Peripheral arteriovenous malformations: Classification and endovascular treatment

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In this review we cover the classification and endovascular management of arteriovenous malformations (AVMs). We begin by viewing AVMs in relation to the broader class of congenital vascular malformations and subsequently go into more depth on the clinical and pathologic characteristics that define AVMs. We then focus on the endovascular treatment options for peripheral AVMs and summarize the functional characteristics of the sclerosants and embolic agents available to clinicians today.

Vascular malformations and their classification

Arteriovenous malformations (AVMs) are abnormal shunts between arteries and veins that result from disturbances in angiogenesis. They are high-flow malformations that are radiographically characterized by a central nidus, a tangle of blood vessels where the abnormal arterial-venous communication exists without a normal intervening capillary bed. They can arise anywhere in the body and therefore have a wide range of presentations, from an asymptomatic birthmark to a life-threatening impingement on vital structures. Typically present at birth, they grow concomitantly with the patient and may be stimulated to grow further after periods of trauma, hormonal change, infection or spontaneous hemorrhage. Historically, surgical resection has been considered the treatment of choice. However, due to the significant morbidity and high rate of recurrence associated with surgical resection, endovascular therapy has emerged as a less invasive alternative with comparable efficacy.[1]

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AVMs fall within the broader category of congenital vascular malformations (CVMs), which are any abnormal blood vessels arising from disturbed angiogenesis. CVMs can involve any portion of the vascular tree, including veins, arteries, capillaries, lymphatics or any combination thereof. Unlike vascular tumors, CVMs generally do not exhibit abnormalities in cellular proliferation.

Two main classification systems are used to describe CVMs: Mulliken and Hamburg. The Mulliken system divides CVMs into either high flow or low flow. This is a practical and useful classification since high flow lesions are treated with catheter-directed embolization and low flow lesions are treated with percutaneous sclerotherapy. The Hamburg system distinguishes vascular malformations based on their predominant histological components (lymphatic, arterial, venous, etc.). It divides each type into truncular, arising from the normal vascular tree, or extra-truncular, arising from outside the vascular tree. Truncular lesions arise from disturbances later in angiogenesis than extra-truncular and, in regard to treatment, tend to be higher flow than extra-truncular. In 2014, the International Society for the Study of Vascular Anomalies combined the Hamburg and Mulliken systems and also expanded on each of them (Table 1).[2] CVMs are now separated into four categories based on clinical context and vessel involvement.

Clinical staging

The Schobinger classification is a clinical assessment of vascular shunting that is predictive of treatment success (Table 2).[3] This classification has four stages, with stage 1 lesions being asymptomatic and stage 4 representing high-output heart failure. Stages 2 and 3 are intermediate, with stage 3 lesions demonstrating ulceration, bleeding,

Table 1. 2014 ISSVA Classification of Vascular Malformations

Simple	Capillary, Venous, Lymphatic, Arteriovenous*, Arteriovenous Fistula*
Combined	Any combination of simple malformations (eg. Capillary – Venous, Venous – Lymphatic, Capillary – Lymphatic – Venous)
Involves Major Vessels (Truncular)	Vessel Involved (Artery, Vein or Lymphatic) Anomaly of: Origin, Course, Number, Length, Diameter, Valves, Communication, Persistence (embryonal)
Part of Clinical Syndrome	Klippel-Trenaunay, Parkes Weber, Servelle-Martorell, Sturge-Weber, Mafucci, Cloves, Proteus, Bannayan-Riley

Simple malformations involve one type of vessel including vessel shunts such as AVMs. There are no longer arterial malformations. The star (*) indicates high-flow lesions. Combined malformations are any combination of simple malformations and can involve more than two different types. Truncular malformations are distinguished by the name of the vessel involved as well its anomalous characteristics. Clinical syndromes have a typical set of malformations that accompany them; thus, they are considered a single clinical entity.

