



Glossitis: Clinical Etiologies, Diagnostic Evaluation, and Multidisciplinary Management in Dental and Medical Practice

Turki Abdullah Altowairqi⁽¹⁾, Abdulsalam Masad Alanazi⁽²⁾, Hani Awad Alasmari⁽³⁾, Qassim Arar Ahmed Alhaqawi⁽⁴⁾, Ahmed Suliman Alfaifi⁽⁵⁾, Bandar Jahaz Alahmadi⁽⁶⁾, Aisha Hamad Mahmoodi⁽⁷⁾, Mansour Mayudh S Alharthi⁽⁸⁾, Mutab Mohammed Alqahtani⁽⁹⁾

(1) Wedakh Primary Health Center – Nifi General Hospital, Ministry of Health, Saudi Arabia,

(2) King Fahd Specialist Hospital – Buraydah, Ministry of Health, Saudi Arabia,

(3) Ministry of Health Branch – Riyadh, Saudi Arabia,

(4) Al-Haram Hospital – Madinah, Ministry of Health, Saudi Arabia,

(5) Hospital – Southern Region, Faifa Governorate, Ministry of Health, Saudi Arabia,

(6) Ministry of Health Branch in Madinah, Saudi Arabia,

(7) Bahra'a Primary Health Care (PHC), Ministry of Health, Saudi Arabia,

(8) Hospital Southern Region – Faifa Governorate (Health Administration), Ministry of Health, Saudi Arabia,

(9) Dammam Prison Health Center Medical Services, Ministry of Interior, Saudi Arabia

Abstract

Background: Glossitis, defined as inflammation of the tongue, is a common clinical presentation with a broad differential diagnosis. It manifests through symptoms like pain, burning, and changes in tongue morphology, such as depapillation, erythema, or swelling. The condition often serves as a visible indicator of underlying systemic disorders, making its evaluation a critical component of both dental and medical practice.

Aim: This review aims to synthesize the diverse etiologies, systematic diagnostic approach, and multidisciplinary management strategies for glossitis, emphasizing its role as a sentinel sign of local and systemic pathology.

Methods: A comprehensive analysis of the literature was conducted, integrating data on anatomy, pathophysiology, clinical classification, and evidence-based management. The diagnostic evaluation and therapeutic principles are drawn from both dental and medical perspectives.

Results: Glossitis has multifactorial etiologies, including nutritional deficiencies (iron, B vitamins), infections (Candida, viral), medication reactions, autoimmune diseases, and local irritants. Clinical patterns—such as atrophic, median rhomboid, geographic, and strawberry tongue—guide the diagnostic workup. Evaluation hinges on a detailed history and physical exam, supplemented by targeted laboratory tests (e.g., CBC, vitamin levels) and biopsy for persistent or suspicious lesions to rule out malignancy. Management is etiology-specific: correcting deficiencies, treating infections, removing offending medications, and providing symptomatic relief with topical therapies.

Conclusion: Glossitis requires a systematic, interdisciplinary diagnostic approach to identify its often-systemic cause. Successful management depends on accurate etiology determination and tailored treatment, ranging from simple nutritional supplementation to addressing complex autoimmune conditions.

Keywords: Glossitis, Tongue inflammation, Geographic tongue, Atrophic glossitis, Nutritional deficiency, Oral diagnosis, Multidisciplinary management, Systemic disease.

Introduction

The tongue is a prominently visible structure within the oral cavity, and patients often become aware of alterations in its appearance or sensation, prompting them to seek clinical evaluation. In many instances, such patients may present directly to medical practitioners or be referred to following an initial assessment by dental professionals. Careful inspection of the tongue, together with a systematic examination of the oral mucosa, represents an essential component of a comprehensive physical examination. This is particularly important because a

wide range of systemic diseases and nutritional, infectious, autoimmune, or hematologic disorders may first manifest with signs or symptoms in the oral cavity. Consequently, detailed assessment of the tongue can provide valuable diagnostic clues and contribute significantly to the early detection of underlying systemic pathology. Glossitis is a broad clinical term that refers to inflammatory conditions affecting the tongue. This entity encompasses a spectrum of presentations, which may vary in severity and chronicity.[1] Clinically, glossitis may manifest as pain, burning, or tenderness of the

tongue, leading to discomfort during speaking, swallowing, or eating. In addition to subjective symptoms, objective alterations in the tongue's surface are frequently observed. These changes can involve modification of the normal texture, such as smoothing, atrophy, or prominence of papillae, as well as alterations in coloration, including erythema, pallor, or other discolorations. In many cases, patients exhibit a combination of both symptomatic and morphological changes. Recognition of these features during routine oral examination is therefore crucial, as glossitis may represent not only a localized inflammatory process but also a visible indicator of broader systemic disease processes that warrant further investigation and targeted management.[1]

