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Review

Vascular Anomalies Review of the Head and Neck for Physicians in Training

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Abstract

A basic understanding of vascular anomalies will aid physicians-in-training as they seek to properly diagnose and determine interventions for these patient presentations. The aim of this review is to create a resource for physicians in training that encompasses the most important clinical aspects of vascular anomalies. Vascular anomalies of the head and neck are divided into two categories: vascular tumors and vascular malformations. This review will first describe vascular tumors followed by vascular malformations and discuss major pathology found in both categories of vascular anomaly. The MEDLINE/PubMed database was searched for primary research and reviews discussing various vascular anomalies which include infantile hemangioma, congenital hemangioma, pyogenic granuloma, tufted angioma, kaposiform hemangioendothelioma, capillary malformations, lymphatic malformations, venous malformations, and arteriovenous malformations. We conducted the search from July 5, 2023, to March 21, 2024. Vascular anomalies are frequently found in pediatric populations and can persist into adulthood, making it important for trainees to identify them on physical exam. This developing field seeks to improve form, function, and quality of life for patients with vascular anomalies and often requires a multidisciplinary approach (i.e., otolaryngology, dermatology, genetics, plastic surgery, interventional radiology). Various medical and surgical treatment options are available. A basic knowledge of these anomalies will allow for accurate, early diagnosis and appropriate intervention which can ultimately improve patient outcomes.

Introduction

Vascular anomalies are diseases that involve abnormal development of blood vessels. Further understanding of their development and behavior have helped us differentiate these diagnoses into vascular tumors and vascular malformations.[3] Proper categorization of these diagnoses informs treatment, helps to identify potential complications, and aids patients' families to understand the natural course of these conditions. One of the most common vascular anomalies, infantile hemangioma, has been found to have an incidence as high as 4.5%,[1] making it crucial for physicians-in-training to recognize and understand the management of its pathology. While various articles and textbooks[2] intended for physicians are available to study vascular anomalies, a review of the common pathologies, their diagnosis and treatment targeted toward trainees does not yet exist. The purpose of this review is to provide a resource for physicians-in-training seeking to aid their patients in diagnosis and treatment decision-making. To achieve an appropriate diagnosis, trainees should recognize various pitfalls that may present. For example, vascular tumors can be mistaken for other tumors or infections. Additionally, different imaging modalities may show varying degrees of malformation that can be misinterpreted by radiologists. Therefore, an adequate history and physical exam should be performed to ensure a comprehensive understanding of the malformation.

Vascular Tumors

Vascular tumors are neoplasms originating from vessels that impact vessel organization and development. They are classified based on their malignant or local destruction potentials.[4] The following is a description of the epidemiology, pathophysiology, diagnosis, and management of some of the common vascular tumors.

Infantile Hemangioma

Epidemiology

Infantile hemangioma (IH) is the most commonly diagnosed soft tissue tumor and is present in 4-10% of children.[5] Though not typically present at birth, they generally become clinically apparent within the first month of life and continue to proliferate until 3-4 months of age.[5] Growth of IH ultimately plateaus, followed by an involution phase. Generally, 50% involution occurs by age 5 and 70% by age 7. Involution is often incomplete and can cause permanent skin changes or disfigurement.[5–7] The most common risk factors for IH include preterm birth, placental abnormalities, female gender, low birth weight, being a product of multiple gestations, and family history.[8]

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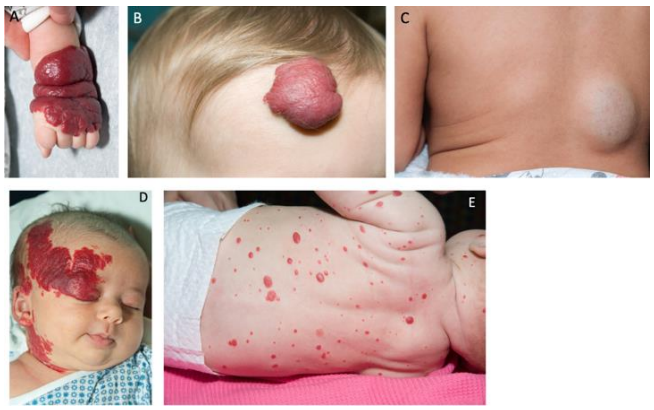
Figure 1. **Infantile Hemangiomas.**



Legend: A. Segmental upper extremity IH. B. Superficial hemangioma of the forehead. C. Deep hemangioma of the back. D. Segmental facial hemangioma, concerning for PHACES.[2] E. Hemangiomatosis. Multiple infantile hemangiomas (≥5) place patients at increased risk of hepatic hemangioma.

