Vascular Anomalies of the Head and Neck Region

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Abstract Vascular anomalies of the head and neck region are a complex group of lesions that challenge the head and neck physicians. From the very understanding of the difference between its two distinct forms, hemangiomas and vascular malformations to its management remain confusing. The review of this anomaly attempts at comprehensively understanding the disease. Vascular anomalies are easily diagnosed by their clinical presentation, but choice of imaging and management for this spectrum of lesions is varied. The author attempts to categorize the required imaging for the lesion with suggestions on the management of both hemangiomas and vascular malformations. The available treatment options are discussed, and a comprehensive algorithm for management is suggested. Further research in developing drugs that could restrict the growth of these lesions would be the future of the management of vascular lesions.

Keywords Vascular anomalies · Hemangiomas · Vascular malformations

Vascular anomalies (VA) by the very terminology describe a broad spectrum of pathologies involving the vascular structures of the human body. Since the biological differentiation in its classification provided by Mulliken and Glowacki, some clarity has crept into the understanding of the disease. The complexity in diagnosing and therefore

managing the pathology has been largely due to its varied spectrum of presentation. Management for VA varies from medical to minimally interventional and finally radical modalities. This article reviews the history, pathogenesis, classifications and available interventional measures in the literature and our own philosophy of management of vascular anomalies.

Historical Review and Nomenclature

Wardrop, a surgeon from London, first recognized in 1818 that vascular malformations were different from hemangioma [1]. This, however, did not help develop a philosophy of treatment. The first public demonstration of ether anesthesia by William Green Morton in 1846 was for surgical removal of a venous vascular malformation [2].

For centuries, laymen and physicians called cutaneous vascular nevi by familiar words for food. The use of such terms as “cherry”, “port-wine stain” and “strawberry” for vascular lesions can be traced back to this false doctrine of maternal impressions [3]. With the advent of histopathology in the middle of the nineteenth century, these anomalies became known as angiomas. Over the next 100 years, clinical and histological terms became hopelessly jumbled, impeding the development of this field.

Classifications

Rudolf Ludwig Karl Virchow (1863), the father of cellular pathology, deserves credit as the first to categorize vascular anomalies by microscopic channel architecture [4]. He called them angioma simplex, angioma cavernosum or angioma racemosum. Since then, many classifications have been proposed (Table 1).

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Table 1 Various classifications of vascular anomalies

Year	Author	Basis of classification
1863	Virchow RLK	Microscopic channel architecture
1877	Wegener	Histomorphologic subclassification of Virchow's classification
1973	Degni and coworkers	Site of origin of the defect
1974	Malan	Embryologic site of origin of the defect
1982	Mulliken JB and Glowacki J	Endothelial characters
1983	Burrows and colleagues	Angiographic flow patterns
1988	International Society for the Study of Vascular Anomalies (ISSVA), Hamburg	Anatomopathologic classification of vascular defects (Hamburg classification)
1989	Belov	Etiologic and pathophysiologic classification system
1992	ISSVA, Colorado	Cellular features, vascular flow characteristics and clinical behavior
1993	Jackson and associates	Flow rate
1996	ISSVA, Rome	Modified ISSVA classification
2011	S C Nair	Anatomical presentation
2014	ISSVA, Melbourne	Modified ISSVA classification

International Society for the Study of Vascular Anomalies (ISSVA) has proposed its first classification in 1988 in Hamburg, which is known as Hamburg classification. And it has been reviewed and modified several times since then, in 1992 in Colorado, in 1996 in Rome and most recently in 2014 in Melbourne [5] (Table 2).

The author introduced a more practical classification to aid in the decision making with respect to diagnostic imaging and the surgical planning as follows [1] (Table 3):

The malformations were categorized into five types depending on their anatomy and depth of location in the head and neck region. In type I superficial lesions requiring excision of skin or mucosa, local or regional flaps have been used in defect reconstruction. Type II submucosal lesions require complete excision after elevation of skin flaps. Type III lymphovenous malformations or venous malformations involving glands of the head and neck are excised along with the affected gland. Type IV intraosseous lesions require excision with involved bone and reconstruction when required. Type V lesions involving deep visceral spaces, such as the parapharyngeal or infratemporal fossa, require skeletal access osteotomy for complete exposure and total excision. The above classification helped in determining the surgical approach and reconstruction necessary for the type of vascular lesion.

