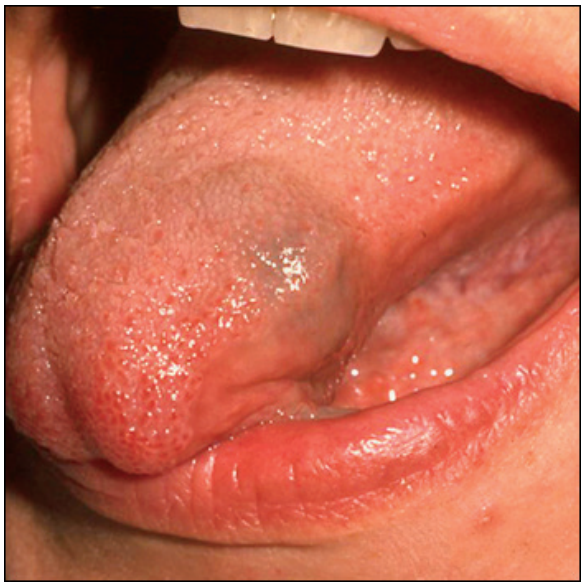
Bluish Mass on the Tongue

Course Author(s): H. Stan McGuff, DDS; Anne Cale Jones, DDS; Michaell A. Huber, DDS;
Online Case: *www.dentalcare.com/en-us/professional-education/case-challenges/case-challenge-074*



The following Case Challenge is provided in conjunction with the UT Health San Antonio School of Dentistry faculty.

Mary is a 43-year-old-female who presents for evaluation of a bluish mass on her left anterior lateral tongue.

After you have finished reviewing the available diagnostic information, make the diagnosis.

Diagnostic Information

History of Present Illness

Mary is a healthy 43-year-old-female who presents for evaluation of a tongue mass. The patient has been aware of an asymptomatic bluish discoloration on her left anterior lateral tongue for the past 5-6 years. 3 months ago this area began to swell and it has now become bothersome. She has occasionally bitten the area and it is beginning to interfere with her speech. She is concerned that she has cancer.

Medical History

- Pertinent Medical History: Hashimoto thyroiditis
- Medications: levothyroxine
- Adverse Drug Reactions: nausea/vomiting with codeine use.
- Family History: paternal - prostate cancer; maternal - osteoporosis; siblings - two healthy younger sisters
- Social History: social alcohol use, recreational marijuana, no tobacco use

Clinical/Radiographic Findings

Extra-oral examination shows normal facial symmetry, no skin lesions, intact cranial nerve function, no trismus, and no cervical lymphadenopathy. The thyroid gland is palpable and multinodular. Intra-oral examination reveals no other mucosal lesions. The dentition is intact and oral hyigene is good with foci of mild marginal gingivitis. Periodontal probing depths are a maximum of 4 mm with focal bleeding on probing interproximally. The occulsion is class I with no evidence of temporomandibular dysfuction or paranormal habits. There is a 2.8 x 1.3 cm deep-seated nontender soft compressible mass on the left anterior dorsal and ventrolateral tongue. The mass is nonpulsatile and does not blanch. The remainder of the tongue is soft and freely mobile. Because of her concern for cancer, the patient requests that a biopsy be performed. and requested a biopsy be performed.

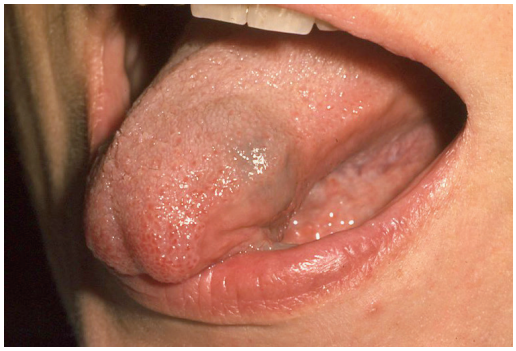


Figure 1. Periapical radiograph showing circumscribed periapical radiolucency with central opacities involving tooth #25.