Pathophysiology

Although the pathophysiology of IH is unclear, growing evidence suggests a placental origin due to multiple similarities including growth pattern (9 months of rapid growth) and presence of similar molecular markers (GLUT1, transcriptome, microRNA profiles).[2,9–11]

Diagnosis

The most common location for IH to present is the head and neck, potentially impacting aesthetics, and functionality.[12] Clinical appearance varies broadly depending on location, depth, extent, and stage of evolution.[5] IH are classified by depth: superficial, mixed, or deep. Superficial IH present as bright red plaques, nodules, or masses. Deep IH appear as skin-colored masses with a bluish tint (***Figure 1, A-C***). Mixed IH are a combination of bright plaques and skin-colored masses.[13] An alternative classification is employed based on extent: IH arising from a single growth focus are categorized as localized/focal, while segmental IH presents in a linear or geographic cutaneous area.[13,14] IH is primarily diagnosed via history and physical exam. Imaging is used when uncertainties arise (i.e., distinguishing among vascular anomalies from more aggressive neoplasia, to delineate the extent of the tumor).[115] Generally, ultrasonography with doppler is the first modality of choice. An IH in the proliferative phase appears as a well-defined mass with non-homogeneous echostructure that demonstrates high-flow uniform vessel distribution.[16] While the mass is involuting, it will appear more hyperechoic due to the increased fat deposition and present with less vascular density.[16] MRI is helpful when determining depth of involvement or in cases of visceral involvement. IH are well-circumscribed masses that are isointense on T1-weighted sequences, hyperintense on T2-weighted sequences, and show avid post-contrast enhancement without arteriovenous shunting (as seen in arteriovenous malformations).[17]

Management and Prevention

It is important to keep in mind that the most common treatment for hemangiomas is observation, as they commonly undergo involution. Treatment is reserved for complicated and high risk IH cases. Complications are typically seen during the proliferative phase for large IH located on the face including bleeding, airway compromise, visual compromise, ulceration, pain, difficulty feeding or risk permanent disfigurement.[18] See ***Table 1*** for special clinical considerations.

First line therapy for IH is oral propranolol, a nonselective beta-adrenergic blocker.[19] Contraindications to propranolol treatment should be considered including cardiogenic shock, sinus bradycardia, heart failure, bronchial asthma, allergy, and history of hypoglycemia. However, these conditions are all uncommon in infants. Dosing of the propranolol for IH is between 1 and 3 mg/kg/day separated into 2-3 doses with 6 hours between doses. Cardiac consult should be obtained before therapy initiation if there is concern for cardiac disease. Patients should be monitored for side effects including bradycardia, hypoglycemia, hypotension, and bronchospasm. These are not common and typically not life threatening.[19,20] Historically, corticosteroids were the first-line medical therapy for IH. Intralesional corticosteroids can still be used to decrease the size of small IH, but propranolol is preferred.[21] Another treatment option is pulsed dye laser (PDL). This technique can be used for ulcerated IH or residual visible vascularity after IH involution. Finally, surgical excision may be performed for patients who cannot undergo systemic therapy. Additionally, surgery may be performed if propranolol is ineffective, for cosmetic deformity, or caregiver preference.[2] See ***Table 2*** for a summary of indications for treatment of IH.

Segmental IH and Syndromes

PHACES Syndrome

PHACES syndrome stands for *p*osterior fossa intracranial abnormalities, *h*emangiomas, *a*rterial abnormalities, *c*ardiac defects and coarctation of the aorta, *e*ye abnormalities, and *s*ternal clefting.[22] This syndrome is associated with cervicofacial IH (***Figure 1C***). Patients with PHACES are more likely to have an airway IH and abnormal cerebral vasculature, a risk factor for stroke.[23]