Anatomy

The tongue is a highly specialized muscular organ occupying a central position within the oral cavity and playing a pivotal role in the coordination of swallowing, articulation of speech, mastication, and taste perception.[2] Structurally, it is composed predominantly of skeletal muscle arranged in intricate bundles that allow for a wide range of precise and finely controlled movements. These muscular components are enveloped by a mucous membrane that differs in character between its dorsal and ventral surfaces, reflecting the functional specializations of each region. The tongue also contributes to oral sealing, bolus manipulation, and the initiation of the swallowing reflex, thereby integrating both neuromuscular and sensory functions essential for normal oropharyngeal physiology.[2] From an embryological perspective, the tongue develops primarily from a median triangular elevation in the floor of the primitive pharynx known as the median lingual swelling, which arises in association with the first pharyngeal (mandibular) arch.[3] This structure, together with lateral lingual swellings and contributions from posterior pharyngeal arches, undergoes complex fusion and differentiation to form the mature tongue. The anterior two-thirds of the tongue is derived mainly from the first pharyngeal arch, whereas the posterior third originates from structures associated with the third and part of the fourth arches, a developmental distinction that underlies its differentiated sensory innervation and mucosal characteristics.[3]

The muscular architecture of the tongue consists of both intrinsic and extrinsic skeletal muscles. Intrinsic muscles are confined entirely within the tongue and are responsible for altering its shape, such as shortening, elongating, narrowing, or flattening. Extrinsic muscles, which include the genioglossus, hyoglossus, styloglossus, and palatoglossus, originate from bony or fibrous structures outside the tongue and insert into its substance, effecting gross positional movements such as protrusion, retraction, elevation, and depression. Motor innervation to nearly all intrinsic and extrinsic tongue muscles is provided by the hypoglossal nerve (cranial nerve XII), reflecting the tongue's dependence on precise neuromuscular control for its complex functions. The sole exception is the palatoglossus muscle, which receives its motor supply via the vagus nerve (cranial nerve X), in association with the pharyngeal plexus.[2] The sensory innervation of the tongue reflects its dual embryologic origin. The anterior two-thirds of the tongue, which lies anterior to the sulcus terminalis, receives general somatic sensation (touch, pain, and temperature) through the lingual nerve, a branch of the mandibular division of the trigeminal nerve. Special sensory innervation for taste in this same region is mediated by the chorda tympani, a branch of the facial nerve that joins the lingual nerve and conveys gustatory information from taste buds located predominantly within specific papillae.[2] In contrast, the posterior one-third of the tongue is supplied for both general and special (taste) sensation primarily by the glossopharyngeal nerve, which also contributes to the gag reflex and integrates with other pharyngeal sensory inputs.

The dorsal surface of the tongue is divided into anterior and posterior regions by an anatomic landmark known as the sulcus terminalis, a V-shaped groove whose apex points posteriorly toward the foramen cecum. The median sulcus runs longitudinally from the tip (apex) of the tongue toward the foramen cecum, reflecting the underlying fusion of embryologic components. These sulci not only serve as important clinical landmarks but also demarcate regions that differ in epithelial type, papillary distribution, and functional specialization.[3] Distributed over the anterior two-thirds of the dorsal tongue are multiple papillae, which are small mucosal projections that increase surface area and, in some types, house taste buds. They are commonly classified into several morphological types. Filiform papillae constitute the most numerous category and are uniformly arranged across much of the dorsal surface. These papillae are slender, conical, and typically measure approximately 1 to 2 mm in height; they are keratinized and do not contain taste buds, instead contributing primarily to mechanical functions such as friction, bolus manipulation, and cleansing of the oral cavity.[1] A subset of these papillae may appear more pointed and

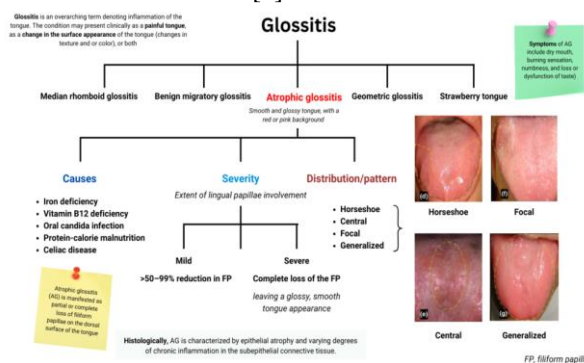


Fig. 1: Glossitis Overview.

give the dorsal tongue its characteristic roughened texture, facilitating efficient licking and mastication by providing traction against food boluses and the palate. Fungiform papillae represent another important category and are scattered predominantly among the filiform papillae on the anterior portion of the tongue, with higher density near the tip and lateral margins. Clinically, they are recognized by their dome-shaped morphology and distinct reddish coloration, which reflects their thinner epithelium and rich vascular supply.[1] Unlike filiform papillae, fungiform papillae typically contain taste buds on their superior surfaces, making them important sites for gustatory perception, particularly for sweet, salty, and umami stimuli. Posteriorly, near the sulcus terminalis, additional specialized papillae (such as circumvallate papillae) and associated taste buds contribute significantly to taste sensation, although these were not detailed in the original description.