Table 2. Schobinger Clinical Grading System of Arteriovenous Malformations

Stage 1	Warm, pink-blue stain
Stage 2	Stage 1 + Enlargement, pulsatile, bruit, thrill
Stage 3	Stage 2 + Ulceration, bleeding, pain, necrosis
Stage 4	Stage 3 + Heart failure

pain, and necrosis. In general, Schobinger stage 3 lesions and above should receive treatment.[4] Whether lower stage AVMs should be treated is still up for debate. One study found that almost all AVMs at Schobinger stage 1 eventually progressed, with nearly half becoming stage 3 and above. The authors, therefore, recommended early treatment regardless of stage.[4]

Imaging findings in AVMs

Initial imaging tests should include Doppler ultrasound and CT with contrast, or MRI. On Doppler, one may observe arterial waveforms and high flows in venous structures which are indicative of vascular shunting.[5] MRI will often show a conglomerate of flow voids on T1- and T2-weighted images that may not respect soft tissue planes (Figure 1.[6] Time-resolved MRA has emerged as a useful tool, not only to delineate nidal anatomy, but also to assess treatment efficacy through the measurement of venous filling times.[7] CT angiography is an alternative to MRA that gives comparable images; however, the 3D reconstruction lacks temporal resolution.[8] 4D-CT angiography is an emerging technology that may be able to overcome this barrier and has shown to be useful for brain AVMs.[9] In general, MRI and MRA are the preferred primary imaging modalities as they deliver both excellent anatomical resolution of the AVM and surrounding soft tissue, which are necessary for analyzing the extent of peripheral AVMs.

Diagnostic angiography should be performed on almost all AVMs and is absolutely required prior to treatment to assess flow rate, visualize anatomy of the nidus in greater detail than MRA and identify vessels required for distal circulation (Figure 2).[10] AVMs can be identified as a tangled mass of blood vessels with early venous filling. If performed during embolization, accessory feeder vessels may begin to be visualized as primary feeder vessels are embolized.[7] The architecture of the nidus on angiography has implications for treatment and outcomes. Cho described four distinct nidal architectures and found that AVMs consisting of multiple feeders emptying into a single vein, type 1 and 2, have the best response to treatment (Figure 3). AVMs with multiple inflows and outflows, types 3a and 3b, have the worst response.[11]

Endovascular treatment options

Embolic materials

Several embolic materials are available for endovascular treatment of peripheral AVMs. These embolic materials include: ethanol, N-butyl cyanoacrylate (NBCA), poly-vinyl-alcohol (PVA) particles, ethylene vinyl alcohol copolymer (Onyx), and endovascular coils and vascular plugs. Each one has specific handling criteria and learning curves. It is often the case that multiple different embolics are used to treat a single AVM. Detergents such as ethanoloamine oleate, polidocanol, and sotradecol have been reported, but are generally avoided because of increased recurrence risk.

Ethanol is widely regarded as the most effective liquid embolic. It directly damages the endothelium by acting as a protein denaturant, denuding the vessel to the internal elastic lamina. It is cost effective, but carries higher rates of complications such as significant edema, skin necrosis and nerve damage. Therefore, it should be diluted