Hemangiomas are subclassified into focal and segmental disease [6]. Focal hemangiomas are localized, unilocular lesions which adhere to the phases of growth and involution. Multifocal hemangiomatosis also exists, and infants with more than five lesions should undergo workup to rule out visceral involvement. Segmental hemangiomas are more diffuse plaque like and can lead to untoward

functional and aesthetic outcomes. The limb and face are common locations for the disease.

Incidence

Overall, infantile hemangiomas have an incidence of 3–10% in the literature, but more evidenced study confirmed that it is likely 4–5% [7]. A multicentric prospective study with an aim to identify demographics of infantile hemangiomas concluded female gender, prematurity, multiple gestations, Caucasian ethnicity, low birth weight and advanced maternal age are the associated risk factors [8].

Among vascular lesions, venous malformations are the common along with lymphatic ones having incidence of 1:5000–10,000, and surprisingly 40% of them usually occur in head and neck regions [9].

Diagnosis

Clinical diagnosis of hemangiomas generally arises from making a thorough physical examination which considers the shape, consistency and character delineation and possible invasive lesion, with subsequent deterioration of vital functions. In most cases no further investigation is needed, but achieving the differential diagnosis of hemangiomas is imperative to rule out other diseases, which are potentially more serious.

Similarly, venous malformations occurring in superficial areas are usually easy to diagnose by clinical examination. However, for those lesions that are deep in the face and neck, it is sometimes difficult to make a correct diagnosis

Table 2 ISSVA classification for vascular anomalies; 20th ISSVA Workshop, Melbourne, April 2014

VASCULAR ANOMALIES						
VASCULAR MALFORMATIONS						
VASCULAR TUMORS	Simple	Combined		Of major named vessels	Associated with other anomalies	
Benign	Capillary malformations	CM+	capillary-venous malformation	CVM	Affect lymphatics	Klippel-Trenaunay syndrome: CM + VM +/- LM + limb overgrowth
Infantile hemangioma / Hemangioma of infancy	Cutaneous and/or mucosal CM, Telangiectasia, CMTC, Nevus simplex, Others	CM+	capillary-lymphatic malformation	CLM	veins	Parkes Weber syndrome: CM + AVF + limb overgrowth
Congenital hemangioma	Lymphatic malformations	CM+	capillary-arteriovenous malformation	CAVM	Anomalies of origin	Servelle-Martorell syndrome: limb VM + bone undergrowth
Rapidly involuting (RICH)	Common (cystic) LM, Generalized lymphatic anomaly (GLA), LM in Gorham-Stout disease, Channel type LM, Primary lymphedema, Others	CM+	capillary-lymphatic-venous malformation	LVM	course	Sturge-Weber syndrome: facial + leptomeningeal CM + eye anomalies
Non-involuting (NICH)	Venous malformations	LM+	capillary-lymphatic-venous malformation	CLVM	number	+/- bone and/or soft tissue overgrowth
Partially involuting (PICH)	Common VM, Familial VM, cutaneo-mucosal (VMCM), Blue rubber bleb nevus (Bean) syndrome VM, Glomuvenous malformation (GVM), Cerebral cavernous malformation (CCM), Others	LM+	capillary-lymphatic-arteriovenous malformation	CLAVM	length	Limb CM + congenital non-progressive limb hypertrophy
Tufted angioma	Arteriovenous malformations	CM+	capillary-venous-arteriovenous malformation	CVAVM	diameter	Maffucci syndrome: VM +/- spindle-cell hemangioma + enchondroma
Spindle-cell hemangioma	Common VM, Familial VM, cutaneo-mucosal (VMCM), Blue rubber bleb nevus (Bean) syndrome VM, Glomuvenous malformation (GVM), Cerebral cavernous malformation (CCM), Others	CM+	capillary-venous-arteriovenous malformation	CLVAVM	(aplasia, hypoplasia, stenosis, ectasia / aneurysm)	Macrocephaly - CM (M-CM / MCAP)
Epithelioid hemangioma	Arteriovenous fistula	CM+	capillary-venous-arteriovenous malformation	CLVAVM	valves	Microcephaly - CM (MICCAP)
Pyogenic granuloma (aka lobular capillary hemangioma)	Sporadic, In HHT, In CM-AVM, Others	CM+	capillary-venous-arteriovenous malformation	CLVAVM	communication (AVF)	Cloves syndrome: LM + VM + CM +/- AVM + lipomatous overgrowth
Others	Locally aggressive or borderline	CM+	capillary-venous-arteriovenous malformation	CLVAVM	persistence (of embryonal vessel)	Proteus syndrome: CM, VM and/or LM + asymmetrical somatic overgrowth
Kaposiform hemangioendothelioma	Others	CM+	capillary-venous-arteriovenous malformation	CLVAVM		Bannayan-Riley-Ruvalcaba sd: AVM + VM + macrocephaly, lipomatous overgrowth
Reticiform hemangioendothelioma		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Papillary intralymphatic angioendothelioma (PILA), Dabska tumor		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Composite hemangioendothelioma		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Kaposi sarcoma		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Others		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Malignant		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Angiosarcoma		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Epithelioid hemangioendothelioma		CM+	capillary-venous-arteriovenous malformation	CLVAVM		
Others		CM+	capillary-venous-arteriovenous malformation	CLVAVM		