SACRAL and PELVIS Syndromes

PELVIS is another syndrome associated with hemangiomas that stands for *p*erineal hemangiomas, *e*xternal genital malformations, *l*ipomyelomeningocele, *v*esicorenal abnormalities, *i*mperforate anus, or *s*kin tags.[24] Furthermore, SACRAL syndrome is associated with angiomas in the lumbosacral region and stands for *s*pinal dysraphism with *a*nogenital, *c*utaneous, *r*enal, and urologic anomalies, associated with *a*ngiomas in the *l*umbosacral region.[25]

Hemangiomatosis

Infants with five or more cutaneous IH (***Figure 1E***) have an increased risk of hepatic IH which prompts screening imaging via abdominal ultrasound.[26] Though often clinically benign, hepatic IH can be complicated by bleeding, congestive cardiac failure, hypothyroidism, and abdominal compartment syndrome.[27] Hepatic IH generally undergo involution similar to cutaneous IH.[27]

Table 1. **Special Considerations for Infantile Hemangiomas.**

Clinical Presentation	Concern	Workup
Hemangiomatosis (>5)	Screen for intrahepatic/visceral hemangiomas	Abdominal ultrasound.[82] Consider thyroid function tests in multifocal and diffuse hepatic hemangiomas.[7]
Segmental facial hemangioma	Rule out PHACES syndrome [posterior fossa brain malformations, segmental cervicofacial hemangioma, arterial abnormalities (vessel abnormalities in the head or neck), cardiac abnormalities or aortic coarctation, eye abnormalities, sternal clefting].[83]	Ophthalmology referral. Echocardiogram, cardiology referral. MRI/MRA of head, neck, and arch.[84]
Midline lumbosacral hemangiomas	Rule out SACRAL syndrome (spinal dysraphism, anogenital anomalies, urogenital anomalies).	Referral to dermatology urology, nephrology, neurology, and/or neurosurgery based on clinical needs.
Large hemangiomas, particularly in the liver	Risk of high-output cardiac failure and hypothyroidism.	Cardiology evaluation and TFT if concern.
Beard distribution segmental hemangioma (facial lower third and neck) or central neck	Rule out airway hemangiomas.[85]	Otolaryngology evaluation to consider airway evaluation.
Ulceration and bleeding		
Perineal, axilla, neck	Risk of ulceration related to friction.	Monitor clinically and treat as needed. Treatment can include topical antibiotic ointment, wound care, and on occasions culture and oral antibiotics.
Compromise vital functions		
Periorbital	Can cause astigmatism, strabismus, or amblyopia.	Ophthalmology referral.
Perioral, lip	Feeding difficulties.	Otolaryngology evaluation and feeding therapy consultation if appropriate.
Airway	Becoming symptomatic (stridor, feeding difficulties, etc.) and can become life-threatening if airway obstruction progresses.	Otolaryngology evaluation for airway assessment.
Nasal	Nasal obstruction.	Otolaryngology evaluation for airway assessment.
Slow Involution or Deformity		
Parotid	Can be deep and without cutaneous manifestations.	Ultrasound can aid in diagnosis.[86] Risk to the facial nerve during surgery. Recommend facial nerve monitoring ±mapping.[87]
Cosmetic Concern		
Nasal tip, ear, large facial	Social impact from facial disfigurement.	Lower threshold to treat.

Congenital Hemangioma

In contrast to IH, congenital hemangiomas are present at birth (***Figure 2A***) and are GLUT-1 negative despite having similar histology.[28] Congenital hemangiomas are divided into two subtypes: rapidly involuting congenital hemangioma (RICH) and non-involuting congenital hemangioma (NICH). RICH will resolve over the first year of life while NICH do not generally resolve spontaneously.[29] Thus, NICH can treated with surgery or laser.

Pyogenic Granuloma

This benign vascular tumor, also known as lobular capillary hemangioma, can arise in both children and adults (***Figure 2B***). Children will generally develop pyogenic granuloma in the head and neck region while adults often develop lesions on the trunk.[30,31] Mucosal pyogenic granulomas also occur in about 2% of pregnancies between the second and fifth months.[32] These tumors are rapidly growing, exophytic, red-colored papules that bleed commonly. Similar to congenital hemangioma, they are GLUT-1 negative on histopathology. The treatment is surgical, either by complete excision or punch excision with curettage or electrodesiccation of the feeding vascular stalk.[33]

