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INVITED REVIEW

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Current classification of vascular anomalies of the head and neck

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Abstract

Vascular anomalies of the head and neck comprise a wide spectrum of phenotypically diverse lesions. Optimal diagnosis and management of these lesions are critically dependent upon establishment of uniform and well-defined histopathologic, clinical, and radiological criteria, but these remain subject of debate. In this paper, we describe the International Society for the Study of Vascular Anomalies classification scheme, which was first published in 1996 and updated in 2014. The strength of this proposal rests on its distinction between vascular malformations and tumors, and is responsible for its wide adoption. This paradigm serves as a developing platform for diagnosis, inter-collegial communication, and treatment, and adhering to it will help clinicians to improve the management of vascular anomalies.

KEYWORDS
classification, head and neck, vascular anomalies

1 | INTRODUCTION

Vascular anomalies comprise a wide spectrum of lesions, ranging from simple birthmarks to large, disfiguring tumors. In the anatomically dense and functionally rich head and neck area, they represent unique diagnostic and therapeutic challenges due to esthetical issues and functional impairment, both of which can be preexistent or treatment-induced. Adequate management is critically dependent upon accurate diagnostic classification. Previous studies have shown that inaccurate designation of vascular anomalies is associated with an increased risk of erroneous management.[1] For example, traditional indiscriminate use of terms such as angioma and hemangioma has caused inappropriate grouping of vascular anomalies which in fact represent separate clinicopathologic entities.[1,2] Because of their heterogeneity, rarity and diverse diagnostic criteria, rates of misdiagnosis of vascular anomalies remain high.

Several classifications of vascular anomalies have been proposed over the course of multiple decades. Historically, vascular anomalies were grouped based on clinical phenotype, specifically the caliber of the vessels from which they arose, as well as the contents of these vessels (lymph or blood). Lesions originating from blood vessels were named hemangiomas. This group was subdivided into capillary hemangiomas, strawberry hemangiomas, and cavernous hemangiomas, based on vessel caliber. Lesions that contained lymph fluid were named lymphangiomas or cystic hygromas. This classification system was replaced in 1863 by Virchow,[3] who reclassified vascular anomalies based on histologic criteria, including their microscopic vascular architecture and divided them into angioma simplex, angioma racemosum, and angioma cavernosum. Over subsequent decades, the range of vascular pathologies increased, and outgrew existing classification schemes. This prompted an extension of taxonomy, which further complicated classification and nomenclature.[2] Significant improvement in uniformity was rendered in 1982 by Mulliken and Glowacki,[4] who proposed differentiating vascular anomalies into tumors and malformations, based on the presence or absence of endothelial mitotic activity. This paradigm shift classified hemangiomas as neoplastic but benign tumors marked by increased proliferation. On the other hand, vascular malformations were defined as representations of a non-neoplastic, localized, and relatively static defect in vascular morphogenesis.[4] The Hamburg classification system, first established in 1988, proposed to further subdivide vascular malformations based on the timing of the interruption in embryonic development.[5,6] This classification allowed for differentiation between truncular and extratruncular lesions. The term truncular was used to indicate lesions of

TABLE 1 ISSVA Classification (2014) basic framework

Vascular anomalies				
Vascular tumors	Vascular Malformations			
	Simple	Combined	Of major named vessels (aka channel/truncal-type)	Associated with other anomalies
Benign • Infantile hemangioma • Congenital hemangioma (RICH, NICH, PICH) • Tufted angioma • Spindle cell hemangioma • Pyogenic granuloma • Others Locally aggressive or borderline • Kaposiform hemangioendothelioma • Papillary intralymphatic angioendothelioma (PILA), Dabska tumor • Kaposi sarcoma • Retiform hemangioendothelioma • Composite hemangioendothelioma • Others Malignant • Angiosarcoma • Epithelioid hemangioendothelioma • Others	Capillary malformations (CM) Lymphatic malformations (LM) Primary lymphedema Venous malformations (VM) Glomuvenous malformations (GVM) Arteriovenous malformations (AVM) Arteriovenous fistulas (AVF)	CM + VM CM + LM CM + AVM LM + VM CM + LM + VM CM + LM + AVM CM + VM + AVM CM + LM + VM + AVM	See details on ISSVA website	See list and known genetic associations on ISSVA website

axial, larger vessels, originating from embryonic defects during truncal vascular development. The term extratruncular indicated lesions of smaller, embedded vessels, originating from defects in earlier embryonic stages. Both models were used by the International Society for the Study of Vascular Anomalies (ISSVA) to develop the most comprehensive classification system to date. First published in 1996, and updated in 2014, this classification now includes embryologic, anatomic, and histologic features, combined with genetic advances in characterization of vascular anomalies.[7] A summary of the 2014 ISSVA classifications is given in Table 1. Although the World Health Organization (WHO) attempted introduction of a vascular tumor classification of its own in 2002, this classification increased confusion rather than that it provided any meaningful improvement. We believe that the 2014 ISSVA guidelines represent the most useful and comprehensive classification of vascular anomalies to date and have chosen its description as the topic of this paper. A summary of relevant characteristics of vascular anomalies discussed in this paper is given in Table 2.