The posterior third of the dorsal tongue differs from the anterior portion in that it lacks the dense papillary covering seen anteriorly and instead contains substantial lymphoid tissue, collectively referred to as the lingual tonsils.[1] This lymphoid aggregation forms part of Waldeyer's ring and plays a role in local immune defense, participating in antigen sampling and immune responses to ingested or inhaled pathogens. The mucosa overlying this region may appear nodular because of the underlying lymphoid follicles and associated crypts. The ventral surface of the tongue presents a smoother mucosal lining and is characterized by visible lingual veins coursing near the mucosal surface, which may be useful landmarks in certain clinical and surgical procedures. A midline mucosal fold, the lingual frenulum, extends from the ventral surface of the tongue to the anterior floor of the mouth, helping to anchor the tongue while still permitting a considerable degree of mobility.[1] Variations in the length, thickness, or position of the frenulum can have functional implications, such as restricted tongue movement (ankyloglossia), which may affect speech and feeding. In close anatomical relationship to the lingual frenulum on the floor of the mouth are the openings of the submandibular ducts (Wharton's ducts), which run approximately parallel to the frenulum before opening near its base.[1] These ducts convey saliva from the submandibular glands into the oral cavity, contributing to lubrication, digestion, and maintenance of oral health. The coordinated interaction of salivary flow, tongue movement, and oral musculature is essential for efficient bolus formation and swallowing, underscoring the central functional importance of the tongue within the stomatognathic system.

Etiology

Glossitis is a multifactorial condition with a broad spectrum of underlying etiologies, encompassing hematologic, nutritional, infectious,

pharmacologic, and idiopathic or systemic causes. Among the most prominent contributors are hematological disorders, particularly various forms of anemia. Iron-deficiency anemia is frequently implicated, as iron plays a vital role in epithelial integrity and cellular proliferation; its deficiency can lead to atrophy of lingual papillae, mucosal thinning, and increased susceptibility to inflammation. Pernicious anemia, which results from vitamin B12 malabsorption due to autoimmune destruction of intrinsic factor-producing cells, is another well-recognized cause of glossitis.[4] In this context, patients may present with a smooth, erythematous, and painful tongue, often described as "beefy red," reflecting both epithelial atrophy and inflammatory changes. These hematologic etiologies highlight the importance of systemic evaluation in patients presenting with persistent or unexplained glossitis. Nutritional deficiencies, particularly of B-complex vitamins, represent another crucial etiologic group. Deficiencies of vitamin B1 (thiamine), B2 (riboflavin), B3 (niacin), B6 (pyridoxine), B9 (folate), and B12 (cobalamin) have all been associated with changes in the tongue, including erythema, swelling, atrophy of papillae, and burning sensations.[5] These vitamins are essential cofactors in a variety of metabolic pathways, and their deficiency results in impaired cellular turnover and repair within the rapidly renewing oral mucosa. For example, riboflavin and niacin deficiencies are classically associated with angular cheilitis and glossitis, while folate and vitamin B12 deficiencies are more commonly linked to megaloblastic changes in the bone marrow and concurrent mucosal atrophy. The overlapping clinical manifestations of these deficiencies may complicate diagnosis; however, recognition of glossitis as an early sign can prompt timely investigation of dietary habits, gastrointestinal absorption, and systemic disease.

Infectious agents also contribute significantly to the etiological spectrum of glossitis. Viral infections, particularly those caused by herpes viruses, may precipitate acute inflammatory changes in the tongue, sometimes in the context of primary herpetic stomatitis or as part of a reactivation phenomenon. Post-herpetic glossitis can persist after resolution of the primary lesions, leaving residual mucosal sensitivity or surface changes. Bacterial infection of the tongue is relatively uncommon in immunocompetent individuals but may occur secondary to trauma, poor oral hygiene, or systemic conditions that compromise host defenses, and is more frequently observed in immunosuppressed patients.[6] Fungal infections, most notably those caused by *Candida* species, are among the most common infectious causes of glossitis.[7] *Candida*-associated glossitis may present with erythema, depapillation, or superimposed pseudomembranous plaques and is often associated with predisposing

factors such as xerostomia, diabetes mellitus, prolonged antibiotic use, or denture wearing. Parasitic infections, including malaria and spirochetal infections, have also been implicated, albeit less commonly, in the pathogenesis of glossitis, underscoring the need to consider geographic, epidemiological, and travel histories in the diagnostic workup.[8][9]

