

The tongue: a bridge between physiology and disorder

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Abstract

The tongue, as a multifunctional organ, exhibits a variety of physiological characteristics and pathological alterations. Identifying and understanding these variations is crucial for ensuring an accurate diagnosis and appropriate management. This article explores the main common physiological variations, such as

geographic tongue, fissured tongue, *etc.*, as well as pathological conditions, including elementary lesions such as vesicles, bullae, erosions, ulcers, plaques, along with surface changes (atrophy), color changes (coatings, pigmentation), or even shape alterations (infiltrations). It discusses current diagnostic tools and therapeutic approaches while emphasizing the importance of regular monitoring to prevent complications and maintain optimal oral health.

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Introduction

The tongue is a multifunctional organ essential for speech, swallowing, and taste, and also serves as an indicator of general health. Changes in color, appearance, and burning sensations can reveal a multitude of conditions, ranging from simple physiological variations to more serious pathologies. Through the analysis of primary mucosal lesions such as vesicles, bullae, erosions, ulcers, plaques, changes in surface condition (atrophy), color (coatings, pigmentations), or even shape (infiltrations), an accurate diagnosis can be established. Certain non-pathological aspects of the tongue may motivate a consultation and must be quickly recognized to reassure the patient of their benign nature. This review article bridges the gap between normal physiological processes and pathological disorders, highlighting the tongue's importance as a window into overall health and offering insights into its potential in early disease detection.

Physiological variations of the tongue

Fissured tongue: a common variant observed in 20 to 30% of the population, characterized by increased fissures and grooves on the dorsal and lateral surface (*Supplementary Figure 1*).

It is a painless, most often isolated condition, though it can sometimes be associated with a geographical tongue. Along with facial edema (macrocheilitis in 66 to 96% of cases) and peripheral facial paralysis, it could be part of the diagnostic triad of Melkerson-Rosenthal syndrome. No specific treatment is necessary. Deeper fissures are often associated with a plicated tongue.^{2,3}

Geographic tongue: also known as benign migratory glossitis, it may be present at all ages. Clinically, it is characterized by areas of lingual depapillation, affecting the filiform papillae, delimiting erythematous regions with a slightly raised white border. These depapillated areas are irregularly arranged (Supplementary Figures 2 and 3). Atypical forms may concern the ventral side or other extralingual oral regions called erythema migrans. No treatment is necessary for asymptomatic forms. An association between geographic tongue and cutaneous psoriasis is described with a common genetic determinism.¹⁻³

Lingual varices: a common developmental abnormality in older adults. The etiology of oral varicosities remains unknown; they correspond to a benign vascular dilatation of the sublingual veins, non-inflammatory and painless, with degeneration of elastic





fibers, which can be confused with an arteriovenous malformation (*Supplementary Figure 4*). The diagnosis is clinical and no treatment is required.^{1,3}

Plica fimbriata: represents an embryonic remnant found under the tongue. These are folds of the mucosa that extend on either side of the lingual frenulum, forming slightly fringed structures (*Supplementary Figure 5*).

Hypertrophy of circumvallate papillae, foliate papillae, and lingual tonsils: often a source of concern for some patients, which can sometimes simulate squamous cell carcinoma. The lingual tonsils are sometimes inflammatory and sensitive during infectious episodes of the upper aerodigestive tract (Supplementary Figure 6). 1,3

Macroglossia: defined as an enlargement of the tongue that can lead to functional problems with ventilation and swallowing. It is characterized by the presence of tooth imprints on the lateral borders of the tongue and may even cause repetitive biting (Supplementary Figure 7). Macroglossia can be due to congenital or acquired causes. Congenital factors include muscular hypertrophy, glandular hyperplasia, neonatal diabetes, and certain genetic syndromes such as Beckwith-Wiedemann syndrome, Melkersson-Rosenthal syndrome, and Down syndrome. Acquired factors include acromegaly, hypothyroidism, amyloidosis, and tongue tumors.³

Coatings

Black hairy tongue: refers to an elongation of the filiform papillae on the dorsal surface of the tongue, which are colored from brown to black (the tip of the tongue and the lateral surfaces are not affected). The coloration is due to the oxidation of keratin, which is attributed to the excessive use of antiseptic mouthwashes, tobacco, poor oral hygiene, or antibiotic therapy. Treatment involves eliminating risk factors and cleaning the dorsal surface of the tongue through scraping and brushing.^{1,3}

Saburral tongue: the dorsal surface of the tongue is covered with a whitish coating, while the mucous membrane below is normal (*Supplementary Figure 8*). This coating is made up of cellular desquamation, food debris, and saprophytic bacterial flora; it disappears when the cause is eliminated.^{1,3}

Pathological variations of the tongue

Pathological changes in the tongue can alter its color, texture, coating, and size, among other features. A comparison of normal and abnormal tongue characteristics is summarized in Table 1. Accurate clinical examination based on the analysis of primary lesions (ulceration, erosion, nodule, plaque, macule, papule, bullae, vesicles, *etc.*) is essential for distinguishing these variations.