Table 3 Categorization of vascular malformation based on anatomical presentation [1]

Type I	Mucosal/cutaneous
Type II	Submucosal/subcutaneous
Type III	Glandular
Type IV	Intraosseous
Type V	Deep visceral

Fig. 1 Clinical presentation of patient having hemangiomatous lesion of face

through clinical examination alone. Imaging studies using ultrasound (US), CT, MRI (MR hemography) are the best diagnostic scans [10].

There are clear differentiating features between hemangiomas (Fig. 1) and vascular malformation (Fig. 2) in their clinical presentation and behavior (Table 4).

The widespread introduction of modernized diagnostic imaging techniques like color Doppler ultrasound (CDUS), MRI, CECT scan, digital subtraction angiography (DSA) (catheterizing the entire vertebral or vascular tree) and MR venography for low-flow lesions has been aiding the surgeon to manage these lesions (Table 5).

In few cases, it is not unusual to see the involvement of parenchyma that provides a valid reason to opt for additional imaging diagnostic modality such as MRI or CT (with or without contrast usage). Sometimes, Doppler USG may be useful in hemangiomas extending to other layers of skin, as a part of changes as in cysts or lymph node changes.

Fig. 2 Clinical presentation of patient having vascular malformation of face

In T2 weighted images (Fig. 3), venous malformations can appear as “venous lakes”. MRI sectional images can avoid signal overlap and thereby demonstrate the relationship between the lesion and the deep structure. MRI is therefore superior to CT (Fig. 4) in demonstrating the relationship between the extent of the lesion and normal tissue.

Histopathologic Features [13] (Table 6)

In distinction to hemangiomas, vascular malformations result from abnormal vascular or lymphatic morphogenesis, not due to abnormal endothelial proliferation. Despite some similarities in appearance between vascular malformations and hemangiomas, the histopathologic features are quite different.

Management of Vascular Anomalies

Several algorithms for management of vascular lesions have been proposed till date (Fig. 5).