Kaposiform Hemangioendothelioma And Tufted Angioma

Both tufted angioma (TA) and kaposiform hemangioendothelioma (KHE) are vascular tumors with a lymphatic component. TA is localized and noninvasive, however KHE invades nearby tissue.[34] KHE presents clinically as violaceous nodules and demonstrates tissue invasion on imaging (***Figure 2C***). TA present similarly, may or may not involve skin, and do not invade local tissue. Both of these conditions are associated with the Kasabach-Meritt phenomenon (KMP), a consumptive coagulopathy characterized by thrombocytopenia, hypofibrinogenemia, and anemia.[35] Surgery and medical therapy (e.g., sirolimus, vincristine) can be employed for treatment of both conditions.[36]

Vascular Malformations

Vascular malformations occur due to errors in the initial development of blood and lymph vessels and tend to be present at birth and grow with the child.[37] Vascular malformations can present with complex clinical presentations and management.[37] See ***Table 3*** for a summary of vascular malformations by common complications and treatment.

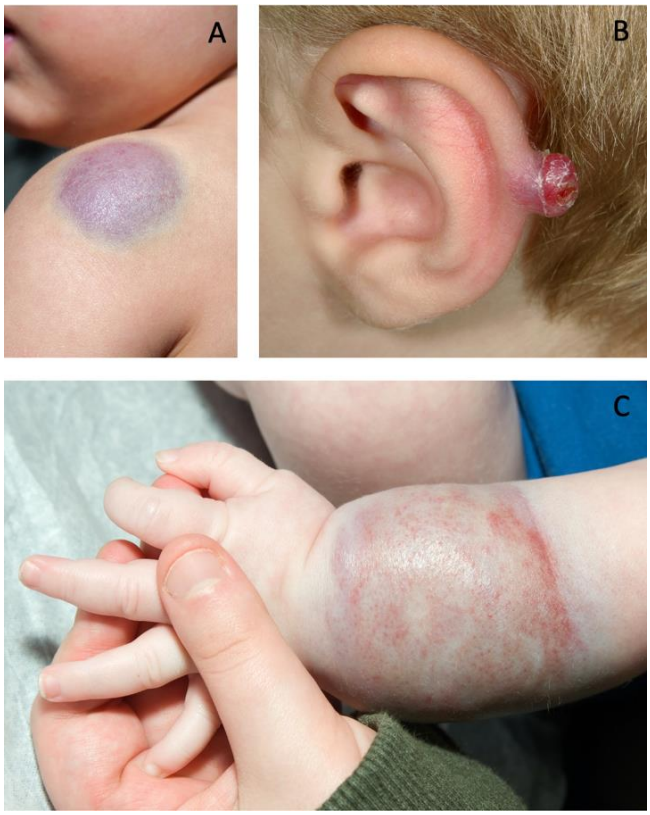
Capillary Malformations

The most common vascular malformations involve capillaries. Capillary malformations (CM) (***Figure 3A***) encompass a wide range of cutaneous malformations linked by their abnormal capillary morphology on histology.

Nevus simplex

This CM, commonly found in Caucasian infants,[38] has a wide range of names including salmon patch, angel's kiss (when located on the glabella), and stork bite (when located on the nape of the neck). These blanching erythematous macules present with indistinct borders and typically resolve over months to years. They are occasionally linked with Beckwith-Wiedmann syndrome, Nova syndrome, and macrocephaly-capillary malformation syndrome (MCAP).[2]

Figure 2. **Vascular Tumors.**



Legend: A. Congenital hemangioma. Blue-purple deep mass with surrounding pale halo. B. Pyogenic granuloma.[2] Pedunculated, erythematous growth that commonly ulcerates and presents with bleeding. C. Kaposiform hemangioendothelioma. Violaceous skin discoloration with palpable mass underlying.

Table 2. **Indications for Treatment of Infantile Hemangiomas.**

Indication	Locations to consider	Special considerations
Compromise vital functions	Periorbital	Can cause astigmatism, strabismus, or deprivation amblyopia. Consider ophthalmology referral.
	Perioral, lip	Can result in feeding difficulties. Consider feeding therapy and/or otolaryngology evaluation.
	Airway	Can present with stridor or feeding difficulties and may become life threatening. Recommend otolaryngology evaluation.
	Nasal	Can disrupt nasal airflow in obligate nasal breathers. Consider otolaryngology evaluation.
Slow Involution or Deformity	Parotid	Can be deep and without cutaneous manifestations so ultrasound aids in diagnosis. [80] Risk to the facial nerve during surgery. Recommend facial nerve monitoring +/- mapping. [81]
Cosmetic Concern	Nasal tip, ear, large facial	Can result in permanent disfigurement.