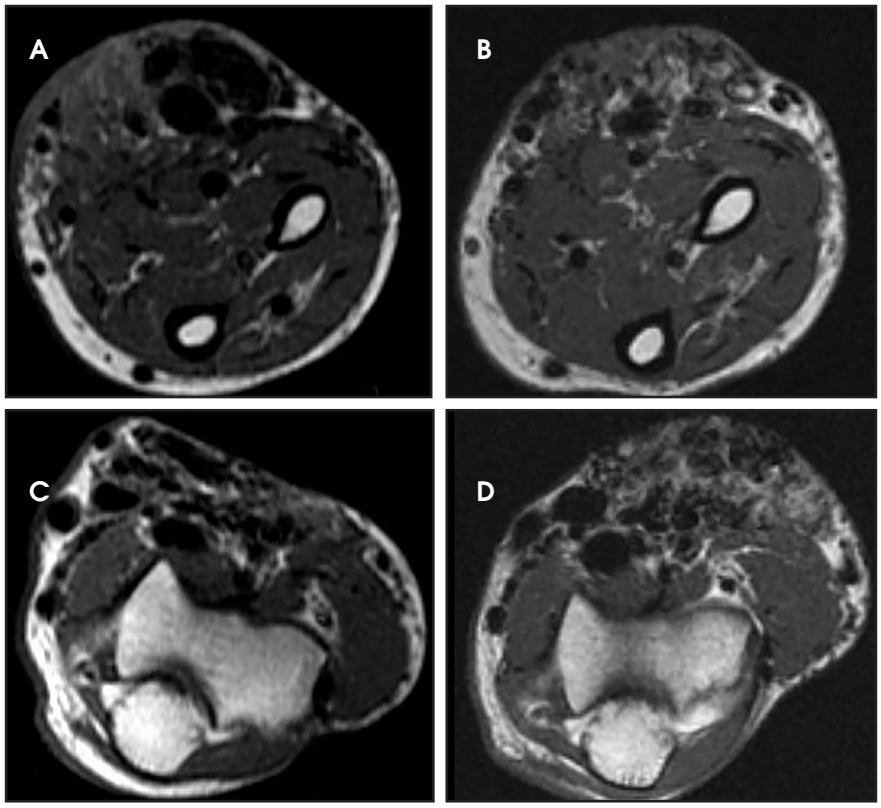

FIGURE 1. *(A,C) Axial T1-weighted images of an AVM obtained prior to embolization. Note the large flow voids on the anterior portion of the arm. (B,D) T1-weighted images obtained after staged embolization display decreased flow; however, with significant residual AVM.*

to 50% or not used at all in AVMs involving significant portions of skin.[12,13] The most-feared complication from ethanol is the dose-dependent risk of pulmonary hypertension and cardiovascular collapse, which necessitates intraprocedural pulmonary arterial pressure (PAP) monitoring.[14] PAP begins to increase with doses higher than 0.14mL/kg and the maximum recommended dose per treatment session is 0.5 – 1mL/kg administered in small 1 – 3cc aliquots; many administer less than 0.5mL/kg.[15] PAP above 25mmHg systolic merit treatment with nitroglycerin infusion at 1mcg/kg/min.[16] PAP is found to peak 10 to 15 minutes post-operatively; therefore patients must be monitored closely.[17] Outflow occlusion reduces the increase of PAP and should be employed whenever possible. Finally, ethanol is very painful and general anesthesia is required. Ethanol can be mixed with iodized oil (Lipiodol) if visualization is desired.[6]

N-butyl cyanoacrylate is a liquid casting adhesive agent generally considered to be safer than ethanol. It may be