Table 4 Features of vascular lesions [11]

Hemangioma	Vascular malformation
Present at birth, most diagnosed by 1 year old	Present at birth but often not diagnosed until second decade
Rapid growth until age 6–8 months, then slows and involutes by 5–9 years	Slow growth throughout life with increase in response to infection, trauma or hormonal fluctuation; does not involute
Neoplastic growth with increased endothelial cell turnover	Growth due to flow dynamics through the lesion and recruitment of collateral supply
Osseous involvement rare	Osseous involvement 35%
Female-to-male ratio 5:1	Female-to-male ratio 2:1
Usually low flow	May be low flow (capillary, venous, lymphatic) or high flow (arterial or arteriovenous)
Frequently does not need treatment	Often requires treatment

Table 5 Key imaging features of most common vascular lesions of head and neck [12, 14]

	US with CDUS	MRI
IH	Hyperechoic or hypoechoic Hypervascular on CDUS	Iso to intermediate signal on T1W, bright signal on T2W, high-intensity flow enhancement on gradient echo, internal flow voids, vigorous enhancement after contrast administration
RICH	Central, non-enhancing, hypodense, hypoechoic, more robust feeding vessels with large diameter than IH	T2W hyperintense component is quite prominent
NICH	Almost similar to IH	Almost similar to IH
VMs	Solid echogenic mass with phleboliths, often multispatial and compressible. Low flow or monophasic or no flow on CDUS	T1W heterogenous intermediate signal, no flow voids, T2 fast spin echo fat saturated or short T1 inversion recovery high signal intensity, T1W spin echo postgadolinium enhancement
LM	Variable multicystic, multispatial masses, with or without fluid or debris levels. No flow pattern on CDUS	T1W low to intermediate signal intensity, T2W high signal intensity, T1W postgadolinium, no enhancement except within septa
AVM	Clusters of vessels with no intervening well-defined mass. High flow (arterial flow) on CDUS. Arterial and venous signals from vessels in the lesions with arterializations of venous structure.	T1W and T2W sequences show serpiginous signal voids without a focal mass

US ultrasound, *CDUS* color Doppler ultrasound, *MRI* magnetic resonance imaging, *T1W* T1 weighted, *T2W* T2 weighted, *IH* infantile hemangioma, *RICH* rapidly involuting congenital hemangioma, *NICH* non-involuting congenital hemangioma, *VM* venous malformation, *LM* lymphatic malformation, *AVMs* arteriovenous malformations

Table 6 Salient histopathologic findings of vasoformative tumors (hemangioma and vascular malformations) [13]

Hemangiomas (proliferative phase)	Endothelial cell hyperplasia forming syncytial masses Thickened (multilaminated) endothelial basement membrane Ready incorporation of tritiated thymidine in endothelial cells Presence of large number of mast cells
Hemangiomas (involuting phase)	Less mitotic activity Little or no uptake of tritiated thymidine in endothelial cells Foci of fibrofatty infiltration Normal mast cell counts
Vascular malformations	No endothelial cell proliferation Contains large vascular channels lined by endothelium Unilamellar basement membrane Does not incorporate tritiated thymidine in endothelial cells Normal mast cell counts

Table 7 Sclerosing agents

Sclerosing agent	Important facts	Advantages	Disadvantages
Bleomycin	First used by Yura and coworkers in 1977 [25]	Can absorb systemically at very low levels, even if administered locally [25]	Development of fatal pulmonary fibrosis even in low doses [26] hyperpigmentation
Pingyangmycin	Chemical structure similar to Bleomycin A5, this anticancer drug can be extracted from gram +ve streptococci	Most effective in treating vascular malformations of size less than 5.0 cm and for superficial lesions. Percutaneously, it is very simple and effective	Allergic reactions, cutaneous or mucosal necrosis and sensory nerve or motor nerve injuries
5% Sodium morrhuate	Earlier, it was successfully used	May remain in lesion, causing sclerosis and involution for a longer duration	Irritating and has tendency for the induction of severe reactions like tissue necrosis
Absolute ethanol	Clinical application over the decades Used globally, even in complicated and extensive lesions [27]	Low cost, remarkable results, quick metabolism and lower recurrence rates [28]	Ethanol sclerotherapy is painful and requires general anesthesia. Facial palsy and allergy
Lauromacrogol aethoxysklerol or polidocanol	Most effective sclerosing agents with low risk of complications and contains 95% hydroxyl polyethoxydodecane with 5% ethyl alcohol	Injection technique is simple, safe and time-saving, painless, rarely allergic and well tolerated by patients	It may cause necrosis and ulceration, if solution leaks out into mucosa or skin
OK-432	OK-432, also called picibanil, is a biological preparation of lyophilized powder containing <i>Streptococcus pyogenes</i> Su strain cells (group A, type 3) treated with benzylpenicillin potassium [29]	No perilesional fibrosis [30] Possibility to perform multiple subsequent injections with additional shrinkage response Low complication rate Better aesthetics results [30]	The main disadvantage of OK-432 is that some lesions require more than one injection to shrink satisfactorily. Other complications include fever, allergy, erythema and swelling [31]
Detergent sclerosants (sodium tetradecyl, the most commonly used sclerosant, ethanolamine and polidocanol)	Detergents came into use in the 1930s They work by a mechanism known as protein theft denaturation	Addition of air results in a microfoam which is felt to be more effective than the bland solution. A reasonable dose limit for image-guided Sclerotherapy is 0.5 ml/kg or 30.	It is painful to inject, but effective and relatively nontoxic