Pharmacologic agents represent another important category in the etiology of glossitis, as a range of medications can induce direct mucosal irritation, allergic reactions, or secondary changes in the oral environment. Angiotensin-converting enzyme (ACE) inhibitors have been associated with angioedema and, less commonly, inflammatory changes of the oral mucosa, including the tongue. Bronchodilator medications such as albuterol may alter salivary flow or pH, predisposing to mucosal irritation in susceptible individuals. Organosulfur antimicrobial drugs, including sulphanilamide and sulphathiazole, have been reported to provoke hypersensitivity reactions that can manifest as glossitis or stomatitis.[10] Hormonal agents, notably oral contraceptive pills, have also been linked with oral mucosal changes, possibly through alterations in vascular permeability, immune modulation, or shifts in the oral microbiome.[11] Additionally, lithium carbonate, widely used in the management of bipolar affective disorder, has been associated with various oral side effects, including glossitis, xerostomia, and altered taste perception, which may exacerbate mucosal vulnerability.[11] Recognition of medication-related glossitis is critical, as modification or discontinuation of the offending agent can lead to resolution of symptoms. Beyond these well-defined categories, a range of other factors can contribute to the development or persistence of glossitis. Psychological factors, such as anxiety disorders and conversion disorders, may play a role in symptom perception and chronicity, particularly in patients with burning sensations of the tongue in the absence of clear clinical abnormalities. Exposure to local irritants, including alcohol, spicy foods, and tobacco, can induce direct chemical and thermal injury to the lingual epithelium, predisposing to inflammation and discomfort. Normal familial or constitutional variants of tongue morphology, such as fissured tongue and geographic tongue, are typically benign but may become symptomatic or secondarily inflamed, mimicking or exacerbating glossitis in some individuals. Mechanical irritation from accidental burns, ill-fitting dental prostheses, sharp teeth, or chronic dental trauma can likewise damage the mucosal surface, leading to localized or diffuse inflammatory changes.

Systemic and developmental conditions also figure prominently in the etiologic landscape of glossitis. Poor hydration can reduce salivary flow, alter oral lubrication, and disturb the homeostasis of the oral environment, increasing frictional trauma and

susceptibility to infection. Individuals with Down syndrome often present with characteristic craniofacial and oral features, including macroglossia and fissured tongue, which may be accompanied by recurrent inflammation or secondary infections. Autoimmune diseases, such as psoriasis and other immune-mediated conditions, may involve the oral mucosa and tongue, producing erythematous, desquamative, or ulcerative lesions that fall within the spectrum of glossitis. Finally, burning mouth syndrome, a chronic pain condition characterized by burning sensations in the tongue and oral mucosa without obvious clinical abnormalities, may present with symptoms overlapping those of glossitis. Although its pathophysiology is not fully understood, it is thought to involve neuropathic and psychogenic components and may coexist with subtle mucosal changes or precipitating local factors. Collectively, these diverse etiologies highlight the importance of a thorough and systematic approach to the evaluation of glossitis. Detailed history-taking, including dietary assessment, medication review, psychosocial evaluation, and inquiry into systemic symptoms, combined with careful clinical examination and appropriate laboratory or microbiological investigations, is essential for identifying the underlying cause. Such an approach not only facilitates targeted and effective management but also enables early recognition of potentially serious systemic conditions that may first manifest through changes in the tongue [8][9][10][11].

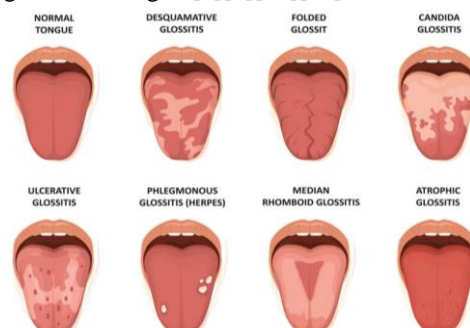


Fig. 2: Types of glossitis.

