

Interventional Radiology Management of Vascular Anomalies



Gulraiz Chaudry, MBChB*, Mohammad Amarneh, MD, Raja Shaikh, MD

KEYWORDS

- Vascular anomalies • ISSVA classification • Sclerotherapy • Embolization • Venous malformation
- Lymphatic malformation • Arteriovenous malformation

KEY POINTS

- Accurate 2025 ISSVA classification is the essential first step; diagnostic errors lead to ineffective procedures, avoidable morbidity, and delayed appropriate therapy.
- Venous malformations are slow-flow congenital anomalies composed of ectatic venous channels; Sclerotherapy agent selection is guided by morphology and anatomical risk; ethanol offers highest efficacy but requires anesthesia; STS foam and bleomycin are lower-risk alternatives.
- For lymphatic malformations, doxycycline is first-line for macrocystic disease; bleomycin preferred for microcystic lesions; cumulative bleomycin dose must be tracked to prevent pulmonary fibrosis.
- Arteriovenous malformations require treatment of the nidus and draining veins; proximal arterial occlusion alone worsens disease. Ethanol and liquid embolics via combined transarterial and direct-puncture approaches are standard.
- Combined vascular malformations and overgrowth syndromes, including PIK3CA-related disorders require individualized treatment targeting embryonic veins and dominant components; sirolimus or alpelisib may provide additional systemic benefit.

INTRODUCTION

Vascular anomalies represent one of the most clinically challenging categories in multidisciplinary care, encompassing a broad spectrum of conditions from self-involuting infantile hemangiomas to life-threatening high-flow arteriovenous malformations (AVMs). The most important early decision is often not how to treat a lesion, but whether it is being correctly classified; errors in diagnosis predictably lead to ineffective procedures, avoidable morbidity, and delayed access to appropriate medical or surgical therapy. The seminal biological classification by Mulliken and Glowacki in 1982, distinguishing hemangiomas from vascular malformations based on endothelial cell kinetics, provided the conceptual foundation for modern management.¹

The International Society for the Study of Vascular Anomalies (ISSVA) has produced iteratively refined classification systems since 1996, with the most recent update released in March 2025.² For a detailed discussion of the full ISSVA taxonomy, the reader is directed to the companion classification article in this issue. The present article focuses on the conditions most frequently referred to and treated by interventional radiology (IR): venous malformations (VM), lymphatic malformations (LM), AVM, combined malformations and overgrowth syndromes, complex lymphatic anomalies, and the newly recognized fibroadipose vascular anomaly (FAVA) and intramuscular fast-flow vascular anomaly (IFVA) entities. Hereditary hemorrhagic telangiectasia (HHT) is also addressed, given its specific and life-saving interventional radiology (IR) indications.

Division of Interventional Radiology, Boston Children's Hospital and Harvard Medical School, Boston, MA, USA

* Corresponding author. Pediatric Interventional Radiologist, Boston Children's Hospital and Harvard Medical School, 300 Longwood Avenue, Boston, MA 02115.

E-mail address: gulraiz.chaudry@childrens.harvard.edu

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Abbreviations	
AVMs	arteriovenous malformations
BEET	Bleomycin electroembolotherapy
BEST	bleomycin electrosclectotherapy
CCLA	central conducting lymphatic anomaly
CMs	capillary malformations
DSA	digital subtraction angiography
EVOH	ethylene-vinyl alcohol copolymer
FAVA	fibro-adipose vascular anomaly
GLA	generalized lymphatic anomaly
GSD	Gorham-Stout disease
HHT	hereditary hemorrhagic telangiectasia
ICTH	intramuscular capillary-type hemangioma
IR	interventional radiology
ISSVA	International Society for the Study of Vascular Anomalies
KLA	kaposiform lymphangiomatosis
KTS	Klippel-Trenaunay syndrome
LIC	localized intravascular coagulopathy
LM	lymphatic malformations
LMWH	low-molecular-weight heparin
NBCA	N-butyl cyanoacrylate
PAVMs	pulmonary AVMs
PIK3CA	phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha
PROS	PIK3CA-related overgrowth spectrum
STS	sodium tetradecyl sulfate
VM	venous malformations

IR offers minimally invasive, image-guided therapies that may be curative, palliative, or serve as an adjunct to surgery and systemic medical management. The IR toolkit has expanded substantially over the past 2 decades, evolving from basic ethanol sclerotherapy to include foam sclerosants, liquid embolics, cryoablation, targeted molecular agents, and electroporation-enhanced drug delivery. Controlled trial evidence, while limited compared with many fields, is beginning to emerge for the selection of sclerotherapy agents, providing an increasingly evidence-based framework for practice.

THE 2025 INTERNATIONAL SOCIETY FOR THE STUDY OF VASCULAR ANOMALIES CLASSIFICATION: INTERVENTIONAL RADIOLOGY-RELEVANT ENTITIES

The 2025 ISSVA update introduced several changes directly relevant to interventional practice.² FAVA is now formally classified within the venous malformation category, having previously appeared only as an emerging entity. The IFVA is a newly recognized, distinct fast-flow lesion corresponding to the entity previously described as intramuscular capillary-type hemangioma (ICTH);

it harbors somatic MAP2K1 and KRAS mutations and requires management separate from classic nidal AVM. Flow status remains the primary determinant of IR strategy: slow-flow malformations (venous, lymphatic) are treated with sclerotherapy or ablation, while fast-flow lesions (AVM, arteriovenous fistula) require embolization.

Accurate preprocedural diagnosis requires clinical examination, MRI with and without contrast, and Doppler ultrasound. In selected cases, genetic or molecular testing may identify actionable mutations (eg, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha [PIK3CA], MAP2K1, TEK Receptor Tyrosine Kinase gene/ Tyrosine Kinase with Immunoglobulin and Epidermal Growth Factor Homology Domains 2 [Tie2]) that guide adjunctive pharmacotherapy.