Histopathologic Findings

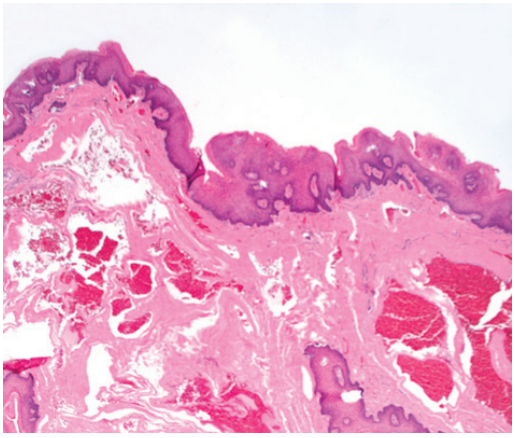


Figure 2. Low-power microscopic image showing numerous dilated blood filled thin-walled venous vascular channels involving the submucosa and tongue musculature.

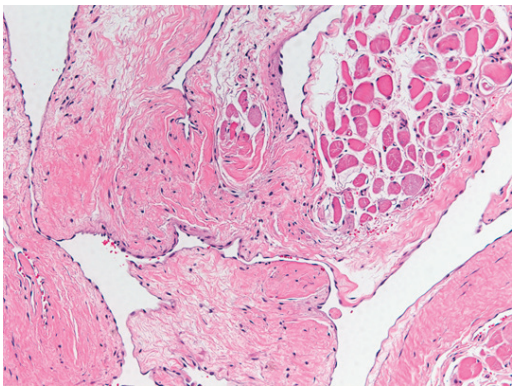
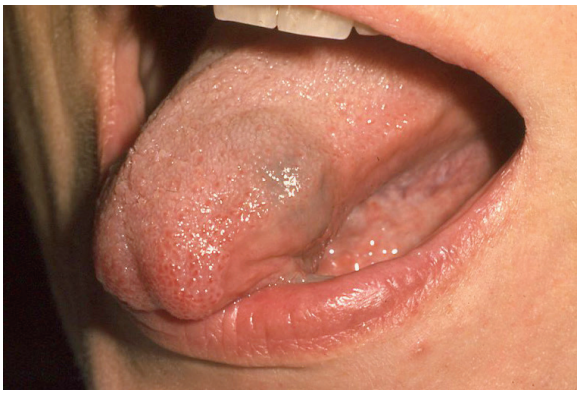


Figure 3. High-power microscopic image revealing ectatic intramuscular venous blood vessels lined by bland flattened endothelial cells.

Select Diagnosis

Can you make the diagnosis

A 43 year-old-female presents for evaluation of a bluish mass on her left anterior tongue. information, make the diagnosis.



Select the Correct Diagnosis

A. Varicosity
B. Cavernous venous vascular malformation
C. Angiosarcoma
D. Lymphangioma

Varicosity

Choice A. Sorry, this is not the correct diagnosis.

Varicosities represent abnormally dilated tortuous veins which are common in older adults.[1-3] The development of varicosities is associated with age-related degeneration of the connective tissue and loss of vascular support. Varicose veins of the lower extremity, hemorrhoids, and esophageal varices may also represent a response to increased venous pressure. In the oral cavity, sublingual varicosities are very common in people older than 60 years of age. These present as multiple bilateral superficial blue submucosal blebs on the ventral and lateral tongue surface (caviar tongue). Solitary varices are less common and are most often seen on the lips and buccal mucosa. These present as a small blue submucosal nodule. Varices are compressible and may blanch under pressure. Oral varices are usually asymptomatic. Occasionally varices can become secondarily thrombosed and present as a firm blue-grey nodule. Microscopic examination will show a dilated thin-walled venous vascular channel(s) lined by flattened endothelial cells. There is usually a minimal smooth muscle component and decreased supporting connective tissue. Thrombosed varices will show an intraluminal clot which may show ingrowth of granulation tissue and evolving recanalization. There may be dystrophic calcification of the clot producing a phlebolith. Sublingual varicosities usually do not require treatment. Solitary varicosities may be excised for diagnostic purposes or for cosmetic reasons. The prognosis for varices is excellent. The histopathologic findings do not support the diagnosis for this case.