Epidemiology

The epidemiology of glossitis is challenging to delineate with precision because the condition encompasses a wide array of underlying etiologies, each with distinct prevalence patterns that vary geographically, nutritionally, and socioeconomically. Glossitis is not a single disease entity but rather a clinical manifestation secondary to diverse systemic, nutritional, infectious, pharmacologic, or idiopathic factors. Consequently, population-based data often reflect the prevalence of these underlying causes rather than glossitis itself. For example, vitamin deficiencies—particularly deficiencies in B-complex vitamins and iron—remain common in regions where malnutrition is prevalent, inadequate dietary intake persists, or conditions affecting nutrient absorption

are widespread. In such settings, glossitis may occur with greater frequency and may serve as an early mucosal indicator of broader nutritional compromise. Because treatment of the underlying deficiency typically leads to remission of lingual inflammation, the transient nature of many cases further complicates efforts to gather consistent epidemiological data. Infectious etiologies likewise contribute variably to the global pattern of glossitis. Infections caused by *Candida* species, herpes viruses, and, less commonly, bacterial or parasitic organisms may result in acute or chronic inflammation of the tongue. The frequency of infectious glossitis correlates with factors such as immunosuppression, antibiotic exposure, oral hygiene, and regional disease burden. Importantly, glossitis attributable to infectious organisms generally resolves once the causative pathogen is appropriately treated, again limiting long-term prevalence assessment. Medication-induced glossitis presents an additional epidemiologic challenge, as it depends on drug utilization patterns within a given population. Glossitis resulting from pharmacologic agents commonly improves after withdrawal of the offending medication, thus contributing further to the temporary and fluctuating nature of reported cases. Despite these limitations, some representative data provide insight into the broader prevalence of tongue-related mucosal lesions. Findings from the Third National Health and Nutrition Examination Survey (NHANES III), which assessed oral mucosal conditions in 17,235 adult participants, revealed that 27.9% exhibited a total of 6,003 clinically identifiable oral lesions. Of these, 14.2% were located on the dorsal surface of the tongue, while 1.3% were confined to the lateral borders. Specific conditions categorized under glossitis also demonstrated quantifiable prevalence. Geographic tongue, also known as benign migratory glossitis, exhibited a prevalence range of 1.41% to 2.29%, reflecting its relatively common occurrence in the general population. By contrast, median rhomboid glossitis—a chronic inflammatory condition associated frequently with *Candida* infection—showed a lower prevalence range of 0.46% to 0.30%, indicating its relative infrequency.[12] These epidemiologic patterns underscore the complex and multifactorial nature of glossitis. Variability in causative factors across populations, the transient course of many forms, and the lack of uniform diagnostic criteria contribute to the difficulty of establishing definitive global prevalence estimates. Nonetheless, available data suggest that tongue lesions, including certain forms of glossitis, represent a meaningful proportion of oral mucosal abnormalities encountered in clinical practice [11][12].

Pathophysiology

The pathophysiological mechanisms underlying geographic tongue, also known as benign

migratory glossitis, remain incompletely understood despite its relatively common clinical occurrence. Current evidence suggests that the condition arises from a complex interplay of genetic, inflammatory, environmental, and psychosomatic factors. One of the hallmark features of geographic tongue is the presence of transient, migratory areas of depapillation affecting primarily the filiform papillae on the dorsal surface of the tongue. These depapillated regions appear erythematous and smooth, bordered by slightly elevated, white or pale serpiginous margins. The cyclic loss and regeneration of filiform papillae reflect localized disturbances in epithelial differentiation and keratinization, yet the precise molecular triggers for these changes remain uncertain. Inflammatory processes are thought to play a central role in the development of geographic tongue. Histopathological studies have demonstrated features resembling psoriasiform mucositis, including neutrophilic microabscesses, elongated rete ridges, and increased turnover of epithelial cells. These findings have prompted speculation about a pathophysiologic relationship between geographic tongue and psoriasis, and indeed, an increased prevalence of geographic tongue has been observed among individuals with psoriatic disease. However, the association is not universal, and geographic tongue occurs frequently in individuals without any cutaneous manifestations, indicating that additional mechanisms must be involved. Psychological factors, particularly emotional stress, have been consistently reported as exacerbating influences. Stress is known to modulate immune responses, alter mucosal barrier function, and increase susceptibility to inflammatory flare-ups. Patients often describe heightened symptoms during periods of psychological strain, supporting the role of neuroimmune pathways in disease activity. Dietary triggers also figure prominently in symptom fluctuation. Many patients report that consumption of acidic, hot, or spicy foods provokes discomfort or intensifies the appearance of lesions, suggesting that altered mucosal sensitivity or compromised epithelial integrity may lower the threshold for irritation.[13]

Environmental and systemic influences, including nutritional deficiencies, hormonal fluctuations, and allergic tendencies, have been proposed as contributing factors, although definitive causal relationships remain elusive. The heterogeneity of clinical presentations further complicates efforts to identify a singular pathogenic pathway. Instead, geographic tongue is increasingly conceptualized as a multifactorial condition in which genetic predisposition, localized epithelial dysregulation, immune-mediated mechanisms, and external triggers converge to produce the characteristic migratory lesions. Ongoing research continues to refine understanding of these interactions, but the condition remains clinically

diagnosed and primarily managed through symptom control rather than targeted interventions [13].