A summary of IR-relevant vascular anomaly subtypes under the 2025 ISSVA framework, including key molecular features and corresponding IR approach, is provided in **Table 1**.

VENOUS MALFORMATIONS Pathophysiology and Clinical Presentation

Venous malformations (VMs) are the most common vascular malformations requiring treatment. They consist of ectatic, thin-walled venous channels with sparse smooth muscle and absent or deficient elastic tissue. Approximately 40% harbor somatic activating mutations in TEK (TIE2) or PIK3CA, leading to constitutive activation of the PI3K/AKT/mTOR signaling pathway. Sporadic VMs present as soft, compressible, bluish-purple masses that expand with dependency, Valsalva, or direct pressure. Intralesional thrombosis and phlebolith formation are hallmarks that contribute to chronic pain and swelling. VMs may occur anywhere but are most frequent in the head and neck, extremities, and trunk.

Ultrasound is the imaging modality of choice for initial evaluation. Hypoechoic channels with slow flow on color Doppler are the typical finding in VM. MRI may be required to evaluate the full extent of the malformation, demonstrating T2-hyperintense, septated, compressible lesions with delayed enhancement.

Localized intravascular coagulopathy (LIC) is present in up to 40% of patients with large or multifocal VMs, manifesting as chronically elevated D-dimer levels, low fibrinogen levels, and thrombocytopenia.³ LIC represents a significant perioperative risk and requires active management: low-molecular-weight heparin (LMWH) should be initiated 5 to 7 days before any sclerotherapy procedure and resumed postoperatively. Failure to recognize and treat LIC

Table 1
The 2025 International Society for the Study of Vascular Anomalies-based classification of vascular anomalies managed by interventional radiology

Major Category	Subtype/Entity (2025 International Society for the Study of Vascular Anomalies)	Key Molecular/Clinical Features	Interventional Radiology Approach
VASCULAR TUMORS – Benign	IH	GLUT-1+; VEGF-driven proliferation; involutes by age 10	Rarely needed; embolization for hepatic IH with high-output cardiac failure
VASCULAR TUMORS – Borderline	KHE	GLUT-1–; PIK3CA/GNA14; KMP	Transarterial embolization for KMP refractory to sirolimus; adjunct to surgery
SLOW-FLOW Venous (VM)	Isolated VM – Spongiform or Phlebotectatic FAVA	TEK/TIE2 or PIK3CA somatic mutation; LIC in up to 40%; phleboliths PIK3CA mutation; intramuscular infiltrative fibroadipose tissue; severe pain; predominantly adolescent females	Percutaneous sclerotherapy (ethanol, STS foam, bleomycin, polidocanol) Cryoablation preferred; sclerotherapy alone ineffective; sirolimus adjunct; surgery for refractory disease
	Multifocal VM, (BRBNS)	GI bleeding, iron deficiency; multiple mucocutaneous lesions	Serial sclerotherapy; endoscopic ablation of GI lesions; sirolimus
SLOW-FLOW Lymphatic (LM)	Isolated LM – Macrocystic/ Microcystic/Mixed Complex LM: GLA, KLA, GSD, and CCLA	PIK3CA somatic mutation; cyst morphology drives treatment response PIK3CA or NRAS; diffuse thoracic/ abdominal lymphatic involvement; high morbidity	Macrocystic: doxycycline, STS foam, bleomycin; Microcystic/mixed: bleomycin Lymphangiography and targeted embolization for chylous leaks; sirolimus systemic therapy
FAST-FLOW Arteriovenous (AVM/AVF)	Isolated AVM	MAP2K1 (MEK1) or KRAS somatic; nidus without capillary bed; Schobinger I–IV	Transarterial + direct-puncture embolization (Onyx, NBCA, Squid, ethanol); preop embolization; MEK inhibitors (trametinib) as neoadjuvant
	IFVA [2025 ISSVA new entity]	Somatic MAP2K1/KRAS mutations; intramuscular fast-flow channels; GLUT-1 negative; no true nidus; previously described as ICTH; does not involute	Targeted vessel embolization; standard AVM nidal protocols do not apply; surgery for definitive resection
	HHT (ENG/ACVRL1/SMAD4) – Pulmonary, hepatic, cerebral AVMs	Germline ENG (HHT1), ACVRL1 (HHT2), SMAD4 (JPHT); PAVM causing paradoxical emboli and hemoptysis	PAVM: coil/vascular plug embolization (feeding artery ≥ 2 mm per current guidelines); bevacizumab for refractory epistaxis/GI telangiectasias
COMBINED VM	CLVM (KTS), CVM, CLM, PROS-associated	PIK3CA somatic (PROS/CLOVES/KTS); features of component lesions; tissue overgrowth	Tailored sequential IR per dominant symptomatic component; alpelisib/ sirolimus for PROS; ablation of persistent embryonic veins.

Abbreviations: BRBNS, blue rubber bleb nevus syndrome; GA, general anesthesia; GI, gastrointestinal; IH, infantile hemangioma; KHE, kaposiform hemangioendothelioma; KMP, Kasabach-Merritt phenomenon; NRAS, neuroblastoma RAS viral oncogene homolog.

may result in acute-on-chronic disseminated intravascular coagulation (DIC) or postprocedural thromboembolism.

Indications for Intervention

Indications for IR treatment of VMs include pain, swelling, functional impairment, disfigurement, bleeding, and coagulopathy. Asymptomatic lesions in noncritical locations may be observed. Multiple treatment sessions are the rule rather than the exception; patients should be counseled that the goal is control rather than cure in most cases.

