

Imaging of Vascular Lesions of the Head and Neck



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KEYWORDS

- Vascular malformation • Head and neck • Hemangioma • Carotid body tumor • Glomus jugulare
- Juvenile nasopharyngeal angiofibroma

KEY POINTS

- Vascular lesions of the head and neck are classified based on their endothelial cell turnover as either malformations or tumors.
- Further subclassification based on flow characteristics helps to direct diagnosis and treatment.
- Imaging plays a key role in the diagnosis of most vascular lesions of the head and neck.
- Ultrasonography is an appropriate screening tool, with MRI providing more details about lesion characteristics and extent.
- Many vascular lesions are treated with sclerosants or embolization as either primary or adjunct therapies.

INTRODUCTION

Vascular lesions of the head and neck represent a challenging pathologic subset for the clinician and the radiologist. The classification system described by Mulliken and Glowacki¹ separates the lesions into 2 groups: Those that are vascular malformations, with normal endothelial cell turnover, and those that are vascular tumors, with high endothelial cell turnover (**Box 1**). Further stratification of these lesions is achieved by evaluating the imaging characteristics and clinical presentation; this allows for an accurate diagnosis and choosing appropriate therapeutic management (**Box 2**). Important imaging features that help to narrow the differential diagnosis include lesion flow characteristics, internal tissue characteristics, anatomic location, and extent. Because the literature has been abundant with confusing terms (such as cavernous hemangioma (HM), which truly are venous vascular malformations), it is important

to understand which terms are interchangeable, and to move toward more consistent terminology (**Box 3**).

Vascular malformations of the head and neck include capillary malformations (CM), venous malformations (VM), lymphatic malformations (LM), arteriovenous malformations (AVM), and mixed-type lesions. Because vascular malformations are nonneoplastic, their growth is proportional to body size.¹ Vascular tumors, on the other hand, are neoplastic, so their growth is independent of body size. Although the list of vascular tumors of the head and neck can be quite long, we discuss some of the common lesions in this article, including HM, carotid body tumors (CBTs), glomus jugulare tumors (GJTs), and juvenile nasopharyngeal angiofibromas (JNA). In this review, we address clinical presentation, differential diagnosis, differentiating features, characteristic imaging findings, as well as a brief discussion of treatment options of these lesions.

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Box 1**Lesion classification**

Vascular Malformations	Vascular Tumors
Capillary malformation	Hemangioma
Venous malformation	Carotid body tumor
Lymphatic malformation	Glomus jugulare tumor
Arteriovenous Malformation	Juvenile nasopharyngeal angiofibroma
Mixed type lesion	

IMAGING TECHNIQUES

Several imaging modalities play an integral role in the diagnosis and treatment planning of vascular lesions of the head and neck. These include ultrasonography (US), computed tomography (CT), MRI, as well as cross-sectional angiography and catheter-guided angiography (magnetic resonance angiography, CT angiography, digital subtraction angiography). In some instances, plain radiography can also contribute to the diagnosis. Because these lesions occur most commonly in pediatric and adolescent patients, judicious use of radiation is paramount.

US is an appropriate initial screening modality, particularly in superficial lesions.² US can help to identify rudimentary characteristics of the lesion, delineate lesion depth, characterize cystic/solid spaces, and identify the lesion's flow characteristics.³ MRI is often used for further characterization because it is the best modality for delineating lesion extent and determining the involvement of soft tissue structures.^{2,4} When available, dynamic magnetic resonance angiography (such as 4D-TRAK) can noninvasively determine the flow pattern within a lesion and thereby help in the diagnosis.⁵ CT can also provide a wealth of diagnostic information and is particularly valuable in determining bony involvement, vascularity, and in

Box 2**Flow characteristics**

High Flow	Low Flow
Arteriovenous malformation	Capillary malformation
Hemangioma (proliferative phase)	Venous malformation
Carotid body tumor	Lymphatic malformation
Juvenile nasopharyngeal angiofibroma	

Box 3**Terminology: new and old**

New	Old
Hemangioma	Capillary hemangioma
Capillary malformation	Port wine stain
Venous malformation	Cavernous hemangioma
Lymphatic malformation	Lymphangioma; cystic hygroma

evaluating for the presence of phleboliths (also identified on plain radiography).² However, CT use should follow the “as low as reasonably achievable” principles to minimize radiation exposure, especially in pediatric patients. Catheter angiography plays a limited role and is mainly employed when embolization therapy is a consideration. In many cases, characterization of the intralésional components and anatomic location allows for a definitive diagnosis.

IMAGING FINDINGS/PATHOLOGY**Vascular Malformations****Capillary malformations**

CM in the head and neck present as a “port wine stain” cutaneous lesion. These lesions follow a dermatomal pattern of the trigeminal nerve in 23% to 43% of patients, but can be much more extensive in other cases.^{6,7} Sturge–Webber syndrome, a congenital cutaneous and neurologic disorder, affects 3% of patients presenting with a port wine stain.⁶ Imaging plays a limited role in evaluation of CM because diagnosis can often be made based on clinical characteristics. However, it can provide value in patients with Sturge–Webber syndrome or in those where the clinical presentation is equivocal. Key differential considerations of CM include superficial HM, VM, and AVM.

On US, CMs are usually located in the dermis and appear isoechoic.⁸ Even though they are generally considered low flow, Doppler signal can be identified in up to 29%.⁸ When Sturge–Webber syndrome is suspected, MRI or CT can be used to evaluate for disease sequelae, including ipsilateral leptomeningeal vascular anomalies.⁷ These modalities also aid in the differentiation of CM from other vascular lesions, which commonly extend deeper into the tissues, involve bony structures, and have more robust internal flow.

CMs are generally benign lesions. They can become darker and more obvious with time, however, which makes them cosmetically bothersome to many patients. In these cases, the treatment of choice is laser therapy.⁹ In recurrent, extensive, or hypertrophic and nodular lesions, wide surgical

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