2 | VASCULAR TUMORS

Vascular tumors, defined by microscopic or molecular evidence of abnormal endothelial cell proliferation, are uncommonly present at birth. Based on cellular behavior, they may be classified as benign, locally aggressive/borderline, or malignant. The most common are benign hemangiomas which are subdivided into infantile hemangiomas and congenital hemangiomas. These tumors are described in detail below. Other vascular tumors include the borderline and locally aggressive vascular tumors, the most well known of which are the immunodeficiency-associated Kaposi sarcomas. Malignant vascular tumors are extremely rare. The most common vascular malignancy is angiosarcoma, an aggressive sarcoma marked by high rates of local spread along blood and lymph vessels and high rates of distant metastasis. These features are responsible for high rates of treatment failure, and dismal outcomes are the rule rather than the exception.

2.1 | Infantile hemangioma

Infantile hemangiomas (IHs) are the most commonly encountered tumors in infants, affecting 4 to 5% with a preponderance for females.[8,9] These benign lesions were previously named juvenile hemangioma or cellular angioma of infancy. IHs present shortly after birth, grow rapidly in the first year, then regress spontaneously due to a massive and relatively abrupt apoptosis. IH endothelial cells express the immunohistochemical marker glucose transporter 1 (GLUT-1),

TABLE 2 Comparative characteristics of vascular anomalies

Name	Type	Timing of presentation	Growth pattern	Involution pattern	Appearance	Location	Organ involvement	GLUT-1	Associated syndromes
IH	Vascular tumor	Shortly after birth	Rapid growth in first year	Spontaneous slow involution	Erythematous plaques (superficial IH), pale or dusky blue nodularities (deep IH), or a combination of both (mixed lesions)	Head and neck (60%), may be anywhere	Liver, intestine, and airway	+	PHACE syndrome and LUMBAR syndrome
CH	Vascular tumor	At birth	Congenitally fully formed, or proportional growth after birth	Rapid and complete (RICH), partial and incomplete (PICH), or no involution (NICH)	Heterogeneous, rounded or oval, pink coloration, superficial telangiectasia, or peripheral veins	Head and neck, limbs	Liver and other viscera (less common)	−	−
CM	Vascular malformation	At birth	Proportional	No involution	Most common type CM (PWS): flat, red, or pinkish-violaceous	Face (90%), may also appear in the neck, trunk, arm, leg, and hand	Leptomeninges, choroid plexus	−	Sturge–Weber syndrome, Klippel–Trenaunay syndrome, capillary malformation-arteriovenous malformation syndrome, and macrocephaly-capillary malformation syndrome
LM	Vascular malformation	Usually present at birth, may present within the first 2 years	Proportional	No involution	Soft, compressible masses, when involving the (epi) dermis or mucosa, surface vesicles may form, which bleed and become dark	Mostly in head and neck area	Airway	Negative	Klippel–Trenaunay syndrome
VM	Vascular malformation	At birth, may not become evident until they cause symptoms or deformity	Proportional	No involution	Bluish, compressible masses, sometimes ulceration, and bleeding	Anywhere, mostly head and neck	Anywhere	Negative	Klippel–Trenaunay syndrome

TABLE 2 (Continued)

Name	Type	Timing of presentation	Growth pattern	Involution pattern	Appearance	Location	Organ involvement	GLUT-1	Associated syndromes
AVM	Vascular malformation	Present at birth, around half are at that time visible	Proportional	No involution	Continue to grow throughout life, leading to tissue destruction	Anywhere, spinal cord, brain, head and neck, limbs, trunk, and viscera	Anywhere, mostly brain and lungs	Negative	Rendu Osler Weber syndrome, capillary malformation-arteriovenous malformation syndrome

Abbreviations: AVM, arteriovenous malformation; CH, congenital hemangioma; CM, capillary malformation; IH, infantile hemangioma; LM, lymphatic malformation; VM, venous malformation.

which helps to distinguish IHs from other vascular anomalies.[10] Both low-birth-weight and Caucasian infants have proven to be at increased risk for IH development for unknown reasons.[11,12] IHs can usually be diagnosed clinically as they appear mostly in the skin and soft tissues. Approximately, 60% of IHs occur in the head and neck area. Extracutaneous IHs may involve the liver, intestine, and airway. Around 50% of infants diagnosed with an airway IH will also have a cutaneous IH. Conversely, only 1%–2% of children with cutaneous IHs will have IHs affecting the airway.[13,14] IHs are, amongst others, classified based on the involvement of the skin, subcutaneous tissue, or both (superficial, deep, or mixed, respectively). Superficial IHs may present as erythematous plaques, whereas deep IHs present as pale or dusky blue nodularities. Mixed lesions display a combination of both. IHs may also be categorized as focal, multifocal, segmental, or indeterminate. Focal IHs are raised, discrete, round, or oval lesions, representing the most common variant. They tend to occur along the lines of embryological fusion lines. Segmental IHs are larger, more flattened lesions covering one or more bodily segments. Indeterminate IHs display both characteristics of both.