Sclerotherapy: Technique

Under fluoroscopic, ultrasound, or combined guidance, the VM is accessed with a 21–25-gauge needle; direct puncture is confirmed by aspiration of blood and contrast injection to characterize venous drainage. Identification of high-risk communications with the deep venous system is mandatory before sclerosant injection.⁴ Multisite access is often required for multiloculated lesions. Dominant draining veins may be coil-occluded via coaxial microcatheter to improve sclerosant retention and dwell time. Staged sessions are preferable to overly aggressive single treatments in infiltrative lesions or cosmetically sensitive areas.

Sclerotherapy: Agents

Absolute ethanol (95%–99%) remains the most potent sclerosant for VMs, achieving durable results through direct protein denaturation and irreversible endothelial destruction.⁵ However, its risk profile demands respect: pulmonary arterial hypertension from systemic absorption, tissue necrosis and skin slough, peripheral neuropathy, and cardiovascular collapse are well-documented. Total ethanol dose should not exceed 0.5 to 1.0 mL/kg per session.

Sodium tetradecyl sulfate (STS, 1%–3%) is increasingly preferred for VMs due to its high efficacy and lower tissue-destructive potential and pain than alcohol.⁶ The maximum dose used is 0.5 mL/kg of 3% STS, not to exceed 30 mL per procedure. Foam preparation using the Tessari method substantially increases surface area contact and enhances efficacy at lower concentrations. Polidocanol (1%–3%), similarly, a detergent agent, is widely used in European practice and produces less injection-site pain than STS.⁶ Both carry lower necrosis risk than ethanol and are supported by observational data and systematic review evidence.

Bleomycin (1 mg/mL) is a valuable sclerosant for VMs in sensitive locations, particularly the orbit, oral mucosa, and deep soft tissues, where swelling and the risk of necrosis with STS or ethanol would be prohibitive. Its mechanism of inducing DNA strand breaks produces an inflammatory fibrosis response. However, it does require prolonged dwell time to be effective, so is often mixed with albumin or used as an adjunct following initial injection of STS. Intralesional bleomycin does not appear to be associated with pulmonary fibrosis at standard sclerotherapy doses (1 unit/kg, with a maximal dose of 15 units per session), but a cumulative lifetime dose limit of approximately 400 units should be observed and tracked across all sessions. Nondose-related acute pulmonary pneumonitis is a severe, but rare, complication that has been reported after sclerotherapy.⁶ More commonly seen, in approximately 3% of patients, is hyperpigmentation in the area sclerosed or at sites of adhesives, necessitating precaution during and immediately after the procedure.⁷

Techniques Beyond Sclerotherapy

Percutaneous interstitial Neodymium-doped Yttrium Aluminum Garnet (Nd:YAG) laser photocoagulation (1064 nm) delivered via a bare fiber under ultrasound guidance achieves direct thermal coagulation of VM channels without the risk of ethanol-induced necrosis. It is particularly useful for superficial oral cavity VMs and small focal lesions in the extremities. Systemic sirolimus (rapamycin), an mTOR inhibitor targeting the PIK3CA/AKT/mTOR pathway, has demonstrated clinical benefit in complex or multifocal VMs and may be used as an adjunct to reduce lesion bulk and coagulopathy before or between IR sessions.⁸ Combined IR and pharmacologic management is increasingly favored for extensive disease. For larger, deeper intramuscular lesions, sclerotherapy can be combined with glue embolization, potentially reducing the number of procedures and the dose of sclerosant (**Fig. 1A–D**).

Fibroadipose Vascular Anomaly

FAVA, now formally recognized in the 2025 ISSVA classification as a distinct venous malformation subtype, is characterized by infiltration of skeletal muscle by fibroadipose tissue and anomalous venous channels.⁹ It most commonly involves a single muscle compartment of the lower extremity. The clinical presentation, characterized by severe intractable pain, contracture, and peripheral neuropathic symptoms, is more aggressive than that of conventional VMs. Somatic PIK3CA

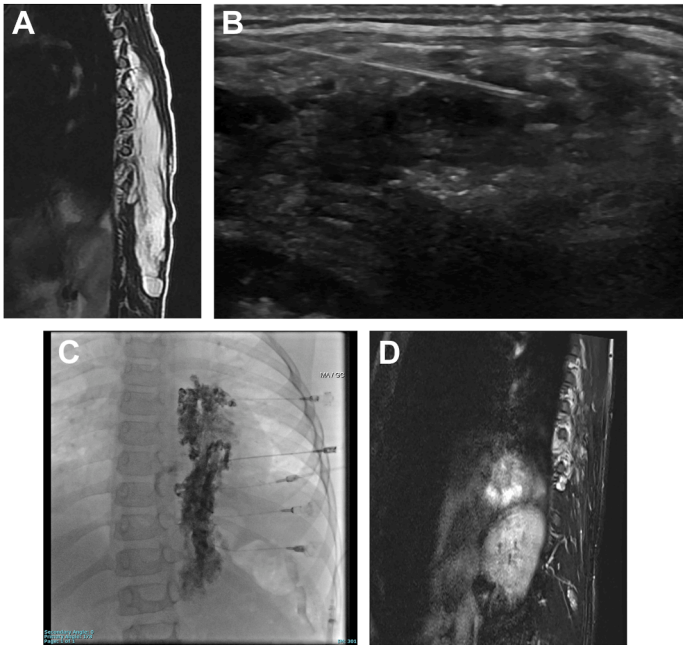


Fig. 1. (A) 6-year-old boy with a left paraspinal venous malformation. Sagittal T2 MRI image. Large hyperintense venous malformation is seen in the left paraspinal muscles. (B) The malformation is accessed under sonographic guidance. (C) End of procedure image demonstrates opacified nBCA and STS throughout the malformation. (D) Sagittal T2 MRI image. Follow-up MRI following 5 sessions of sclerotherapy. Marked decreased in size and signal intensity of the venous malformation.

mutations are identified in most cases. MRI demonstrates T1-hyperintense fibroadipose infiltration within the affected muscle, with heterogeneous enhancement on postcontrast sequences.