History and Physical

When assessing a patient with glossitis, obtaining a detailed and structured clinical history is fundamental to accurate diagnosis and appropriate management. The history should begin with an exploration of the patient's overall nutritional status, including typical dietary patterns, any deliberate dietary restrictions, and possible malabsorptive conditions that may predispose to deficiencies in iron, B-complex vitamins, or other nutrients known to contribute to glossitis. Particular attention should be given to the intake of fresh fruits, vegetables, protein, and fortified foods, as well as to any history of weight loss, gastrointestinal surgery, chronic diarrhea, or other features suggestive of malnutrition or malabsorption. In parallel, it is essential to document tobacco use in all forms, alcohol consumption, and exposure to other oral irritants, as these factors may both precipitate and perpetuate mucosal inflammation, alter salivary flow, and contribute to coexisting oral pathology. The clinician should also inquire about fluctuations in symptoms in relation to specific foods or environmental exposures. Many patients report worsening pain, burning, or sensitivity of the tongue when consuming hot, acidic, or spicy foods, suggesting a compromised mucosal barrier that responds adversely to chemical and thermal stimuli. A comprehensive past medical history is equally important. This should encompass known immunosuppressive conditions such as HIV infection, hematologic malignancies, or the use of systemic immunosuppressive agents, as well as a history of solid organ or bone marrow transplantation. A documented history of cancer is relevant both for its potential direct effects and for the implications of chemotherapy or radiotherapy on oral tissues. Autoimmune diseases, including conditions such as Sjögren syndrome, systemic lupus erythematosus, and psoriasis, should be specifically explored, as should endocrine disorders such as diabetes mellitus and thyroid disease, all of which may influence oral health and predispose to glossitis or secondary infections [12][13].

Medication history is another critical component of the evaluation. The patient's current daily medications, including over-the-counter drugs, herbal supplements, and recent short-term prescriptions, should be reviewed meticulously. Any recent initiation, discontinuation, or dosage adjustment should be correlated temporally with the onset or progression of glossitis symptoms, as numerous agents have been implicated in mucosal changes, xerostomia, or hypersensitivity phenomena. The most commonly reported symptoms in glossitis include pain or burning of the tongue, diffuse or localized redness, swelling, loss or flattening of lingual papillae, and the development of new visible lesions on the tongue surface. Patients may also

report difficulties with eating, speaking, or swallowing, as well as altered taste perception or dysgeusia. Because some forms of glossitis, such as benign migratory glossitis, may have a familial component, the history should include specific questions about similar tongue changes in first-degree relatives, particularly recurrent or migratory lesions suggestive of a hereditary predisposition. The physical examination of the tongue and oral cavity is an indispensable part of the overall clinical assessment. Inspection should be systematic and conducted in good lighting, ideally with the assistance of tongue depressors and gauze to allow adequate visualization and manipulation of the tongue. The dorsal and ventral surfaces of the tongue must be examined carefully, with attention to color, moisture, presence or absence of papillae, and any focal or diffuse abnormalities. The mucosa should be assessed for whether it appears wet and adequately lubricated or dry and desiccated, and for changes such as erythema, leukoplakic patches, ulcerations, or exophytic lesions. The dental status must be evaluated concurrently, as chronic friction from sharp, broken, or malpositioned teeth, as well as from ill-fitting dentures or other prostheses, can cause traumatic mucosal alterations and ulcerations that may mimic or exacerbate primary tongue pathology. Distinguishing between frictional keratosis, traumatic ulceration, and intrinsic glossitic processes is essential to avoid misdiagnosis [13].

Palpation of the tongue and the floor of the mouth is also crucial. The examiner should gently palpate the entire mobile tongue, including its lateral borders, to identify any areas of tenderness, induration, or submucosal masses. The floor of the mouth should be palpated bimanually to detect salivary gland enlargement, sialoliths, or other soft tissue lesions. A comprehensive head and neck examination is required, with particular focus on regional lymph nodes. The presence of cervical lymphadenopathy may suggest infectious, inflammatory, or neoplastic processes and must be interpreted in conjunction with the intraoral findings. Various clinical subtypes of glossitis exhibit characteristic patterns on examination. Atrophic glossitis, for example, is typified by a diffusely erythematous tongue with a striking reduction or complete absence of lingual papillae, imparting a smooth, shiny, and often dry appearance to the dorsal surface.[14] The tongue may appear thinner and more atrophic overall, and patients frequently complain of burning pain and heightened sensitivity. This pattern is classically associated with nutritional deficiencies, particularly of iron, folate, and vitamin B12, as well as with certain systemic diseases, making its recognition an important diagnostic clue.[14] Median rhomboid glossitis presents as a central, often symmetrical, rhomboid- or ovoid-shaped area on the midline of the dorsal tongue, typically located anterior to the circumvallate papillae. The lesion may

be hyperkeratotic or erythematous and plaque-like in appearance and is sometimes tender to palpation. This condition is frequently associated with chronic *Candida* infection and is thought to represent a localized inflammatory response in a region of developmental susceptibility. Awareness of its characteristic location and morphology helps differentiate it from other midline tongue lesions [14].