The location and growth patterns of segmental head and neck IHs frequently coincide with the trigeminal nerve (nV) distribution. Well-known representations of segmental hemangiomas within the head and neck area are the IHs with beard-like distribution, occurring along the mandibular division of the trigeminal nerve (nV3) distribution area, including the parotid gland, lips, chin, and neck. These beard-like lesions are associated with a higher frequency of coinciding airway IHs. A previous study showed IHs in a beard-like distribution to be associated with airway hemangiomata located in the subglottis in 60% of cases.[15] Segmental IHs in the median distribution (lower lip, lower gingiva, chin, and anterior neck) are associated with highest risk of simultaneous airway IH.

A summary of relevant characteristics of vascular anomalies discussed in this paper is given in Table 1.

Known IH associations include PHACE syndrome (posterior fossa brain malformations, hemangiomas, arterial anomalies, cardiovascular defects, and eye anomalies, with or without midline ventral defects such as sternal clefting or supraumbilical raphe) and LUMBAR syndrome (lower body hemangioma, urogenital anomalies/ulceration, myopathy, bony deformities, anorectal anomalies/arterial anomalies, and renal anomalies).[16,17] The risk of airway involvement is considerably higher in individuals with PHACE syndrome, more than half of which show airway involvement.[18]

2.2 | Congenital hemangiomas

Congenital hemangiomas (CHs) are congenitally fully formed and present by definition at birth. They are less common than IHs and do not show disproportionate growth after birth as most vascular tumors do. CHs may be found in the skin and subcutaneous soft tissue, and less commonly the liver and other visceral organs. They are clinically heterogeneous, usually rounded or oval in shape with pink coloration and are often associated with superficial telangiectasia or peripheral

veins. In contrast to IH, their endothelial cells are GLUT-1 negative which helps the differentiation.[19] CHs may involute rapidly (rapidly involuting congenital hemangioma [RICH]) during infancy, or do not involute at all (non-involuting congenital hemangioma [NICH]). The third subset named partially involuting congenital hemangioma (PICH) is rare. PICHs will show involution similar to RICHs, but will not regress completely.

3 | VASCULAR MALFORMATIONS

Vascular malformations are congenital abnormalities, resulting from abnormal vessel development and morphogenesis. They are present at birth and grow proportionally with the rest of the body. Vascular malformations tend to persist, despite having limited postnatal endothelial mitotic activity. They may contain any combination of venous, capillary, lymphatic, or arterial components. Vascular malformations are currently classified as either simple or combined.[7] Simple malformations may be further subdivided into capillary malformations (CMs), lymphatic malformations (LMs), venous malformations (VMs), arteriovenous malformations (AVMs), and congenital arteriovenous fistulas (AVFs). These may be further subdivided based on the presence or absence of unique genetic variants. Combined vascular malformations are lesions showing clinical features of two or more of the main groups of simple malformations.

3.1 | Capillary malformations

CMs constitute an array of different clinicopathologic entities. CMs are also named venulocapillary malformations. They may occur as part of syndromes such as Sturge–Weber syndrome, Klippel–Trenaunay syndrome, capillary malformation-arteriovenous malformation syndrome, and macrocephaly-capillary malformation syndrome.[20,21] For most of these syndromes, an associated gene defect has been identified. The most commonly encountered is the cutaneous CM, which most commonly affects the head and neck area. This lesion is also known as port-wine stain (PWS) or nevus flammeus.[22] These lesions initially present as flat and may be colored red, or pinkish-violaceous. They are typically located in the face (90%), but may also appear in the neck, trunk, arm, leg, and hand.[23–25] During the first few months of life, they will lighten, as circulating blood hemoglobin concentration decreases.[26] This should not be confused with spontaneous regression. Over time, most PWSs will thicken, become darker and nodular, especially when located in the face.[27] They mainly affect the dermis, but may extend deeply into the subcutis. An associated hypertrophy of local soft tissue and/or bone is commonly seen. Gingival hypertrophy and subsequent dental abnormalities may also be present.[28] PWSs have an incidence of 0.3% in newborns, with an equal male-to-female presentation ratio.[29] They are most commonly unilateral and segmental, but may also be bilateral and/or multisegmental. In case of involvement of the ophthalmic branch of the trigeminal nerve (nV1) distribution area, there is an increased frequency of concomitant leptomeningeal or choroid CM has been documented. This is especially true for larger or bilateral PWSs in this area. This combination is also part of the clinical picture of Sturge–Weber syndrome. Although the pathologic origin of CMs is still largely unknown, a somatic mutation in *GNAQ*, interfering with vascular development, has been identified in isolated PWSs by Shirley et al.[30]