Port Wine Stain and Sturge Weber Syndrome

One CM that does not resolve is the port wine stain (PWS), also known as nevus flammeus. PWS will grow in conjunction with the child and present as pink to dark red patches with well-demarcated borders (***Figure 3B***). Also, it may thicken or become darker over time and is associated with soft tissue and bone hypertrophy, gingival hyperplasia, and dental anomalies.[39] Around 10% of patients with facial PWS have Sturge-Weber syndrome (SWS).[40] SWS is the association of facial CM with leptomeningeal angiomatosis and glaucoma. Some patients with SWS may develop epilepsy. MRI with contrast is the gold standard for diagnosis.[41]

Telangiectasias and hereditary hemorrhagic telangiectasia

Telangiectasias are small, dilated capillary vessels that present as red macules with stellate shape and a pale halo. Isolated, they are nonsyndromic; however, multiple telangiectasias together may indicate hereditary hemorrhagic telangiectasia (HHT). This autosomal dominant disorder presents with telangiectasias affecting multiple systems, arteriovenous malformations (AVM), and vascular dysplasia. This disorder most commonly presents at age 12 and its severity increases with age. HHT can lead to life-threatening bleeding.[42]

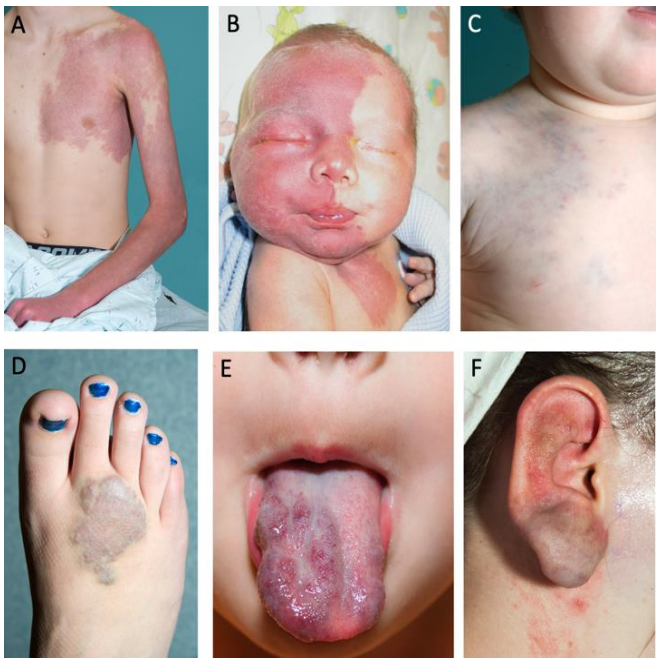
Treatment

CM treatment is tailored to each individual presentation and the needs of the patient. Generally, a balance is sought between intervening before the lesion thickens and becomes nodular, while minimizing risks of anesthetics (generally after 6 months of age).[2] First-line treatment for CM is pulsed dye laser with or without topical sirolimus.[43,44]

Lymphatic Malformations

Lymphatic malformations (LM) are a subset of vascular malformations that present with abnormal lymphatic vessel morphology. LM occur in approximately 1 in 4000 live births. Prior nomenclature used to describe LM included cystic hygroma and lymphangioma, however these terms imply a watery tumor rather than a collection of ill-formed lymphatic vessels and are no longer in use. Some LM such as posterior cervical LM or extensive cervicofacial LM can be diagnosed prenatally. Posterior cervical LM are sometimes associated with syndromic conditions and regress by birth.[45] Ventral, isolated LM can also be diagnosed prenatally or shortly after birth and commonly present in the neck and face. In utero, LM may be associated with polyhydramnios if swallowing function is impaired and/or may have airway compromise at birth. A LM with airway obstruction requires delivery planning and a possible EXIT (ex-utero intrapartum treatment) procedure.[46,47]

***Figure 3*. Vascular Malformations.**



Legend: A. Capillary malformation of the left upper extremity and chest. B. Segmental facial port wine stain (PWS) associated with Sturge Weber syndrome (SWS).[2] Venous malformations. C-E: cutaneous presentation of venous malformations of the chest (C) and foot (D) seen as violaceous discoloration of the skin. E: mucosal venous malformation of the tongue. F. Arteriovenous malformations of the ear.