Please re-evaluate the information about this case.

Cavernous venous vascular malformation

Choice B. Congratulations! You are correct.

Venous vascular malformations (VM) are common developmental abnormalities composed of large dilated vascular channels.[1,4-7] VMs may develop in a variety of sites including skin, soft tissue, bone, and visceral organs. The malformations may range from small clinically insignificant to complex deforming lesions. The head, neck, and oral cavity are frequent sites of occurrence for VMs. The abnormal vessels are present at birth but often do not become clinically evident until later in childhood or adulthood. Venous lesions are typically low-flow and present clinically as a blue to red compressible soft tissue mass. Malformations with an arterial component (arteriovenous malformations) will be high-flow and produce a palpable pulsation or a bruit on auscultation. VMs tend to grow in proportion with the patient. Increased venous pressure may cause enlargement of the lesion. The vessels insinuate into adjacent structures and will produce a mass affect. Otherwise, most lesions are asymptomatic. Secondary thrombosis, phlebolith formation, pain, bleeding and ulceration may occur. Intraosseous vascular malformations present as a multi to unilocular, honeycombed, or subtle ill-defined radiolucency. Cortical expansion, tooth mobility, and gingival bleeding may also be present. Histologic examination will show increased numbers of large ectatic thin-walled venous vascular channels lined by bland flattened endothelial cells. The vessels are filled with blood. There are intervening bands of fibrous connective tissue separating the venous channels. The vascular malformation is poorly delineated and permeates through adjacent tissues. Unlike congenital hemangiomas, there is no active endothelial proliferation. Small asymptomatic VMs may not require treatment. Management of large deforming, cosmetically concerning, or high-flow lesions may be problematic. Treatment options include surgery, embolization and sclerotherapy. There is a risk of severe life-threatening bleeding, especially with high-flow lesions occurring in bone. Needle aspiration of undiagnosed jaws lesions is recommended to rule-out a central vascular lesion before surgical entry or tooth extraction. Unlike congenital hemangiomas, VMs do spontaneously regress. Overall, the prognosis for VMs is good. There appears to be no significant risk of malignant transformation.

Angiosarcoma

Choice C. Sorry, this is not the correct diagnosis.

Angiosarcomas are uncommon malignant soft tissue neoplasms of vascular endothelial differentiation.[1,8-11] The most common sites of occurrence for angiosarcoma are the skin, breast, and liver. More than half of all angiosarcomas develop in the skin of the scalp and forehead. Angiosarcomas of the oral cavity are distinctly uncommon, but have been reported in the tongue and mandible. Most cases arise in elderly adults with equal gender predilection. Risk factors include lymphedema (post-mastectomy), ionizing radiation, and exposure to carcinogens. It is uncertain if sun damage plays a role in cutaneous lesions. Early angiosarcomas may deceptively resemble a bruise. As the lesion grows it becomes tumifactive and may ulcerate. Some lesions will appear multifocal. Microscopic examination will show a highly infiltrative neoplasm composed of complex irregular anastomosing vascular channels lined by increased numbers of atypical mitotically active endothelial cells. Areas of necrosis and hemorrhage are common. Treatment for angiosarcoma consists of radical resection, radiation therapy, and chemotherapy. Because of the infiltrative nature of angiosarcoma, complete removal of the tumor is difficult to achieve. Angiosarcoma also has a tendency to metastasize. The prognosis for angiosarcoma is poor. The histopathologic findings do not support the diagnosis for this case.

Please re-evaluate the information about this case.

Lymphangioma

Choice D. Sorry, this is not the correct diagnosis.