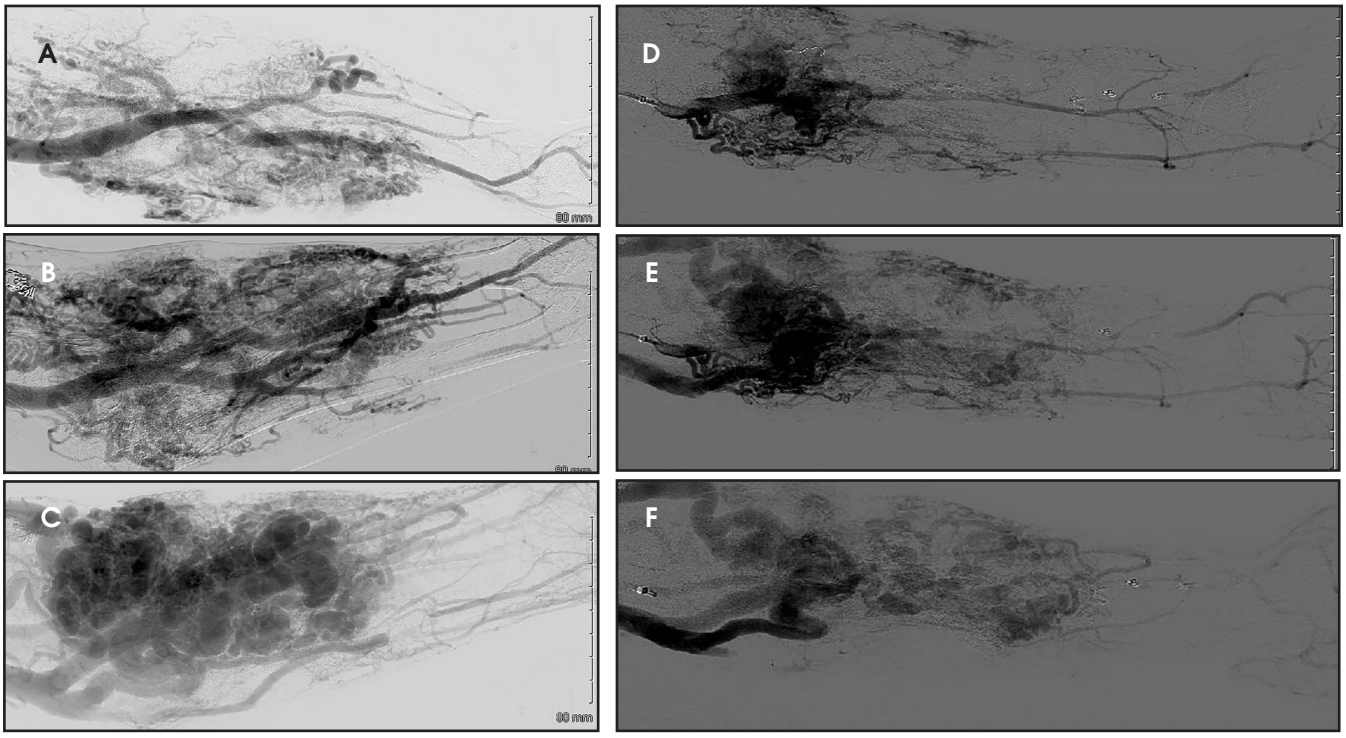

FIGURE 2. *(A,B,C) Arterial, capillary, and venous phases of angiogram performed prior to embolization. (D,E,F) Arterial, capillary, and venous phases of postembolization angiography. Although flow is significantly reduced and the patient's clinical symptoms have greatly improved, there is still a significant portion of residual AVM.*