Diagnosis and classification

LM are diagnosed via a combination of history, physical exam, and imaging. Depending on the extent of the malformation, MRI and CT are commonly used modalities that can aid in the diagnosis of LM and selection is based on preference. The authors prefer MRI given the better soft tissue delineation and avoidance of radiation in children. If radiographic cystic spaces in the LM are >2cm in diameter they are classified as macrocystic. Those less than 2cm in diameter are classified as microcystic. LM that presents in the head and neck are classified using de Serres stages, which is based on laterality and location relative to the hyoid.[48] More than 80% of LM are stage I-III with unilateral involvement.

***Table 3*. Complications and Treatment for Vascular Malformation Categories.**

Vascular malformation	Complications	Treatment
Capillary malformations	Generally cosmetic (see ***Figure 3A***), can be associated with Beckwith-Wiedemann, Nova, macrocephaly-capillary malformation, and Sturge Weber syndromes.[9, 36]	Pulsed dye laser and topical sirolimus.[9]
Lymphatic malformations	Impaired swallowing function, airway compromise, cosmetic.[42, 43] Can be associated with CLOVES and KTS syndromes.[48]	Many resolve spontaneously.[45] Sclerotherapy, surgical excision, medications (sirolimus; aspirin and PI3K inhibitors under investigation).[51]
Venous malformations	Pain and localized intravascular coagulopathy which may predispose to disseminated intravascular coagulopathy. Associated with Klippel-Trenaunay syndrome.[54]	Perioperative treatment with low molecular weight heparin (LMWH) and surgery. Medical treatments include aspirin and sirolimus. Compression may be helpful with extremity presentations.[62]
Arteriovenous malformations	Deformity, bleeding, and high output cardiac failure. Other complications include infiltration of surrounding tissues and increased size during pregnancy, puberty, and trauma.[9, 65]	Medical management with doxycycline, sirolimus, and trametinib for severe cases. Intralesional bleomycin has shown variable results. Other options include ablation of draining veins with flashlamp-pumped pulsed dye laser (FPDL) and Nd:YAG laser. Surgical resection for focal AVM.[9, 72, 73]

Generally, unilateral LM do not cause functional compromise and can resolve spontaneously.[49] Large, bilateral LM typically cause functional compromise and do not resolve spontaneously.[49] LM are not hereditary, rather, they are associated with somatic mutations in *PIK3CA*, an oncogene associated with cancer and other overgrowth disorders.[50,51]

Associated syndromes: CLOVES and KTS

LM are not generally associated with a syndrome and present in isolation but may be associated with other *PIK3CA*-related overgrowth syndromes (PROS). PROS associated with LM include CLOVES syndrome (congenital lipomatosis, overgrowth, vascular malformations, epidermal nevi, and skeletal anomalies) and Klippel-Trenaunay syndrome (KTS) (port wine stains, venous malformations, LM, and overgrowth).[52] Overgrowth in CLOVES syndrome can be very severe and generally involves multiple limbs and possibly trunk. Klippel-Trenaunay syndrome generally only has overgrowth of a single extremity.[53]

Management and Prevention

Treatment of LM is typically pursued in response to functional or aesthetic compromise. Many resolve spontaneously, especially unilateral macrocystic LM.[49] If infected, it typically responds well to systemic antibiotics and corticosteroids. Another treatment that may be pursued for macrocystic LM is sclerotherapy, a procedure in which interventional radiology drains the associated fluid and injects a sclerosing agent.[54] This procedure is often required multiple times to achieve a sustained result. Surgical excision is also an option. The most commonly used medication for LM is sirolimus, an MTOR inhibitor. Aspirin and targeted PI3K inhibitors (alpelisib) are undergoing investigation as potential treatments for LM.[55]

Venous Malformations

Epidemiology

Around 1-4% of individuals have a venous malformation (VeM). VeM commonly present in the head and neck, generally on mucosal surfaces or within muscles.[56] The next most common area for a VeM to arise is the upper and lower extremities. VeM often clinically present at puberty, but most are present even at birth and grow proportionately with the child.[57] VeM are visualized as a mass with a bluish hue overlying the skin but may also present without the blue hue (***Figure 3C-E***). Mucosal VeM typically presents with blue-purple discoloration.