Conventional sclerotherapy with ethanol or STS foam is largely ineffective in FAVA due to the dominant solid fibroadipose component.⁹ Percutaneous image-guided cryoablation has emerged as the most effective IR intervention in published series. Multiple overlapping freeze-thaw cycles (typically -40 to -60°C active freeze) are applied under computed tomography (CT) or MRI guidance. Cryoablation is particularly suited to FAVA because freezing of sensory nerve fibers directly addresses the neuropathic pain component (Fig. 2A–D).¹⁰ Systemic sirolimus may reduce lesion activity and serves as a useful bridge to or adjunct of ablative therapy in extensive multi-muscle disease.⁸ Resection of the muscle should also be considered when the disease is extensive and involves a muscle that can be resected without significant functional impairment.

LYMPHATIC MALFORMATIONS

Classification and Clinical Features

Lymphatic malformations (LMs) are slow-flow anomalies arising from errors in lymphatic vessel morphogenesis. They are classified morphologically as macrocystic, microcystic, or mixed, a distinction with critical implications for treatment response and agent selection. Macrocystic LMs contain large cysts amenable to image-guided

percutaneous access and sclerotherapy; microcystic LMs consist of numerous small channels that are too small for effective conventional sclerotherapy access. Commonly cited size thresholds for macrocystic classification (often ≥ 1 cm or ≥ 2 cm) are arbitrary and not clinically reliable, as thin-walled cysts of smaller diameter can be accessed and effectively sclerosed under imaging guidance. The majority harbor somatic PIK3CA gain-of-function mutations. LMs are most commonly identified in the head and neck (approximately 75% of cases), with axillary, mediastinal, retroperitoneal, and extremity involvement less common.

Clinically, LMs present as soft, transilluminable masses. They may be asymptomatic at baseline but are prone to intralesional hemorrhage, which can cause rapid, sometimes painful enlargement, and to recurrent infections, the 2 primary indications for urgent intervention. Large cervicofacial LMs can cause airway compromise and dysphagia, requiring expedited management; postprocedural swelling should be anticipated, and airway planning undertaken in advance.

Percutaneous Sclerotherapy: Technique

Under ultrasound guidance, with fluoroscopic confirmation where appropriate, the cyst is accessed, and fluid is aspirated. Confirmation of straw-colored lymphatic fluid distinguishes LM from VM. If required, contrast can then be instilled to confirm intralesional positioning. After near-

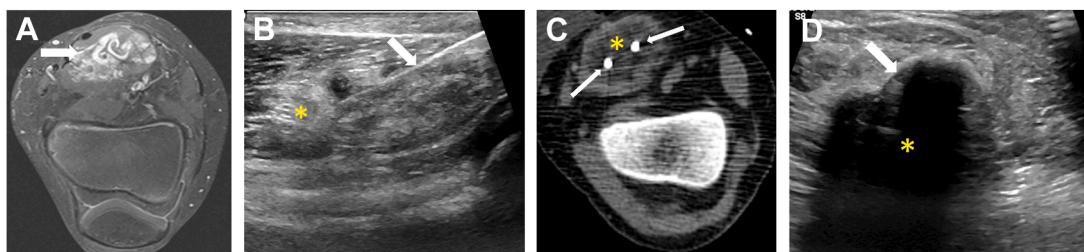


Fig. 2. (A) Axial T1W fat-saturated postgadolinium MRI image demonstrates an enhancing heterogeneous FAVA (arrow) in the soleus muscle. (B) US image demonstrates one of the cryoprobes (arrow) navigated into the lesion (asterisk). (C) Axial CT image demonstrates cryoprobes (arrows) into the lesion and the hypodense ice-ball in the lesion (asterisk). (D) US image demonstrates near edge of the ice-ball (arrow) with posterior acoustic enhancement (asterisk).

complete aspiration, the sclerosant is instilled and allowed to dwell before spontaneous resorption.¹¹ Large lesions may require multiple catheters to treat noncommunicating locules. Temporary drain placement is useful for infected, hemorrhagic, or very large lesions and facilitates interval repeat treatments. Multiple sessions are typically required for macrocystic disease; microcystic and mixed lesions require more sessions and demonstrate slower responses.

Sclerotherapy: Agents

For macrocystic LMs, doxycycline (10 mg/mL) is the most widely used first-line agent in North America, achieving response rates of 60% to 90% in published series.¹¹ Large-volume instillation is possible, with dwell times of 4 to 6 hours under general anesthesia, optimizing efficacy. A Boston Children's Hospital series demonstrated the efficacy and safety of doxycycline sclerotherapy specifically for intra-abdominal macrocystic LMs, achieving excellent response rates.¹²

Bleomycin (1 mg/mL) is the agent of choice for microcystic and mixed LMs; a Boston Children's Hospital (BCH) series of 31 patients established the safety and efficacy of bleomycin sclerotherapy for microcystic LM, achieving complete or partial response in 96% of cases.¹³ Per-session dosing of up to 15 U is used, with doxycycline administered concurrently for any macrocystic component in combined disease to reduce total bleomycin exposure. Corticosteroids are administered postprocedure in patients with lingual or floor-of-mouth LM to mitigate postsclerotherapy swelling.¹⁴ The cumulative lifetime bleomycin dose limit of approximately 400 units must be tracked across all sessions to mitigate pulmonary fibrosis risk. However, this risk is substantially lower with intralesional than intravenous administration.

STS foam (3%, Tessari method) has demonstrated efficacy as a versatile alternative for both macrocystic and mixed LMs, with a favorable safety profile and no cumulative dose limit.⁶ Absolute ethanol is generally avoided in LMs, given the risk of severe inflammatory response and tissue necrosis within these infiltrative lesions adjacent to critical structures, but it is effective in the treatment of macrocystic disease.