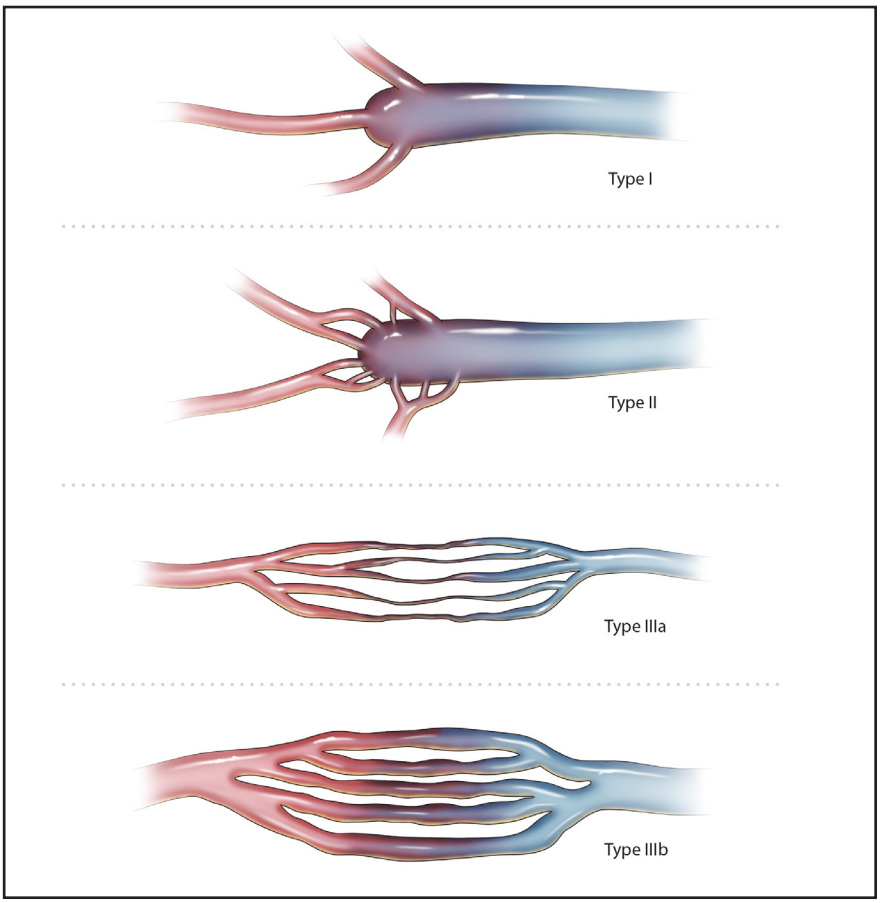

FIGURE 3. *The four types of AVM architecture described by Cho et al. Type I AVMs are defined by no more than three arterial feeders with one outflow vein. Type II AVMs contain multiple arterioles leading to a single outflow vein. Type III lesions are the most common and consist of two subtypes: IIIa, which are multiple non-dilated shunts, and IIIb, which consist of multiple dilated shunts.*

preferred in AVMs with large, draining veins that would require great amounts of ethanol or Onyx, or in the pediatric population where ethanol dosing needs to be limited.[18,19] It polymerizes quickly and irreversibly when exposed to anions and is effective in the setting of coagulopathy.[20] To administer, ingredients are mixed in a glass vial and only polypropylene syringes and catheters (Tru-Fill recommends the Prowler, Prowler Select or Transit family of catheters) may be used. To minimize premature polymerization, NBCA should diluted with non-ionic solvent (Lipiodol) and the catheter flushed with 5% dextrose solution. The catheter used for angiography may also be used for embolization if perfectly flushed, but some opt to use a new catheter. The ratio of NBCA to Lipiodol is determined based on lesion flow dynamics, with more concentrated NBCA being used for higher flow lesions. In our experience, an NBCA-to-Lipiodol ratio of 1:3 is a good starting point with an expected polymerization time of 1-4 seconds. Exact casting time is difficult to predict and may lead to complications such as embolizing too proximal or having the catheter become glued into the vessel lumen.[21] Operators should allow an adequate buffer zone so the catheter can be slowly withdrawn during administration. Adequate visualization is paramount as the operator must watch for any reflux of the embolic. Another method to prevent reflux is to "push" an aliquot of n-BCA out of the catheter with D5W allowing deeper penetration into the AVM and away from the catheter tip. If the catheter becomes adhered, a quick tug followed by prolonged traction often will remove it. However, if unsuccessful, an open removal may be necessary.

Ethylene vinyl alcohol copolymer (Onyx) is another liquid casting agent with an extensive track record for safety and efficacy in treatment of central nervous system AVMs. It has many of the same applications as nBCA and is gaining in popularity because it is easier to use.[22,23] Solubilized in DMSO, it polymerizes from outside in allowing further penetration into the nidus. It is injected slowly; the maximum injection rate is 0.1mL/min to avoid vasospasm caused by DMSO. Like NBCA, the viscosity and casting time can be changed by varying the concentration. 6-8% are the typical concentrations for adequate distal penetration from intra-arterial access. However in high flow fistulous malformations concentrations as high as 20% may be needed to achieve adequate casting time.[22] Using a test injection of contrast to determine the amount of Onyx to use is inaccurate due to the differences in viscosity and changes in flow rate during injection of the embolic. If reflux is seen around the catheter, administration should be stopped for up to 2 minutes to allow the refluxed Onyx to solidify. Afterward administration can be resumed. In the event of reflux, the catheter may be dislodged by aspirating whilst applying constant, gentle traction. It may take several seconds to dislodge and the cast may stretch up to 3 cm during this period, but this is normal. The primary advantage of Onyx over NBCA is the slow flow and longer casting time, giving the operator greater control over administration. The downside is longer treatment times and greater expense. In superficial lesions, it may cause "tattooing" of the skin.