Diagnosis

A combination of clinical history, physical exam, and imaging is used for diagnosis. Choosing the imaging modality depends on the location, patient age, and ability to receive contrast. Ultrasound, CT, and MRI may all provide valuable information. Many VeM present with pain and there may also be localized intravascular coagulopathy (LIC) which can occasionally predispose to disseminated intravascular coagulopathy (DIC). LIC is most commonly seen in large lesions (>10 mL), when phleboliths are present, in multifocal disease, and when associated with Klippel-Trenaunay syndrome.[58] Elevated D-dimer levels in a patient with VeM has a high specificity (>97%) for LIC.[59]

Etiology and associated syndromes

VeM can arise sporadically or hereditarily. Various mutations are linked with VeM: sporadic venous malformations are associated with somatic mutations in *TIE2/TEK* and *PIK3CA*,[60,61] some familial VeM are associated with mutations in *RASA1*,[62] and hereditary venous malformations, also known as familial venous malformation cutaneo-mucosal (VMCM), are associated with inherited mutations in *TIE2/TEK*.[63] Glomuvenous malformation is a hereditary disease associated with inherited mutations in *GLMN* (glomulin) with a distinct pathologic phenotype that involves glomus cells, a type of immature vascular smooth muscle cell.[64] Blue rubber bleb nevus syndrome is a form of VeM that presents with compressible mucocutaneous VeM and involves the visceral organs, most commonly in the gastrointestinal tract.[65]

Management and Prevention

Patients with bothersome VeM or who present with LIC are candidates for treatment. A patient with VeM and elevated D dimer should be treated perioperatively with LMWH for any sedated procedure as well as 14 days pre and post procedure.[66] Medical therapies include aspirin and sirolimus. For patients with VeM on the extremity, compression can help to relieve symptoms though it is not known whether it helps to prevent LIC.

VeM often requires invasive therapy such as laser therapy, sclerotherapy, and surgery. Laser therapy can include PDL or Nd:YAG for superficial lesions. Sclerotherapy is also available but often requires multiple procedures to achieve an effect.[67] Surgical excision can be performed, often with glue embolization in which interventional radiology injects the VeM with n-BCA glue immediately prior to resection to decrease the risk of bleeding and allow for more thorough resection.[68]

Arteriovenous Malformations

Epidemiology

Arteriovenous malformations (AVM) involve atypical connections between arteries and veins (Figure 3F). AVM have a central nidus, one or more arteries that feed the nidus, and one or more veins that drain it. AVM can be intracranial or extracranial. When extracranial they are most likely to occur in the head and neck. AVM are present at birth but commonly increase in size during pregnancy, puberty, or trauma.[2,69] Contrary to other vascular malformations, AVM commonly infiltrate adjacent tissue. Common complications include deformity, bleeding, and high output cardiac failure if large in size. If extracranial, the Schobinger classification can categorize AVM based on presentation and exam. Intracranial AVM are classified based on size and morphology with the Spetzler-Martin scale.[70] Imaging is helpful for diagnosis and requires arteriography or MR-angiogram. Smaller AVM with a single feeding artery may be treatable, but effective therapeutic treatments for diffuse AVM are more challenging.

Etiology and associated syndromes

AVM are generally sporadic and can be associated with somatic mutations in *KRAS*, *BRAF* and *MAP2K1*.[71–73] Some forms of AVM can be inherited. For example, CM-AVM is an autosomal dominant disorder, commonly associated with mutations in RASA1, that presents with multiple round to oval vascular stains with a vasoconstrictive halo.[62] Around one third of individuals with a CM-AVM also have an AVM in the brain, spine, bone, skin, or soft tissue.[74] One subtype of CM-AVM is Parkes-Weber syndrome which can present with arteriovenous fistula (AVF) and lower extremity limb overgrowth. Hereditary hemorrhagic telangiectasia (HHT), a syndrome associated with mutations in the TGF-beta pathway, is most commonly associated with pulmonary AVM, but may also present with gastrointestinal tract, liver, spine, or brain AVM.[75]