Adjunctive Interventions

Percutaneous image-guided cryoablation has been applied to cervicofacial LMs with good results, particularly in anatomic zones where real-time ice-ball visualization on CT or ultrasound provides a critical safety margin adjacent to facial nerves and major vessels.¹⁰

Urgent drainage may be necessary for hemorrhage, infection, or airway compromise. Surgery remains valuable in selected localized lesions.

COMPLEX LYMPHATIC ANOMALIES

Complex lymphatic anomalies include generalized lymphatic anomaly (GLA), kaposiform lymphangiomatosis (KLA), central conducting lymphatic anomaly (CCLA), and Gorham-Stout disease (GSD). These disorders may involve thoracic, abdominal, osseous, or mediastinal lymphatics and can present with chylothorax, chylous ascites, pain, respiratory compromise, or skeletal destruction. These patients benefit most when procedural planning is integrated with hematology/oncology, surgery, pulmonology, and genetics from the outset.¹⁵

Dynamic contrast MR lymphangiography, intra-nodal lymphangiography, selective leak embolization, drainage procedures, and multidisciplinary medical therapy are increasingly important components of care. Technical success depends on careful ultrasound-guided inguinal nodal access,

slow contrast injection to opacify the central lymphatic system, meticulous central lymphatic catheterization when required for therapeutic access, and thoughtful selection of glue or coil embolization targets based on the site of chylous leak. Sirolimus has demonstrated benefit as primary therapy or as a bridge to reduce lesion burden before procedural intervention.⁸

ARTERIOVENOUS MALFORMATIONS

Pathobiology and Natural History

Arteriovenous malformations are fast-flow vascular anomalies defined by direct arteriovenous connections through an intervening network of dysplastic vessels, the nidus, that bypasses the normal capillary bed. The resulting arteriovenous shunting drives progressive enlargement, ischemia of surrounding tissues, and risk of life-threatening hemorrhage. Most peripheral AVMs harbor somatic mutations in MAP2K1 (MEK1) or KRAS, activating the MAPK/ERK signaling cascade.¹⁶

The Schobinger classification describes clinical progression: Stage I (quiescence, pink-bluish stain, and warmth), Stage II (expansion, pulsation, and thrill), Stage III (destruction, ulceration, and bleeding), and Stage IV (cardiac decompensation).¹⁷ Unlike slow-flow malformations, AVMs do not involute and invariably progress. Key triggers for acceleration include trauma, hormonal change (puberty, pregnancy), and, critically, incomplete prior embolization, which stimulates angiogenic collateral recruitment and paradoxically worsens the lesion. This last point has profound practical importance: proximal arterial occlusion alone is the commonest technical error in AVM management, and partial treatment is often worse than no treatment.

Preprocedural Evaluation

Comprehensive imaging is mandatory before AVM intervention. MRI, with and without gadolinium, delineates the full extent of the malformation, including muscle and bone involvement. Time-resolved contrast-enhanced MR angiography or digital subtraction angiography (DSA) characterizes the feeding arterial architecture, nidal structure, and venous drainage pattern.¹⁷ DSA remains the gold standard for procedural planning and is typically performed in the same anesthetic session immediately before embolization. Detailed delineation of the number, caliber, and potential anastomoses (eg, external-to-internal carotid communications in head and neck AVMs) is essential for agent selection and approach planning.

Genetic or molecular analysis for MAP2K1 mutations may guide neoadjuvant pharmacotherapy.

Embolization: Principles

The cardinal principle of AVM embolization is permanent obliteration of the nidus or draining veins.¹⁷ Proximal arterial occlusion alone, whether with coils, particles, or ligation, is definitively contraindicated as sole therapy, as it eliminates angiographic access to the nidus while stimulating collateral formation. Retrograde access from the venous side of the malformation and obliteration of the nidus and draining veins offer the best chance of success. Cure is rarely achievable, and long-term surveillance and repeat treatment are required.

Transarterial embolization employs superselective microcatheter positioning as close to or within the nidus as possible. Direct puncture embolization, accessing the nidus percutaneously under fluoroscopic or ultrasound guidance, often achieves deeper nidal penetration than is possible transarterially and is the preferred approach for peripheral extremity AVMs. Combined transarterial and direct-puncture strategies in a single session optimize nidal fill. The Yakes classification system, which categorizes AVM angioarchitecture into defined subtypes, provides a useful framework for selecting the optimal interventional approach.¹⁸

Embolic Agents

Absolute ethanol remains the most potent agent for AVM embolization, inducing irreversible endothelial destruction and permanent obliteration. Under general anesthesia with pulmonary artery pressure monitoring, ethanol is injected in aliquots under roadmap fluoroscopic guidance. Its use demands significant experience; complications, including skin necrosis, peripheral nerve injury, pulmonary hypertension, and deep venous thrombosis (DVT), are well-documented and serious.

Multiple liquid embolic agents are used in the treatment of AVMs. N-butyl cyanoacrylate (NBCA) is a liquid embolic adhesive preferred for very high-flow AVM compartments and is the most commonly used liquid embolic in peripheral AVMs. Coils play a limited adjunctive role and can be used with or without glue to embolize the draining venous aneurysms seen with some AVMs. Onyx (ethylene-vinyl alcohol copolymer, EVOH), dissolved in dimethyl sulfoxide (DMSO) has become the dominant liquid embolic agent in most practices.¹⁹ Available in multiple viscosities (Onyx 18 for smaller vessels, Onyx 34 for higher-flow lesions), it forms a nonadhesive spongy cast

upon DMSO diffusion, permitting extended, controlled injection from a single microcatheter position and enabling deep nidal penetration, although the slower solidification of EVOH can risk venous penetration.¹⁹ Squid (EVOH-based, Balt) employs modified formulations aimed at reducing proximal reflux and improving penetration into smaller nidal vessels.¹⁹