Benign migratory glossitis, commonly termed geographic tongue, is characterized by multiple, irregularly shaped areas of depapillation that create smooth, erythematous patches with a thin, slightly raised whitish or yellowish border. These areas can change in number, size, and position over time, giving the impression of migration across the dorsal tongue surface. Lesions may be asymptomatic or may cause discomfort, particularly with certain foods. Importantly, benign migratory glossitis must be distinguished from stable leukoplakic or erythroleukoplakic lesions, which do not migrate and may represent premalignant or malignant conditions requiring biopsy and closer surveillance. Geometric glossitis manifests as painful, linear fissures and grooves traversing the mobile tongue. These fissures can vary in depth and configuration and may collect debris or become secondarily inflamed or infected, leading to further discomfort. Careful cleansing and management of predisposing factors are often necessary in such cases. Strawberry tongue is another descriptive term used in specific clinical contexts, such as scarlet fever or Kawasaki disease, and is characterized by a red, denuded appearance of the dorsal tongue with prominent, persistently hypertrophic fungiform papillae protruding against the erythematous background.[15] This striking appearance reflects both mucosal inflammation and selective papillary hypertrophy and should prompt consideration of the associated systemic conditions in the appropriate clinical setting. In summary, meticulous history-taking combined with a detailed and systematic examination of the tongue and oral cavity allows the clinician not only to characterize glossitis accurately but also to identify underlying local and systemic factors. This integrated approach is essential for formulating an appropriate differential diagnosis, guiding further investigations, and instituting targeted therapy [13][14][15].

Evaluation

The evaluation of glossitis relies fundamentally on a comprehensive history and meticulous physical examination, both of which serve as the cornerstone for determining the underlying cause and guiding further diagnostic testing. Because glossitis represents a clinical manifestation rather than a single disease entity, the diagnostic approach must be individualized and informed by the patient's symptoms, medical history, risk factors, and physical findings. A detailed history focusing on nutritional

status, systemic illness, medication use, environmental exposures, and associated oral or systemic symptoms provides essential clues to potential etiologies. The physical examination then allows direct evaluation of the appearance, texture, and surface characteristics of the tongue and helps identify patterns suggestive of specific clinical subtypes such as atrophic glossitis, geographic tongue, or median rhomboid glossitis. Nevertheless, when history and examination do not yield a clear diagnosis, additional studies may be warranted to refine the differential diagnosis and guide management. Biopsy is an important diagnostic tool in select circumstances. Although it is not routinely required for all cases of glossitis, it becomes particularly valuable when the clinical presentation raises concerns for underlying mucocutaneous blistering disorders, such as pemphigus vulgaris or bullous pemphigoid. In such cases, histopathology and direct immunofluorescence can confirm the diagnosis and distinguish between autoimmune blistering diseases that may manifest with erythema, erosions, or ulcerations on the tongue. Biopsy is also essential when the clinician identifies a stable, non-migratory lesion or any area with concerning features such as induration, ulceration, leukoplakic or erythroplakic changes, or persistent surface abnormalities. These features may indicate premalignant or malignant pathology, and tissue sampling is critical to rule out dysplasia or oral squamous cell carcinoma.[1] In this context, early biopsy can substantially alter the clinical trajectory by facilitating timely diagnosis and treatment [1].

Laboratory studies play an important adjunctive role when the clinical history suggests systemic involvement. Because nutritional deficiencies are common causes of glossitis, serum levels of iron, folate, and B-complex vitamins—including B1, B2, B3, B6, B9, and B12—are particularly informative in patients at high risk for malnutrition, malabsorption, or restrictive dietary practices. In geographic areas where nutritional deficiencies are endemic or among individuals with known high-risk conditions, assessment of vitamin levels may be imperative to guide replacement therapy. Autoimmune and rheumatologic conditions, such as Sjögren syndrome, systemic lupus erythematosus, or systemic sclerosis, may also present with glossitis or related mucosal changes. In these cases, laboratory evaluation may include rheumatoid factor, anti-Ro (SSA), anti-La (SSB), erythrocyte sedimentation rate, C-reactive protein, or additional targeted serologic studies depending on the suspected disorder. These tests can help identify underlying systemic inflammation or autoimmune activity that contributes to lingual mucosal alterations. Because glossitis is sometimes associated with immunosuppression, a complete blood count can help detect anemia, leukopenia, or other hematologic