Erythemas

Erythroplakia: a rare lesion with malignant potential, characterized by a velvety red area, well defined, and most often uniform without traces of keratinization. Risk factors include tobacco and alcohol. The rate of malignant transformation is much higher than that of leukoplakia, making any erythroplakia lesion eligible to be biopsied and treated promptly. Long-term follow-up is necessary.

Kawasaki disease: a vasculitis that tends to occur in children before the age of five. It is characterized by prolonged fever, bilateral conjunctivitis, exanthema, cervical lymphadenopathy, and palmar-plantar erythema with edema and oropharyngeal involvement. Oral involvement includes dry, cracked lips and the typical appearance of a strawberry tongue with prominent papillae. ^{1,5}

Scarlet fever: a bacterial infection caused by group A *Streptococcus*, characterized by a rash, high fever, sore throat, and enanthema; the initially saburral tongue loses its whitish coating from the periphery to the center and becomes uniformly red with prominent papillae, also known as a strawberry tongue.¹

Anemia and vitamin deficiency: iron deficiency anemia is responsible for angular cheilitis with cracked lips and depapillation of the tongue. Treatment is based on anemia correction with iron supplementation. Anemia due to folate (vitamin B9) or vitamin B12 deficiency can cause Hunter's glossitis, which manifests itself at an early stage by lingual paresthesias, burning sensations, intolerance to dental prostheses, and/or dry mouth. Subsequently, red, glossy, depapillated areas are noticed.

The advanced phase of Hunter's glossitis shows a fissure network with a plicated appearance and can sometimes present a pultaceous coating (*Supplementary Figure 9*). Etiological and replacement treatment of the deficiency allows the symptoms to disappear.^{1,7}

Oral candidiasis: can affect the entire oral mucosa. However, some variants can manifest exclusively on the tongue, such as median rhomboid glossitis. It is characterized by a rhomboid erythematous area on the dorsal surface of the tongue, anterior to the lingual V. The area may be smooth and pink, flat, or papillary, often confused with hypertrophied circumvallate papillae. A mirror-image palatal erythematous lesion may be associated (Supplementary Figures 10 and 11).8

Vesicles

Basic vesicular lesions are essentially linked to viral attacks, giving general and oral manifestations. These include herpes simplex virus (HSV) (*Supplementary Figure 12*), varicella-zoster virus (VZV), and hand, foot, and mouth syndrome. These viral infections generally result in painful post-vesicular erosions. However, some particularities are observed in the tongue, such as herpetic geometric glossitis, which often develops in immunocompromised patients. Lesions appear as branched longitudinal

Table 1. Normal vs. abnormal tongue features.

Feature	Normal tongue	Abnormal tongue
Color	Pink, uniform color	White, red, pale, blue, purple or yellow: the tongue's color change could be a symptom of an underlying health condition
Texture	Smooth, slightly rough due to papillae	Rough, swollen or smooth with a shiny appearance
Coating	Thin coating	Thick, yellow, white or black coating
Shape/size	Normal	Enlarged, swollen or atrophic
Movability	Free, fluid, able to move in all directions	Restricted movement
Edges	Normal, slightly tapered at the tip	Scalloped edges, indentations, or teeth marks
Moisture	Moist, not excessively dry or wet	Dry or excessively moist tongue





linear fissures, accompanied by extremely painful post-vesicular erosions preventing swallowing and speech. 9,10 VZV can particularly affect the lingual nerve. In this case, the patient describes lingual pain, decreased sensitivity, an alteration of taste, and eating discomfort associated with erosions covered with a pseudo-membranous coating, complicating swallowing.

Bullae

Autoimmune bullous stomatitis, such as pemphigus or cicatricial pemphigoid, or immuno-allergic stomatitis, such as ervthema multiforme or hemorrhagic bullous angina, can all be revealed by lingual damage. 1 Erythema multiforme (EM) is characterized by targetoid skin lesions associated with lip involvement (Supplementary Figure 13).11 However, when it is accentuated (major EM), it presents lesions in the tongue and oral mucosa. Contrary to its name, hemorrhagic bullous angina can occur anywhere in the oral cavity, including the tongue, in the form of a large bulla with hematic content, giving way to painful ulceration. 12 Pemphigoid, in its cicatricial form (Supplementary Figure 14), primarily affects the gingiva, presenting as erosive gingivitis. Lingual involvement is more frequent in pemphigoid compared to pemphigus vulgaris, which primarily affects the skin and presents as painful, irregularly shaped ulcers that hinder swallowing and speech. These conditions are often subject to candidal superinfections.13