Hereditary Hemorrhagic Telangiectasia

HHT is an autosomal dominant disorder caused by germline mutations in ENG (HHT1), ACVRL1 (HHT2), or SMAD4 (juvenile polyposis-HHT), which affect the TGF- β /BMP signaling pathway.²⁰ Pulmonary AVMs (PAVMs) occur in 30% to 50% of HHT patients and carry the risk of paradoxical cerebral emboli (causing stroke and brain abscess) and massive hemoptysis. Current international guidelines recommend embolization of PAVMs with feeding arteries ≥ 2 mm in diameter, even in asymptomatic patients, to prevent neurologic complications; this threshold supersedes the earlier ≥ 3 mm criterion and is supported by contemporary evidence and consensus recommendations.²¹

Technically, PAVM embolization employs a coaxial microcatheter system for superselective delivery of detachable coils, Amplatzer Vascular Plugs, or both to the feeding artery as close to the aneurysmal sac as possible.²¹ Reperfusion rates of 5% to 15% mandate lifelong surveillance imaging approximately every 3 years (CT at 6–12 months after embolization and then every 3 years per published HHT guidelines). For patients with HHT-related epistaxis or gastrointestinal telangiectasias refractory to local treatment, systemic or intranasal bevacizumab (anti-vascular endothelial growth factor A [VEGF-A]) has become an established adjunct, reducing lesion vascular density and intervention burden (Fig. 3A–D).

Intramuscular Fast-Flow Vascular Anomaly

The 2025 ISSVA classification introduces IFVA as a distinct entity within the fast-flow category.² IFVA corresponds to the entity previously described as intramuscular capillary-type hemangioma (ICTH)—characterized in a radiologic-pathologic correlation series as a glucose transporter type 1 (GLUT-1) negative fast-flow vascular lesion presenting beyond infancy with no evidence of involution.²² Subsequent molecular analysis demonstrated somatic MAP2K1 and KRAS mutations in the majority of specimens, establishing their biological overlap with extracranial AVM. On imaging, IFVA presents as an intramuscular enhancing mass with a fast-flow signal. Crucially,

no true nidus is identified on angiography, distinguishing IFVA from classic AVM. Standard nidal embolization protocols are not applicable and may cause harm. Surgical resection is the primary treatment where feasible; targeted embolization of dominant feeding vessels may be employed as a preoperative adjunct or for palliation in unresectable disease.

Combined Surgical and Interventional Approaches

For peripheral and head/neck AVMs where complete percutaneous embolization is not achievable, preoperative embolization 24 to 72 hours before surgical resection substantially reduces intraoperative blood loss and facilitates complete excision.¹⁷ Immediate postoperative DSA may identify residual nidus requiring reembolization. Where complete resection is not feasible due to involvement of critical structures, staged embolization for long-term disease control is the alternative therapeutic model.

COMBINED MALFORMATIONS AND OVERGROWTH SYNDROMES

Combined malformations involve multiple malformed vascular channel types and are often associated with tissue overgrowth. Common examples include Klippel-Trenaunay syndrome (KTS) (Capillary-Lymphatic-Venous Malformation [CLVM]), CLOVES syndrome, and broader disorders. These conditions arise predominantly from somatic PIK3CA gain-of-function mutations and require individualized, longitudinal, multidisciplinary management focused on the dominant symptomatic component.

Venous interventions—sclerotherapy for symptomatic VM components and venous ablation/embolization for persistent embryonic veins, such as the marginal vein (of Servelle) and the sciatic vein—are the most common IR procedures in this population. Lymphatic procedures are indicated for chylous complications or macrocystic LM components. Orthopedic care, compression therapy, and selective surgery address associated limb overgrowth and skeletal involvement. Targeted systemic therapy with sirolimus (mTOR inhibitor) or alpelisib (PI3K α inhibitor) has demonstrated meaningful reductions in lesion burden and improvements in symptoms in selected patients with PIK3CA-mutant PROS disorders.⁸ Head and neck vascular anomalies in the context of combined malformations require particularly careful multidisciplinary planning given proximity to the airway, great vessels, and cranial nerves.

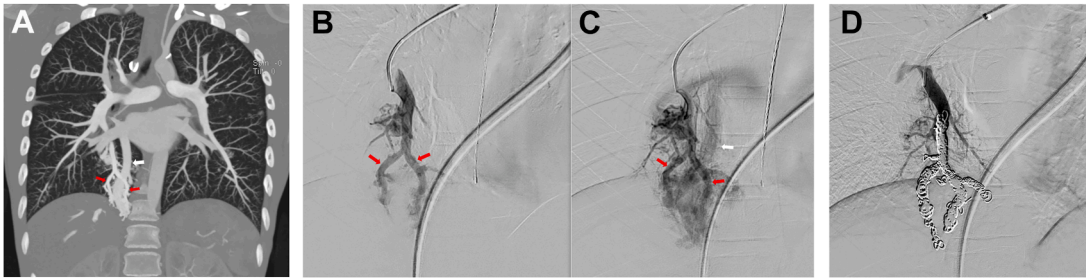


Fig. 3. (A) 13-year-old male presenting with hypoxemia (resting oxygen saturation 88%). CT angiography of the chest (coronal MIP) demonstrates a complex right lower lobe pulmonary arteriovenous malformation (PAVM) with multiple feeding arteries (red arrows) and a dominant early draining vein (white arrow). (B, C) Selective angiography of a segmental right lower lobe pulmonary artery, early arterial phase (B) and later phase (C), demonstrates multiple feeding arteries (red arrows) (B) with immediate arteriovenous shunting into the draining vein (white arrow) (C). (D) Postembolization selective angiography of the segmental artery supplying the lesion demonstrates no residual arteriovenous shunting following coil embolization of the feeding arteries.