The following algorithm was proposed by R. Mattassi, D.A. Loos and M. Vaghi for congenital vascular anomalies (Fig. 6).

These as well as other algorithms given proposed different approaches toward management of hemangioma and vascular malformations.

Management of Hemangiomas

Hemangiomas due to its biological behavior of proliferation followed by involution can be treated effectively with medications both systemic and intralesional.

As per the literature, around 90% infantile hemangiomas are small, focal and non-effective to function or aesthetics.

It is encouraged to have a close observation of these lesions during its proliferation [15]. Intervention is only necessary after involution phase, particularly in the presence of excess residual tissue, scarring or telangiectasia. Also, clear indications for intervention are also given when vital systems or structures such as airway or visual pathways are affected, risks of amblyopia, symptomatic or asymptomatic ulcerations, permanent disfigurement threatened by facial lesions or large lesions resulting in cardiac overload [16].

Beta Blockers

The serendipity of Leaute-Labraze et al. in 2008 resulting in efficacious use of propranolol is remarkable [17]. In their original landmark study, they have done treatment for

Table 8 Different embolic agents

Embolic agent	Important facts	Advantages	Disadvantages and limitations
Polyvinyl alcohol particles	Non-resorbable	Potential to occlude target vessel at desired point [34]	High Incidence of inflammatory and foreign body reactions Thrombosis and focal angionecrosis of the vessel wall [36]
Trisacryl gelatin microspheres	Non-resorbable hydrophilic, biocompatible nontoxic [37]	No tendency for aggregate formation	Fatal sepsis and diffuse necrosis
Polyvinyl Alcohol Microspheres	Resorbable water soluble	Mild or no inflammatory response	Higher compressibility of particles and early proximal occlusion
Gelfoam	Resorbable water-insoluble [38]		Ischemic and infectious complications Dermatome paresis
Oxycel/surgicel	Resorbable	No tissue reaction [39]	
Ethanol or absolute alcohol	Resorbable	Very effective Intravascular use	Untargeted Neighboring tissues and skin necrosis
Cyanoacrylate	Fast and efficient non-resorbable non-radiopaque [40]	Safe excellent control of the glue penetration Less painful	Special skill and experience required Ischemia or infarction of neighboring tissue
Detergent-type sclerosants	Non-resorbable	Not painful Effective in lesions with little flow Less severe allergic and inflammatory reactions [41]	Anaphylactic shock, temporary trismus, pleural effusion, pneumonia, and hemolytic reactions [42]
Coils and metallic embolization	Non-resorbable	Permanent desired occlusion	High cost Less flexible
Microcatheters	Non-resorbable Revolutionized interventional radiology	More flexible and small Easier placement at distal locations	Excellent fluoroscopy and digital imaging are required

Fig. 3 T2 weighted image showing venous malformation

hypertrophic obstructive cardiomyopathy in an infant with oral propranolol. Contrarily, Seigfried et al. warned for the side effects of propranolol which includes hypoglycemia,

bradycardia, hypotension, bronchospasm, CHF, sleep disturbance and hypothermia and recommended the close monitoring of infants having propranolol therapy [18]. But in many case series reports, almost 100% efficacy has been reported in arresting these lesions, and vice versa, therapy failure has also been reported indicating use of alternate treatment modalities. Although many centers including the authors' have introduced this therapy as first line of treatment, trials are still going on to prove its efficacy and more conclusive data will be forthcoming [19].