Treatment

AVM differ from VeM and LM in that they should not be observed. They warrant aggressive therapy given their propensity to infiltrate surrounding tissue. However, functional and aesthetic outcomes associated with treatment must be taken into consideration. Medical management has seen some response with doxycycline (matrix metalloproteinase inhibitor), sirolimus (MTOR inhibitor), and trametinib (MAP2K1 inhibitor) for severe AVM.[76,77] Intralesional bleomycin has also been trialed with variable results. Other options include flashlamp-pumped pulsed dye laser (FPDL) and Nd:YAG laser to ablate superficial draining veins.[2]The primary treatment of AVM is embolization and surgical resection. Embolization can be done with various agents including onyx, ethanol, and n-BCA (glue). Onyx and n-BCA embolization are generally used as an adjunct to surgery in both intracranial and extracranial AVM.[78] Surgical resection is most successful in focal AVM and is generally performed after or in conjunction with embolization.[79]

Discussion

This review of vascular anomalies has discussed the pathogenesis, diagnosis, and management of the two main categories: vascular tumors and vascular malformations. Vascular anomalies are prevelant[5] and recognizing them clinically and understanding management will aid trainees in their clinical efforts. Various reviews of vascular anomalies have previously been published. For example, a brief review by Cox et. al[80] organized vascular anomalies by subtype to help clinicians develop a clear understanding of the clinical aspects, diagnostic tools, imaging modalities, and options for interventions available. More extensive reviews, such as Perkins and Balakrishnan,[2] have been developed to provide a comprehensive resource of current evidence-based management of head and neck vascular anomalies for providers. The purpose of this review was to provide an educational tool for physicians-in-training seeking to gain an understanding and assist in the recognition of these pathologies, as well as to offer an insight into the various treatment methods.

This review has several strengths, including a concise delineation of common vascular anomalyies, visual images to support recognition, and its originality as the first vascular anomalies review with physicians-in-training as the target audience. Limitations, however, do exist. Our understanding of the epidemiology of vascular anomalies continues to develop and studies evaluating the effectiveness of treatment options continue to be published. Thus, our understanding and explanations of certain vascular anomalies is extensive, while less is known about more recently identified pathologies. This understanding will continue to develop as future research will likely focus on the refinement of genetic and molecular therapies, improved diagnostic techniques, and the development of personalized treatment protocols.[3,19,81]Future reviews of vascular anomalies should continue to be published in an effort to provide up-to-date resources for this developing field.

Conclusion

This review seeks to describe the epidemiology, diagnosis, and treatment of the most common vascular anomalies. It is intended for physicians-in-training seeking an up-to-date resource for understanding and managing vascular anomalies that is appropriate to their training level. As medical school curricula vary in the depth of coverage of vascular anomalies, this review will allow students to have a comprehensive, trainee-level guide. As studies continue to further our knowledge of these pathologies, updated reviews should continue to be published to provide up-to-date resources for trainees and clinicians.

Summary – Accelerating Translation

Title: Vascular Anomalies Review of the Head and Neck for Physicians in Training

The main problem to solve: Limited training on vascular anomalies of the head and neck is given to medical students. However, a basic understanding of vascular anomalies can aid physicians-in-training as they seek to properly diagnose and determine interventions for these patient presentations.

Aim of this review: To create a resource for physicians in training that encompasses the most important clinical aspects of vascular anomalies.

Methodology: Current evaluation, diagnosis, and treatment for vascular tumors and vascular malformations were compiled into a trainee-level resource through literature review.

Results: Current evaluation, diagnosis, and treatment guidelines were described for infantile hemangioma, segmental IH and syndromes, pyogenic granuloma, kaposiform hemangioendothelioma and tufted angioma, capillary malformations, lymphatic malformations, venous malformations, and arteriovenous malformations.

Conclusion: A basic knowledge of these anomalies will allow students to assist in accurate, early diagnosis and appropriate intervention of vascular anomalies. This can ultimately improve patient outcomes.

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Conceptualization: CMA, KBZ, JBV. Writing - Original Draft: CMA, KBZ, JBV. Writing - Review Editing: CMA, KBZ, JBV.

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