Ulcers

Traumatic lesions: the tongue is frequently affected by traumatic lesions due to its mobility. These lesions can generally give rise to ulcerations following physical trauma (*Supplementary Figure 15*) (sharp edge, coronal fracture, hook, poorly adapted prosthesis), chemical (application of concentrated antiseptic, use of tobacco) (*Supplementary Figure 16*), and thermal (very hot foods or liquids). Removal of the causative agent allows the lesion to heal.¹⁴

Aphthous stomatitis and recurrent aphthous stomatitis: aphthous ulcers are very common lesions of the oral mucosa. They present as painful ulcers varying in size from a few millimeters (for minor aphthae) to up to two centimeters (for major aphthae), with a rounded or oval shape and surrounded by an erythematous halo (*Supplementary Figure 17*). They typically occur on non-keratinized mucosa, including the lateral and ventral surfaces of the tongue, and rarely on keratinized mucosa (dorsal surface of the tongue). Minor aphthous ulcers heal without scarring, unlike major aphthae. Recurrent aphthous stomatitis is diagnosed if there are at least three episodes of multiple aphthous ulcers per year. Certain pathologies can cause aphthoid ulcerations, such as chron-

ic inflammatory intestinal diseases, hematological disorders (anemia, neutropenia) (Supplementary Figure 18), and taking certain medications (nicorandil, nonsteroidal anti-inflammatory drugs, bisphosphonate, etc.)

Eosinophilic ulcer: known as Riga-Fede disease. It is a rare benign lesion of the oral mucosa located mainly on the anterior part of the tongue following its friction on the edges of the temporary incisors during sucking. It presents as a painful ulcer, most often rounded, measuring a few mm to 2-3 cm in diameter, covered by a pseudo-membranous coating. Treatment is symptomatic for analgesic purposes.¹⁵

Ulcerations linked to vasculitis, Horton's disease: lingual damage can be a revealing sign of this arteritis, characterized by pain/burning, loss of taste sensitivity, claudication of the tongue associated with ulcerations rapidly evolving into necrosis and lingual gangrene.¹⁶

Bacterial origin, syphilis: an infectious sexually transmitted disease caused by *Treponema pallidum*. Primary syphilis, characterized by syphilitic chancre, can affect the entire oral mucosa without particularities in the tongue. On the other hand, in secondary syphilis, the lingual lesions are described as characteristic "mowed lawn" plaques. These damages can progress to necrotic ulcerations in the case of syphilitic gummas; we then speak of associated atrophic glossitis.¹

Malignant tumors, squamous cell carcinoma: is the most common lymphophilic cancer, surpassing verrucous carcinoma and basal cell carcinoma. The most common sites are the ventral surface, lateral borders (*Supplementary Figure 19*), and posterior border, which have a guarded prognosis compared to other locations with a delayed diagnosis (revealed with simple swallowing difficulty). The ulceration varies in size and has an irregular shape with an indurated, elevated, and sometimes everted edge. The base is indurated, extending beyond the ulceration and deep into the tissue.¹⁷

Nodules/papules

These lesions are generally indicative of a benign condition, most often associated with either human papillomavirus (HPV) infection (papillomas or warts) or benign tumors, such as fibroma, nerve tumors, which are most frequent in the tongue compared to other oral sites (neuroma, neurofibroma, schwannoma, Abrikossof tumor). Vascular tumors, such as angiomas, lymphangiomas, and arteriovenous malformations, may also appear as nodules or papules and may pose a risk of asphyxia.¹⁸

White lesions

Leukoplakia: a term used to clinically describe any white lesion of the oral mucosa, idiopathic or associated with tobacco

Table 2. Systemic diseases and their tongue manifestations.

Disease	Signs on the tongue	Clinical relevance
Anemia	Pale, smooth tongue with atrophy of papillae	Associated with fatigue, weakness, and difficulty swallowing
Vitamin B12 deficiency	Glossitis (inflamed, smooth, red tongue)	Lingual paresthesias, burning sensations, intolerance to dental prostheses, and/or dry mouth
Diabetes	Dry, cracked tongue; may also show fungal infections (<i>e.g.</i> , candidiasis)	Infections and oral health issues: cavities/gum disease/periodontitis
Scarlet fever	Strawberry tongue (red with prominent papillae)	Characterized by a rash, high fever, sore throat and enanthema
Horton's disease	Ulcerations rapidly evolving into necrosis and lingual gangrene	Pain/burning, loss of taste sensitivity and claudication of the tongue
Uremic stomatitis	4 forms: ulcerative, hemorrhagic,	Xerostomia, foul breath with an ammoniacal odor,
	pseudomembranous and hyperkeratotic	bad taste and burning sensations



use. Lesions can be homogeneous or inhomogeneous, presenting a high risk of malignant transformation and requiring regular monitoring.²