Lymphangiomas are developmental malformations of lymphatic vascular channels that have a predilection for the head and neck region.[1,12-14] Most are present at birth or arise during childhood. Those that involve the neck are known as cystic hygromas. These are typically compressible deep cervical macrocystic soft tissue masses. Rapid enlargement of the lymphatic malformation may occur following upper respiratory infections. Cystic hygromas have been associated with Turner syndrome. Lymphangiomas of the oral cavity typically affect the anterior two-thirds of the tongue and can present as superficial pebbly clear microcystic vesicles and/or a deep seated painless compressible soft tissue mass causing macroglossia. Microscopic examination will show an unencapsulated mass of dilated lymphatic channels lined by bland endothelial cells. The vascular lumens contain pink proteinaceous material. Erythrocytes may also be seen in mixed angiolymphatic lesions or with hemorrhage following trauma. The lymphatic vessels tend to insinuate into adjacent normal tissues and have a poorly delineated border. The supporting fibrous connective tissue stroma interspersed between the lymphatic vessels may contain scattered lymphoid aggregates. The management of lymphangiomas depends of the size and location of the lesions. Small lymphangiomas may only require observation. Larger lesions that cause functional or esthetic concerns can be treated with surgery or sclerotherapy. Complete surgical removal of these lesions is difficult and recurrences are common. Massive lesions have the potential to compress adjacent structures resulting in airway obstruction and death. Overall, the prognosis for lymphangiomas is good. The histopathologic findings do not support the diagnosis for this case.

Please re-evaluate the information about this case.

References

1. Neville BW, Damm DD, Allen CM, et al. Oral and Maxillofacial Pathology. 4th ed. St. Louis, MO. Elsevier. 2016.
2. Assi R, Kessler HP, Clark CL. Oral and maxillofacial pathology case of the month. Varix with phlebolith. Tex Dent J. 2012 Jul;129(7):684-5, 712-3.
3. Canaan TJ, Meehan SC. Variations of structure and appearance of the oral mucosa. Dent Clin North Am. 2005 Jan;49(1):1-14, vii. doi: 10.1016/j.cden.2004.07.002.
4. Kobayashi K, Nakao K, Kishishita S, et al. Vascular malformations of the head and neck. Auris Nasus Larynx. 2013 Feb;40(1):89-92. doi: 10.1016/j.anl.2012.02.002. Epub 2012 Apr 23.
5. Perez D, Leibold D, Liddell A, et al. Vascular lesions of the maxillofacial region: a case report and review of the literature. Tex Dent J. 2010 Oct;127(10):1045-57.
6. Buckmiller LM, Richter GT, Suen JY. Diagnosis and management of hemangiomas and vascular malformations of the head and neck. Oral Dis. 2010 Jul;16(5):405-18. doi: 10.1111/j.1601-0825.2010.01661.x. Epub 2010 Mar 9.
7. Glade RS, Richter GT, James CA, et al. Diagnosis and management of pediatric cervicofacial venous malformations: retrospective review from a vascular anomalies center. Laryngoscope. 2010 Feb;120(2):229-35. doi: 10.1002/lary.20715.
8. Lin SC, Chang TS. Cutaneous angiosarcoma of the scalp mimicking facial cellulitis. Ear Nose Throat J. 2016 Oct-Nov;95(10-11):438-440.
9. Hwang K, Kim MY, Lee SH. Recommendations for therapeutic decisions of angiosarcoma of the scalp and face. J Craniofac Surg. 2015 May;26(3):e253-6. doi: 10.1097/SCS.0000000000001495.
10. Patel SH, Hayden RE, Hinni ML, et al. Angiosarcoma of the scalp and face: the Mayo Clinic experience. JAMA Otolaryngol Head Neck Surg. 2015 Apr;141(4):335-40. doi: 10.1001/jamaoto.2014.3584.
11. Nagata M, Yoshitake Y, Nakayama H, et al. Angiosarcoma of the oral cavity: a clinicopathological study and a review of the literature. Int J Oral Maxillofac Surg. 2014 Aug;43(8):917-23. doi: 10.1016/j.ijom.2014.02.008. Epub 2014 Mar 19.
12. de Queiroz AM, Silva RA, Margato LC, et al. Dental care management of a young patient with extensive lymphangioma of the tongue: a case report. Spec Care Dentist. 2006 Jan-Feb;26(1):20-4.
13. Brennan TD, Miller AS, Chen SY. Lymphangiomas of the oral cavity: a clinicopathologic, immunohistochemical, and electron-microscopic study. J Oral Maxillofac Surg. 1997 Sep;55(9):932-5.
14. Thompson TL, Gungor A. Diffuse, encasing lymphangioma of the supraglottis. Am J Otolaryngol. 2016 Jan-Feb;37(1):41-3. doi: 10.1016/j.amjoto.2015.09.009. Epub 2015 Sep 10.