3.2 | Lymphatic malformations

LMs were traditionally named lymphangiomas, despite the absence of endothelial mitotic activity. They are thought to result from developmental errors of the lymphatic system leading to lymphatic tissue formation in abnormal locations.[31] They tend to appear as soft and compressible lesions and are usually present at birth or may present within the first 2 years. Deeper or smaller lesions may remain hidden and only become evident when leading to symptoms or deformity. They are most commonly located in the head and neck area, where they may cause significant deformity and psychosocial morbidity. LMs may often cause soft tissue or bony overgrowth. When LMs involve the (epi)dermis or mucosa, surface vesicles may form, which bleed and become dark. Superficial lesions are often associated with deeper seated lesions, which may explain the frequency of recurrence after resected lesions. LM of the head and neck is associated with a significant risk of upper airway obstruction. The most common complications of LMs are bleeding and infection. Intralesional bleeding occurs in up to 35% of LMs, causing pain or swelling.[32]

LMs are subclassified according to cyst diameter into microcystic, macrocystic, or combined.[4,33] Macrocystic LMs are defined by a cyst diameter of at least 2 cm. They are usually located in the soft tissues of the neck, chest wall, axilla, and groin. Their size may fluctuate with the increase and decrease of lymph fluid within the lymphatic vessels. Microcystic LM consists of cysts smaller than 2 cm. They represent the most common form of LM, and may occur in any area where lymphatic vessels are present. Mixed LMs contain both macro- and microcystic components. Historically, macrocystic LMs were named cystic hygromas, whereas microcystic LMs were referred to as lymphangioma circumscriptum. The problem with these previous terms is their reference to neoplasia, which is not a characteristic of LMs.[34]

3.3 | Venous malformations

VMs are lesions arising from errors in venous network development, resulting in dilated venous channels due to smooth muscle cell deficiency. They present as bluish, compressible masses and may affect all bodily tissues and sites. VMs are solitary and sporadic in around 90% of cases, they may also occur segmental, superficial, or deep. More than 40% arise in the head and neck area, representing the third most common head and neck vascular anomaly, along with IHs and LMs.[35,36] VMs are present at birth but may not become evident until they cause symptoms or deformity. Common complications are ulceration, bleeding, as well as localized intravascular coagulopathy in

larger and/or deeper lesions. VMs do not spontaneously regress, but progressively expand and may compress or invade other tissues.[4,7] In the head and neck, VMs may disrupt normal respiration, swallowing, and speech.

3.4 | Arteriovenous malformations

AVMs are high-flow vascular malformations that stem from abnormal connections between arteries and veins, leading to vascular staining and soft tissue growth. AVMs are present at birth but only around 50% at that time are visible. The remainder will become visible later in childhood or adulthood. They may involve several tissues such as skin, subcutis, soft tissue, bone, and visceral organs. AVMs do not involute but continue to grow throughout life, leading to tissue destruction. They may occur anywhere in the body, but most often develop in spinal cord and brain. The head and neck area is the most common site of extracranial AVM, other frequent presentation sites are the limbs, trunk, and viscera.[37] AVMs may be classified as focal or diffuse. Focal AVMs usually present early in life, have distinct borders and one or two arterial feeders, and are most commonly found in the lip and tongue.[38,39] Diffuse AVMs present in late childhood as diffuse, multiple lesions mostly invading the brain or lungs. AVMs may compress or invade other tissues and cause disfigurement. A palpable thrill may sometimes be present at the AVM site due to fast local arterial blood flow. Arteriovenous shunting reduces capillary oxygen delivery, leading to ischemia. Angiogenesis and/or vasculogenesis may be involved in progression and recurrence of AVM.[40]

4 | CONCLUSION

Accurate diagnosis of vascular anomalies remains challenging, due to their phenotypic diversity and changing classification systems. Inconsistent naming results in miscommunication and suboptimal management of these lesions. The 2014 ISSVA system, based on distinction of vascular tumors and malformations, serves as a sound basis for diagnosis, communication, and treatment. Adhering to this classification of vascular anomalies will allow clinicians to better identify and manage these challenging clinical entities.

AUTHOR CONTRIBUTIONS

Karin P. Q. Oomen: Conceptualization; writing – original draft. **Volkert B. Wreesmann:** Conceptualization; writing – review and editing.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during this study.

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