PVA particles may be used on rare occasions to de-vascularize a lesion either as a pre-surgical adjunct or in the management of an acutely bleeding AVM. However, this carries a high risk of complications related to non-target embolization due to selection of improper particle size. Authors have used particle sizes from 150 – 500 um.[24,25]

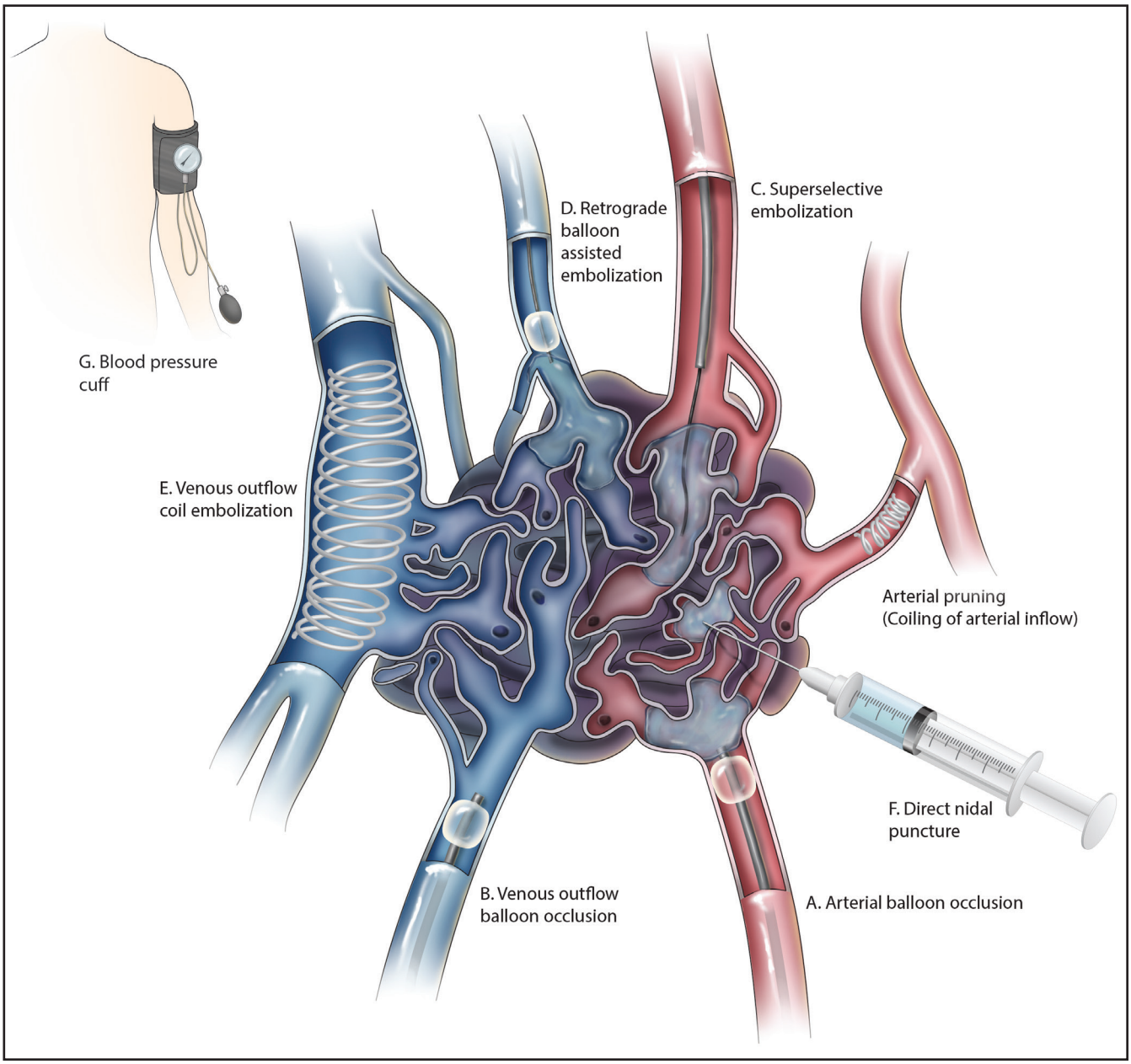

FIGURE 4. *Endovascular AVM embolization treatment techniques include: Arterial balloon occlusion (A), Venous outflow balloon occlusion (B), Superselective embolization (C), Retrograde balloon-assisted embolization (D), Venous outflow coil embolization (E), Direct nidal puncture (F), Arterial pruning (coiling of arterial feeders) (G), and the use of blood pressure cuffs to occlude venous outflow (H).*

There are several reported cases of pulmonary embolism after PVA embolization [26, 27]. We do not recommend the use of PVA particles in the management of AVMs except for in the most experienced hands and only for the above mentioned indications.