Lichen planus: the tongue is the second oral site most frequently affected by this chronic inflammatory disease after the posterior jugal mucosa. Lingual lichen planus presents a particular form at the beginning, which manifests itself as more or less rounded plaques resembling "candle wax spots" (*Supplementary Figure 20*). These lesions evolve inconsistently towards an erosive form under the influence of certain factors. In the long term, the mucosa becomes atrophic with the disappearance of the lingual papillae. This thin and fragile mucosa presents an increased risk of malignant transformation, requiring regular monitoring. ¹⁴

Hairy oral leukoplakia: this lesion occurs on the borders of the tongue and is associated with Epstein-Barr virus, observed in immunocompromised individuals. Clinically, it presents as an asymptomatic white plaque with a hairy or undulating surface that resists scraping. However, rare cases affecting other sites of the oral mucosa have been observed.¹⁹

Uremic stomatitis: a rare condition in patients with renal failure. There are 4 forms of uremic stomatitis: ulcerative, hemorrhagic, pseudomembranous, and hyperkeratotic (*Supplementary Figure 21*). The tongue and the buccal floor are most often affected. Associated symptoms are xerostomia, foul breath with an ammoniacal odor, bad taste, and burning sensations.¹

Thus, systemic diseases can often manifest with characteristic changes in the tongue, serving as valuable indicators in clinical diagnosis. Table 2 summarizes some common systemic conditions and their associated tongue manifestations.

Infiltrations

Amyloidosis: a rare disease characterized by the presence of amyloid deposits in various tissues and organs, including the oral cavity. Lingual damage can reveal macroglossia with indentations over the entire lingual margin (*Supplementary Figure 22*). If the salivary glands are affected, xerostomia may develop. The diagnosis is histological, showing positive Congo red staining, which indicates the presence of amyloid proteins. Amyloidosis is mostly found in patients with multiple myeloma.²

Others

Lingual abscess: more common in diabetic individuals, this condition often presents as a painful swelling that causes protrusion of the tongue, dysphagia, odynophagia, and difficulties with speech (*Supplementary Figure 23*). Local trauma, such as injury, bite, spread of a dental infection, sting, or foreign body, is the primary predisposing factor for anterior abscesses. In contrast, lingual tonsillitis or an infected thyroglossal duct cyst are predisposing factors for posterior abscesses, which are considered potentially life-threatening. Management of a lingual abscess requires prompt and aggressive treatment: antibiotic therapy combined with surgical drainage.²⁰

Conclusions

Distinguishing between physiological and pathological variations of the tongue is crucial for accurate diagnosis and appropriate management. Although many variations are benign, it is essential to remain vigilant for abnormal changes in the tongue to prevent complications or detect undiagnosed systemic pathologies. Regular follow-up is recommended to maintain oral health.

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Online Supplementary Material:
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Supplementary Figure 1. Fissured tongue associated with atrophy of the dorsal surface.

Supplementary Figure 2. Geographic tongue with a single depapillated area located at the lateral border of the tongue.

 $Supplementary\ Figure\ 3.\ Geographic\ tongue\ associated\ with\ a\ fissured\ tongue.$

Supplementary Figure 4. Lingual varicosities.

Supplementary Figure 5. Plica fimbriata on the ventral side of the tongue.

Supplementary Figure 6. Hypertrophy of circumvallate and foliate papillae.

Supplementary Figure 7. Macroglossia with teeth marks on the tongue's lateral sides.

Supplementary Figure 8. Saburral tongue aspect.

Supplementary Figure 9. Hunter's glossitis in the context of Biermer's anemia.

Supplementary Figure 10. Median rhomboid glossitis with palatal mirror erythema.

Supplementary Figure 11. Median glossitis associated with pigmentation in a patient with APECED syndrome.

Supplementary Figure 12. Herpetic gingivo-stomatitis.

Supplementary Figure 13. Erythema multiforme affecting the lips and the ventral surface of the tongue.

Supplementary Figure 14. Cicatricial pemphigoid.

Supplementary Figure 15. Traumatic ulceration.

Supplementary Figure 16. Chemical burn of the tongue (NaOH2).

Supplementary Figure 17. Aphthous ulcer located on the lateral border and the ventral surface of the tongue.

Supplementary Figure 18. Neutropenic ulceration.

Supplementary Figure 19. Squamous cell carcinoma at the lateral border of the tongue.

Supplementary Figure 20. Lingual lichen planus.

Supplementary Figure 21. Uraemic hyperkeratotic stomatitis.

Supplementary Figure 22. Advanced stage lingual amyloidosis.

Supplementary Figure 23. Lingual abscess on the ventral side in a diabetic patient.

