM rebleeding rates following this therapy [50].

5. Conclusion

Transvenous embolization is a safe and effective noninvasive treatment modality for AVMs, either as standalone treatment, offering immediate obliteration by targeting the nidus and nidus-adjacent draining vein, or as a component in a multimodal treatment strategy. The transvenous approach achieves complete nidal obliteration, eschews arterial reflux, and reduces ischemic complications [26]. The technique is improved by strategies such as reducing arterial inflow and coiling the nidus-adjacent draining vein immediately prior to nidal Onyx injection to facilitate nidal embolisation penetration. Obliteration rates are excellent, as are functional outcomes, and procedure-related neurologic morbidity is low. Further studies are necessary to more precisely define obliteration and complication rates, as well as to make transvenous embolization more feasible and safer in the treatment of deeper arteriovenous malformations of greater angioarchitectural complexity. Further improvement and refinement in endovascular technology and strategies will facilitate these goals.

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Ethical approval

All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Conflict of interest

No conflicts of interest to disclose.

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