Endovascular coils and vascular plugs have a limited role in the treatment of peripheral AVMs. Their major drawback is their size which may limit future vascular access if subsequent embolization is required. They have been used as stand-alone therapy exclusively in pulmonary and renal AVMs where the large diameter and simple architecture are amenable to such treatment.[21] As an adjunct, coils and vascular plugs are useful agents for outflow occlusion, especially in nidi with a dominant outflow vein.[19, 28] Especially in very high flow AVMs, coils may sometimes may be displaced and migrate, posing a potential embolic risk; however, this is not commonly reported.[29]

Treatment techniques

The goal of AVM embolization is to obliterate the nidus while simultaneously minimizing non-target embolization. This can be best achieved by slowing the flow to improve operator control and intra or juxtanidal positioning of

Table 3. Selected review of results of AVM embolization in various clinical situations and regions of the body.

Author	Patients	Embolic (patients)	Response (patients)	Complications (number of patients)
Acute Bleeding				
Churojana, 2012[38]	5	NBCA (33 - 50%)	Recurrence (3)	Infection (2)
McGrath, 2012[25]	9	PVA (355 - 500 um, 150 - 250 um)	Requiring second embolization for control (1)	None
Pre-surgical				
Pompa, 2011[39]	11	PVA (7), Coils (4)	> 90% Flow reduction in 7 cases	None Reported
Lee, 2004[40]	16	NBCA (13), Ethanol (3)	Cure (16)	Pulmonary Embolism (1)
Head and Neck				
Kim, 2015 [41]	45	Ethanol, NBCA (15), Onyx (1)	No response (3), Cure (8), Recurrence (5)	Skin necrosis (18 - 3 require grafting), bullae (12), Stroke (1), swelling requiring decompression (1)
Dmytriw, 2014[32]	89 (244 sessions)	PVA (96), NBCA (103), Coils (5)	Cure (52 - 28 w/combined surgery, mostly small AVMs), Recurrence (2)	Swelling requiring intubation (1), post-operative bleeding (5), vision loss (1), stroke (1)
Pekkola, 2013[13]	19	Ethanol	6 with residual symptoms, 1 recurrence, 1 progression of symptoms	Skin necrosis (5 - 2 with secondary infection, 1 requiring surgery), mucosal necrosis (2), tissue necrosis (2), transient paresthesia (1), blindness (1)
Srinivasan, 2014[23]	7	Onyx 18	Partial response	Skin necrosis (1), ankle stiffness (1),
Trunk, Pelvis, Extremities				
Wohlgemuth, 2015[34]	11	Onyx, Coils (4)	Cure (8), Partial (3), Recurrence (1)	No complications
Tan, 2004[42]	13	NBCA (11), PVA (5, 300 - 500um)	No response (4), Cure (2), Recurrence (2)	Fracture (1), transient paralysis (1)
Cho, 2006[30]	66	Ethanol	Cure (21), No relief (17)	Skin necrosis (31), bullae (10), paresthesias (2), stroke (1), embolism (3), permanent nerve injury (2), infection (2), tissue necrosis (1)
Rockman, 2003[43]	50 (30 avm)	PVA (11) NBCA (22), ibca (6), coils (1)	Unchanged (4), Cured (14)	hematoma (1), recurrence requiring amputation (1)
Yakes, 2010[44]	48	Ethanol, coils	Cured (36)	Transient nerve (4), blistering (4), PE (1), tissue necrosis requiring bowel diversion (1)
Pulmonary				
Mager, 2004[29]	112	Coils	Recanalization (19)	TIA (3), cerebral abscess (2), coil migration (5 - 3 requiring retrieval), pulmonary infiltrate (1), angina (1), DVT (1)
Letourneau, 2010[45]	24	Vascular Plug	Recanalization (2 - of 19 pt f/u), all had sx improvement	No complications
Renal				
Murata, 2014[46]	12	Coils, NBCA (3), Gel Sponge (1), Ethanol (3)	Recurrence (2 - treated with coils only), Cure (10)	No complications
Dominant Outflow Vein Architecture				
Conway, 2015[19]	14	Plug and coils (13), Ethanol (2), NBCA (11 - 1:1)	No response (1), Cure (5)	No complications
Sung, 2008[31]	19	Ethanol, coil (13)	Cure (13)	Embolism (3), stroke (1), bladder necrosis (1), skin necrosis (3)