About the Authors

H. Stan McGuff, DDS

H. Stan McGuff, D.D.S. is a Professor of Pathology in the School of Medicine at The University of Texas Health Science Center at San Antonio. He graduated from the Dental School at The University of Texas Health Science Center at San Antonio in 1977. Dr. McGuff practiced dentistry as an officer in the United States Air Force and as a general dentist in Live Oak, Texas. In 1993 Dr. McGuff completed a residency in general anatomic pathology and a fellowship in oral, head and neck pathology at The University of Texas Health Science Center at San Antonio. He has remained at The University of Texas Health Science Center at San Antonio as a faculty member for 28 years. The main focus of his career has been diagnostic surgical pathology of the oral cavity, head and neck region. He is involved in graduate and undergraduate dental and medical education. His research interests include head and neck cancer, the immunopathology of Sjogren's syndrome, metabolic bone disease, bone wound healing and tissue interactions with biomaterials.

Email: mcguff@uthscsa.edu

Anne Cale Jones, DDS

Anne Cale Jones graduated from the University of Alabama in 1981 with the Bachelor of Science degree (Magna Cum Laude) in Natural Sciences. She received a Doctor of Dental Surgery degree (Magna Cum Laude) from the Medical College of Virginia, Virginia Commonwealth University in 1986. Following a three-year residency program in Oral and Maxillofacial Pathology at Booth Memorial Medical Center in Queens, New York, Dr. Jones joined the faculty at the University of Florida, College of Dentistry. In 1998, she became a faculty member at The University of Texas Health Science Center at San Antonio. She is currently a Distinguished Teaching Professor in the Department of Pathology and is board certified by the American Board of Oral and Maxillofacial Pathology.

Email: jonesac@uthscsa.edu

Michaell A. Huber, DDS
Professor
Department of Comprehensive Dentistry
The University of Texas Health Science Center at San Antonio, School of Dentistry, San Antonio, Texas

Dr. Michaell A. Huber is a Professor of Oral Medicine, Department of Comprehensive Dentistry, the UTHSCSA School of Dentistry. He received his DDS from the UTHSCSA in 1980 and a Certificate in Oral Medicine from the National Naval Dental Center, Bethesda, Maryland in 1988. He is certified by the American Board of Oral Medicine. Dr. Huber served as Graduate Program Director in Oral Medicine at the National Naval Dental Center, Bethesda, Maryland. In addition he served as Specialty Leader for Oral Medicine to the Surgeon General of the United States Navy, Washington, DC; and Force Dental Officer, Naval Air Force Atlantic, Norfolk, Virginia.

Since joining the faculty in 2002, Dr. Huber has been teaching both pre-doctoral and graduate dental students at the UTHSCA School of Dentistry. In 2014, he was awarded the UTHSCSA Presidential Teaching Excellence Award. He is a Past President of the American Academy of Oral Medicine. Dr. Huber has spoken before many local, state, and national professional organizations. He has published over 70 journal articles, book chapters, and online postings.

Phone: (210) 567-3360
Fax: (210) 567-3334

Email: huberm@uthscsa.edu