Steroids

Use of steroids in VM started in 1963 after an accidental find when a case of a thrombocytopenic child on systemic steroids (2–5 mg/kg/day) showed decrease in infantile hemangiomas [20]. In other large studies, when prednisolone was given in larger doses of 20 mg daily, it evidenced an 84% response after 2 months of initiating the therapy [21]. But 35% rate of side effects was also documented with this relatively high response rate. It is suggested that a mixture of triamcinolone 40 mg/ml and betamethasone 6 mg/ml in 1:1 ratio is to be injected

Fig. 4 64-slice spiral CT angiography (CTA)

1–2 ml intralesionally, response of which may be rapid, much more favorable and drastic in a matter of few days. Local complications, one may expect, are periocular ophthalmic artery embolization, fat atrophy, cutaneous hypopigmentation, full-thickness eyelid necrosis and calcification, but these are avoidable with skill adaptations and following proper technique with recommended dosage.

Fig. 5 Algorithm for management of vascular anomalies [11]

Vincristine

It is an alkaloid cancer chemotherapeutic agent which induces apoptosis by interference with mitotic spindle microtubule. It can thus be used as an alternative to steroids for infantile hemangioma because of steroid-sparing effects. Although it has been documented with side effects like hematologic toxicity, peripheral neuropathy, jaw pain and constipation, its favorable efficacious results overcome these known risks or side effects [22].

Interferon Alpha

It works as an anti-angiogenic agent with an advantage of steroid-saving effects. Although one case series documented regression of half or more lesions in 90% of cases treated by interferon alpha, this was associated with serious side effects of spastic diplegia and neutropenia [23]. Subsequently, this treatment modality was abandoned due to unacceptable high risk of neurotoxicity (10–30%) with its use, despite its favorable efficacious results [24].

Management of VM

This is largely dependent on the fluid dynamics of the lesion. Preliminary imaging with US Doppler will differentiate a high flow from a low-flow lesion. In low-flow lesions with large vascular channels and spaces in the head and neck region, total excision is a surgical challenge and misadventure. The philosophy of management then hinges on decompressing the lesion or partial elimination. This

Fig. 6 Algorithm for management of congenital vascular anomalies [14] **Fig. 7** Algorithm for management of vascular malformations [14] **Fig. 8** Corset suturing

can be achieved with administration of intralesional medicaments which may be sclerosing in nature (Fig. 7).

Intralesional sclerosants (Table 7)

Sclerotherapy has become the current mainstream treatment for venous malformation. It can be used alone or combined with surgery and/or laser therapy. For large lesions, multiple treatments are necessary. Recurrence is seen; this may possibly happen with some sclerosing agents that

Fig. 9 Intraoperative ECA control

incompletely treat the VMs being injected. The sclerosants commonly used are 5% sodium morrhuate, pingyangmycin (PYM), anhydrous ethanol and lauromacrogol.

The mechanism of action of these sclerosing agents is clearly understood and unique in destroying the blood vessel's endothelial cells, acceleration of protein coagulation in blood of lesions, formation of thrombosis, platelets adhesion promotion and causing vascular occlusion through thrombotic mechanisms.

Laser Treatment

One of the non-medicinal modalities of treatment employed in IH is laser. Several types of laser have been tried, including argon laser, Nd:YAG laser, carbon dioxide laser, fractional photothermolysis and pulsed dye laser (PDL).