CAPILLARY MALFORMATIONS

Capillary malformations (CMs), including port-wine birthmarks, are primarily managed by laser therapy (pulsed-dye laser, 585–595 nm) rather than interventional radiology. IR has a limited but defined role in the management of progressive CMs associated with Sturge-Weber syndrome, in which leptomeningeal pial AVM components may occasionally require embolization at specialist centers. Cutaneous CM components of combined malformations (eg, Klippel-Trenaunay syndrome) are addressed with dermatologic laser therapy concurrently with IR management of the underlying venous or lymphatic components.

SCLEROTHERAPY AGENTS: COMPARATIVE OVERVIEW

High-level randomized controlled trial evidence for selecting sclerotherapy agents in vascular anomalies remains limited. For lymphatic malformation sclerotherapy, Cahill and colleagues reported outcomes of percutaneous sclerotherapy for head and neck lymphatic malformations in neonates and infants,¹⁴ Burrows and colleagues established doxycycline efficacy for macrocystic disease,¹¹ and Chaudry and colleagues demonstrated doxycycline efficacy for intra-abdominal macrocystic LMs.¹² These data have informed consensus practices favoring bleomycin for microcystic and mixed LMs and doxycycline as first-line for macrocystic disease in North America. Prospective randomized trials directly comparing these agents in pediatric LM remain an active and unmet need in the field.

For venous malformation sclerotherapy, the systematic review by van der Vleuten and colleagues remains the most comprehensive comparative

analysis available, incorporating 35 studies and over 500 patients across eight treatment modalities.²³ The review reported high success rates for polidocanol (90%) and STS (86%), broadly comparable to ethanol (74%) when accounting for necrosis risk, with the important caveat that all included studies had a high or unclear risk of bias. STS foam and polidocanol have an established favorable safety profile compared with absolute ethanol, supporting their preferential use in anatomically sensitive locations. Ethanol retains its role in large, deeply situated VMs and in AVMs where maximal efficacy is required.

The principal sclerosants and electrosclerotherapy agents used in the IR management of vascular anomalies, including their mechanisms of action, lesion targets, key clinical considerations, and evidence levels, are summarized in **Table 2**.

EMERGING AND CUTTING-EDGE INTERVENTIONAL THERAPIES

Percutaneous Cryoablation

Cryoablation, using argon-cooled probes to achieve lethal intracellular and extracellular ice crystal formation (typically -40 to -60°C at the ablation margin), has been increasingly employed for vascular anomalies resistant to sclerotherapy.¹⁰ Its primary advantage is real-time visualization of the ice ball on CT or ultrasound, enabling precise control of the ablation margin in anatomically complex territory. It is the preferred IR modality for FAVA and has been employed with good results for cervicofacial LMs in proximity to the facial nerve and major vessels. Combination cryoablation and instillation sclerotherapy in a single session has been described with synergistic effects in selected VM and LM cases.¹⁰

Table 2
Sclerotherapy and electrochemotherapy agents used in interventional management of vascular anomalies

Agent	Lesion Type	Mechanism	Key Considerations	Evidence
Absolute ethanol 95%–99%	VM, AVM (adjunct)	Protein denaturation; endothelial destruction	Highest efficacy: GA + hemodynamic monitoring required; necrosis, neuropathy, pulmonary HTN risk; dose limit 0.5–1.0 mL/kg	Level II–III
STS foam 1%–3% (Tessari method)	VM, LM (superficial/macrocytic)	Detergent: disrupts cell membrane lipids	Foam increases surface contact and efficacy; lower necrosis risk than ethanol; anaphylaxis is rare	Level II–III (systematic review of observational series)
Doxycycline 10 mg/mL	Macrocytic LM	Inflammatory sclerosant; fibrosis induction	First-line macrocytic LM in North America; 4–6 h dwell under GA; 60%–90% response; volume limits apply	Level II–III (retrospective series)
Bleomycin 1 mg/mL	LM (micro/mixed), VM	DNA strand breaks; anti-tumor antibiotic	Preferred for microcystic/mixed LM and periorbital VM; cumulative lifetime dose ≤ 400 units; pulmonary fibrosis risk	Level II–III (retrospective series)
Polidocanol 1%–3%	VM, superficial	Detergent; endothelial damage	Less injection pain than STS; foam form available; widely used in Europe	Level III (observational series; systematic review)
BEST	VM, LM (therapy-resistant)	Electroporation-enhanced drug uptake; reversible electric pulses increase membrane permeability	Emerging; multicenter consensus protocol established; meaningful symptom improvement in pediatric cohorts; post-procedural swelling common; equinus risk for calf lesions	Level III (retrospective series; multicenter cohort)

Evidence levels: Level I, randomized controlled trial; Level II, prospective observational or cohort study; Level III, retrospective series, systematic review, or case reports.

Abbreviations: HTN, hypertension; RCT, randomized controlled trial.

Bleomycin Electrosclerotherapy

Bleomycin electrosclerotherapy (BEST) has emerged as a promising adjunctive technique for slow-flow vascular malformations, particularly venous and lymphatic lesions that are infiltrative, therapy-resistant, or anatomically difficult to treat with conventional sclerotherapy alone.²⁴ The technique combines intralesional or systemic bleomycin with delivery of reversible electric pulses to enhance local drug uptake via electroporation.²⁴ Contemporary consensus documents and multicenter practice reports have standardized procedural workflows, and recent cohort studies demonstrate meaningful improvements in pain, function, cosmesis, and lesion-related symptoms with acceptable safety profiles.

In a 2025 pediatric cohort of 45 children undergoing 68 BEST sessions for slow-flow malformations, physician-rated symptom severity and patient-reported pain, mobility, and social participation improved significantly; the overall complication rate was 14.7%, with postprocedural swelling common and calf-muscle lesions carrying particular risk of transient or persistent equinus deformity.²⁵ In a 2025 study focused on lymphatic and lymphatic-dominant malformations, BEST was reported to be safe and clinically beneficial, supporting its role as an option for selected lesions when conventional instillation sclerotherapy is likely to be suboptimal.²⁰ In a systematic review focused on lymphatic malformations of the head and neck, sclerotherapy with various agents, including bleomycin, demonstrated meaningful response rates across modalities.