the catheter (Figure 4). The techniques to achieve these goals depend on AVM nidal architecture as well as anatomic location. We will organize treatment approaches based on the angiographic classification developed by Cho (Figure 3).[30]

In AVMs with a dominant outflow vein, types one and two, retrograde or direct puncture have become the preferred methods of access. Flow is first reduced either via manual occlusion of the draining vein or blood pressure cuff. For lesions not amenable to the above methods, such as those in the trunk, balloon occlusion of the inflow or outflow may be useful. In large aneurysmal draining veins, coils and glues have been successfully used to occlude the outflow.[28, 31] In small AVMs of this type NBCA alone has been shown to be potentially curative.[32] In most cases though, after the outflow is occluded, retrograde filling of the nidus can be achieved either with ethanol or Onyx.[19,28, 33, 34] Operators are cautioned when using high amount of outflow occlusion as sclerosant can reflux into the arterial system if injected too quickly.

For AVMs with multiple feeders and outflows, types 3a and 3b, a trans-arterial or direct puncture approach is recommended.[30] Direct puncture may be necessary in situations where the tortuosity of the arterial feeder precludes juxtanidal positioning or when the operator is unable to assess proper positioning of the catheter in very complex AVMs.[12] This may be difficult and time-consuming for small vessels and runs the risk of sclerosant .[35, 36] Flow occlusion continues to be paramount to ensure adequate contact time with the nidus. Manual compression of the draining vein or a blood pressure cuff should be employed when possible. A combination approach has been described using a trans-arterial NBCA injection to slow flow followed by direct puncture of sclerosant into nidal vessels.[35]

At our institution, we first perform catheter-based diagnostic angiography with selective and super-selective catheter positioning using compression adjuncts when appropriate. For all but the most simple AVMs, we then bring the patient back for staged treatment. In the interim, the patient's history, physical exam findings and MRI and angiographic images are reviewed by a team that includes interventional radiology, diagnostic radiology, plastic surgery and vascular surgery.

Postoperative pain and swelling should be expected and can sometimes be significant. The use of perioperative corticosteroids to control postoperative swelling is not well studied, but is recommended by those with considerable experience treating these lesions. [37] Patients are typically seen in clinic once a month after treatment begins, sooner if indicated, until the primary endpoint of symptomatic relief is reached or further endovascular treatment is precluded due to anatomical or clinical considerations.

Outcomes

There is limited data on the treatment of peripheral AVMs with most reports being small case series. Outcomes vary considerably mostly due to the heterogeneous nature of AVMs. Large, diffuse AVMs are often not curable and embolotherapy is merely palliative.[11] Small AVMs, especially those with a single outflow vein, have a high chance of cure with embolotherapy alone.[32] However, if treated correctly, most patients will experience at least symptomatic improvement after endovascular therapy. Recurrence is a common problem, especially if prior treatment resulted in loss of preferred methods of access. Table 3 provides a selected review of the literature on various AVMs.

Conclusion

Peripheral AVMs have myriad manifestations, and treatment depends on the acuity of the situation, nidal architecture and anatomic location. Treatment is often a long process requiring multiple rounds of embolization and lifelong follow up. We emphasize the use of a multidisciplinary team and to personalize treatment to the patient's wishes, as currently there is no cure. We have provided a description of treatment techniques, complications and outcomes associated with various sclerosants. We also describe our institutional techniques and literature based techniques for treating these lesions. Larger, controlled studies are needed to provide more robust data on the safety, efficacy, treatment techniques, and periprocedural management of these patients.

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