Fig. 10 Endovascular embolization

Nd:YAG laser and KTP laser are most frequently used laser systems in the treatment of vascular malformations. Mechanism of action lies behind the ability of hemoglobin to absorb laser energy and generating a high temperature and that causes coagulation and immediate shrinkage of lesion. As these laser systems have the tendency to penetrate only 1–3 mm and to form a scar on nerves or skin of superficial lesion, their interstitial use has been recommended [32].

Surgical Management

Surgery continues to be a viable alternative to all of the above modalities. Although it yields nearly instantaneous

resolution of the lesion, it carries with it the risk of bleeding, despite the well-circumscribed nature of IH. Walker et al. demonstrated impressive clinical results following surgical removal of 12 visually significant hemangiomas, half of which had failed prior therapy; however, two of those cases also necessitated intraoperative blood transfusions due to hemorrhage [33]. Patients must be selected very carefully for excision accounting for the size, location and surgical risk to surrounding structures. Surgery is generally reserved for lesions that are refractory to less invasive treatments. Various techniques and technologies have aided in limiting and controlling intraoperative bleeding, rendering surgical excision a more favorable option in appropriate clinical scenarios.

The technique of intratumoral ligation was reported by Popescu in 1985 [34]. The aim of the treatment was to occlude by ligation the afferent and efferent vessels as well as the dilated vascular elements which make up the tumor mass. The technique required the use of large curved needles and strong suture material which would not cut out easily.

Corset Suturing

Similarly, the use of continuous vertical or horizontal mattress sutures helped compress the large venous compartments into smaller compartments which eventually replaces the vascular spaces with fibrous tissue. This technique was conveniently named Corset suturing by the

Fig. 11 Author's simplified algorithm

author. The use of long-standing PDS sutures helped in the fibrous transformation and helped reduce size of the lesion (Figs. 8, 9).

Managing High-Flow Lesions

High-flow lesions such as AM or AVM's had the risk of excessive hemorrhage intraoperatively and needed presurgical vascular control prior to its excision. Vascular control in high-flow lesions could be achieved by ECA control or endovascular embolization (Table 8).

The oldest form of surgical treatment for AVM's was proximal arterial ligation, which originated from Hunter's successful management of a popliteal aneurysm. This approach is rarely curative, however, and in fact runs a significant risk of compounding problems by putting a greater demand on collateral vessels. In this situation the collaterals not only sustain the original lesion but in the process of their subsequent enlargement act to increase its overall size. Judicious control rather than ligation was therefore the key.

Vascular Control (Understanding Embolization)

Embolization is defined as the “therapeutic introduction of various substances into the circulation to occlude vessels, either to arrest or prevent hemorrhaging; to devitalize a structure, tumor, or organ by occluding its blood supply; or to reduce blood flow to an arteriovenous malformation” [35].

Embolization has three therapeutic goals in the treatment of vascular malformations which are adjunctive goal, curative goal and palliative goal. Specific materials used for embolization are ethanol, cyanoacrylate, coils, polyvinyl alcohol, sodium tetradecyl sulfate, gelatin sponge and microspheres (Fig. 10).

Macrocoils and microcoils are of different sizes used for embolization. Platinum coils, type of microcoils, can be introduced into vascular system using microcatheters. These are highly radiopaque, thrombogenic and biocompatible. The potential disadvantages of this type of coil embolization are collateralization and proximal occlusion, making repeat embolization very difficult.

New interest has been inspired by the recent advances in terms of the course, its pathogenesis and successful treatment of infantile hemangiomas. One of the treatment modalities needs to be discussed here. As VEGF-A concentrations are high in infants with proliferative lesions, bevacizumab, an anti-VEGF antibody extracted from humans, has given remarkable results in early clinical trials in nosebleeds of patients suffering from hereditary hemorrhagic telangiectasia but currently clinical trials are still in process.

The management of vascular lesions starts from its very appearance and behavior. Identifying the type of lesion will determine its future management. There are several algorithms, as mentioned before, which help in the treatment protocol. The algorithm proposed by the author (Fig. 11) simplifies the decision making in the management of vascular anomalies.

In conclusion there are variables in managing individual vascular lesions and having a policy of “One size fits all” may be ill advised.

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