Bleomycin Electroembolotherapy

Bleomycin electroembolotherapy (BEET) has more recently been described for selected extracranial arteriovenous malformations, particularly fine-fistulous lesions close to vulnerable skin, end arteries, or other structures where standard embolization or resection may carry unacceptable risk.²⁶ The rationale is that electroporation-enhanced bleomycin may provide a nonthermal, vessel-targeted adjunct in AVMs that are recurrent, refractory, or poorly suited to conventional nidus penetration. Current evidence remains preliminary and limited to recent retrospective and cohort studies rather than randomized trials.

In a 2025 comparative clinical report of BEST and BEET for AVMs, the combination of reversible electroporation and bleomycin was found to be effective, with BEET appearing to yield better outcomes than BEST in that series. However, the authors emphasized the need for prospective validation.²⁶ Accordingly, both BEST and BEET

should be regarded as emerging techniques with encouraging early efficacy and acceptable but incompletely defined complication profiles, rather than established standards of care.

COMPLICATIONS AND MANAGEMENT

All patients undergoing sclerotherapy or embolization should be counseled regarding anticipated postprocedural swelling, pain, and low-grade fever, which reflect the desired inflammatory cascade and are managed with analgesics, anti-inflammatory agents, and short-course corticosteroids as needed.⁴ Standardized peri-procedural pathways, including analgesia, anti-inflammatory medication, compression when appropriate, hydration, and clear return precautions, improve recovery and patient satisfaction.

Severe complications include skin necrosis and ulceration (ethanol, less commonly STS), peripheral nerve injury (ethanol, cryoablation in proximity to neural structures), deep venous thrombosis and pulmonary embolism (particularly post-VM sclerotherapy in patients with untreated LIC), nontarget embolization causing ischemia of adjacent structures, and pulmonary arterial hypertension from systemic ethanol absorption.⁵ The latter demands real-time hemodynamic monitoring and strict dose limitation.

Delayed complications include lesion recurrence (universal in AVMs, common in infiltrative VMs and LMs), postsclerotherapy fibrosis causing functional limitation (particularly oral cavity and periocular lesions), and the paradoxical acceleration of AVM growth following incomplete treatment.¹⁷ Long-term clinical and imaging surveillance is essential for all treated vascular anomalies.

Patients and parents should also be advised that treatment of vascular anomalies is rarely curative and is primarily aimed at alleviating symptoms, improving function and cosmesis, and preventing future complications.

SUMMARY

Across all vascular anomalies, the most durable results are achieved when procedures are thoughtfully staged, expectations are set early, and patients are followed longitudinally within an experienced multidisciplinary program.

Interventional radiology plays an indispensable role in multidisciplinary care for vascular anomalies. Venous malformations are primarily treated with image-guided sclerotherapy; agent selection is guided by lesion morphology, anatomic risk, and evolving comparative data, with STS foam and bleomycin providing effective lower-risk

alternatives to ethanol in sensitive locations. FAVA requires cryoablation rather than sclerotherapy. Lymphatic malformations are managed with percutaneous sclerotherapy using bleomycin and doxycycline as the principal agents, depending on cyst morphology. Complex lymphatic anomalies require intranodal lymphangiography, targeted embolization, and sirolimus within an integrated multidisciplinary framework. Arteriovenous malformations demand permanent occlusion/obliteration of the nidus and draining veins with ethanol and liquid embolics via combined transarterial and direct-puncture approaches. IFVA requires a distinct non-nidal management strategy with surgical resection as the primary treatment. Combined malformations and overgrowth syndromes require individualized, longitudinal multidisciplinary management, with sirolimus or alpelisib for PIK3CA-related PROS disorders. HHT-related pulmonary AVMs require embolization even when asymptomatic. Emerging therapies, such as BEST and BEET for therapy-resistant lesions, continue to expand the IR armamentarium. Optimal outcomes depend on accurate diagnosis within the 2025 ISSVA framework, thorough preprocedural planning, and sustained multidisciplinary collaboration.

CLINICS CARE POINTS

- Accurate diagnosis and use of correct nomenclature according to the International Society for the Study of Vascular Anomalies classification are essential in optimizing the treatment of vascular anomalies.
- Interventional treatment is often the mainstay of treatment of focal vascular malformations, but treatment should always be delivered in an interdisciplinary setting.
- Doxycycline is the agent of choice in the treatment of macrocystic lymphatic malformations, with bleomycin the preferred agent for microcystic and mixed lymphatic malformations.
- Sodium tetradecyl sulfate foam and polidocanol achieve durable responses in venous malformation sclerotherapy with a substantially safer profile than absolute ethanol across multiple comparative series; reserve ethanol for large or deeply situated venous malformations where superior efficacy is required, and avoid it in lesions adjacent to nerves, orbit, or oral mucosa.
- For arteriovenous malformations, proximal arterial occlusion without nidal penetration is strictly contraindicated as sole therapy and will reliably worsen disease by stimulating collateral recruitment; incomplete treatment is often worse than no treatment.

- Bleomycin electrosclerotherapy and bleomycin electroembolotherapy are promising emerging techniques for therapy-resistant slow-flow malformations and selected extra-cranial arteriovenous malformations, respectively; they should be considered adjunctive options in specialist centers while prospective validation is awaited.
- Complex lymphatic anomalies (generalized lymphatic anomaly, kaposiform lymphangiomatosis, Gorham-Stout disease, and central conducting lymphatic anomaly) require multidisciplinary management integrating hematology/oncology, surgery, pulmonology, and genetics; lymphangiography with targeted embolization and sirolimus as the principal IR and systemic therapeutic modalities.

DISCLOSURE

The authors have nothing to disclose.

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