abnormalities, while HIV testing is warranted when risk factors or opportunistic infections are present. Secondary infections, particularly candidiasis, are more prevalent among immunocompromised individuals, and identifying underlying immunodeficiency is essential to effective treatment. Likewise, endocrine abnormalities may influence oral mucosal health; hemoglobin A1c levels can help evaluate glycemic control in suspected diabetes mellitus, while thyroid function tests may identify hypothyroidism or hyperthyroidism, both of which can contribute to mucosal atrophy or altered epithelial turnover [1][3].

Imaging is generally not part of the routine evaluation of glossitis. Because glossitis typically reflects mucosal or superficial inflammatory processes rather than deep structural abnormalities, imaging studies rarely offer additional diagnostic value beyond what can be obtained from history, examination, and laboratory testing. However, when malignancy is suspected based on persistent lesions, induration, regional lymphadenopathy, or other concerning features, cross-sectional imaging becomes critical. Computed tomography (CT) of the neck with intravenous contrast is typically indicated in such cases to assess the extent of disease, involvement of adjacent structures, and regional lymph node status. Imaging may also be employed in the context of biopsy-confirmed malignancy to guide staging and inform treatment planning. Overall, the evaluation of glossitis is best approached through a systematic and targeted diagnostic framework. While many cases can be attributed to benign and reversible causes such as nutritional deficiencies, infections, or medication effects, others may reflect significant underlying systemic disease or herald early malignant transformation. A combination of thorough clinical assessment, judicious use of laboratory studies, and selective biopsy or imaging enables clinicians to differentiate among these possibilities and implement appropriate therapeutic strategies [1].

Treatment / Management

The management of glossitis is highly dependent on the underlying etiology, as the condition itself is a clinical manifestation rather than a singular disease process. In many cases, glossitis is mild, transient, and self-limiting, resolving spontaneously once the precipitating factor is removed or treated. Because of this, the initial approach centers on optimizing oral hygiene, reducing exposure to local irritants, and providing symptomatic relief. Good oral hygiene practices—including regular brushing, flossing, and the use of non-irritating alcohol-free mouthwashes—can help reduce local inflammation and minimize discomfort. Many patients experience relief with formulations of compounded “magic mouthwash,” which may include corticosteroids, antihistamines, antacids, or lidocaine. These mouth rinses serve to soothe the inflamed mucosa, reduce pain, and provide temporary

symptomatic benefit, particularly during acute exacerbations of migratory glossitis or other inflammatory variants. Management becomes more targeted when specific subtypes of glossitis are identified. Atrophic glossitis, which is frequently associated with nutritional deficiencies, requires correction of the underlying deficit. In cases of vitamin B12 deficiency, intramuscular injections of cobalamin are often necessary to rapidly replenish stores and restore normal epithelial turnover. Treatment of iron or folate deficiency similarly involves targeted supplementation. Identification and correction of nutritional causes not only improve glossitis but also address potentially serious systemic implications. Median rhomboid glossitis, commonly linked to chronic *Candida* infection, typically requires antifungal therapy only if the lesion is symptomatic. Nystatin swish-and-swallow therapy is a commonly employed treatment, helping to reduce fungal load and alleviate discomfort. In asymptomatic patients, observation and reassurance are generally sufficient, as this variant often persists without causing significant morbidity [12][13].

Benign migratory glossitis, or geographic tongue, usually requires no medical intervention. Because the condition is benign and tends to fluctuate in appearance, reassurance is central to management. For patients experiencing burning, sensitivity, or discomfort during flares, mouth rinses or topical anesthetics can provide relief. Avoidance of dietary triggers—particularly acidic, spicy, or hot foods—may also help minimize symptoms. Geometric glossitis, characterized by painful fissures across the mobile tongue, is likewise managed primarily with reassurance and supportive care. Although antiviral therapy has been attempted in acute episodes, the overall success remains limited, and treatment is largely focused on symptom reduction and elimination of local irritants. Strawberry tongue, observed in conditions such as nutritional deficiencies or systemic inflammatory diseases, may improve with vitamin B12 supplementation when associated with cobalamin deficiency. Treatment of the underlying systemic illness remains essential to resolving the oral manifestations. Medication-induced glossitis requires identification and discontinuation of the offending drug whenever possible. Review of the patient’s medication list is crucial, as many cases resolve once the causative agent is withdrawn. Substitution with an alternative therapy should be considered in consultation with the prescribing clinician. Infectious causes of glossitis should be treated with antimicrobial therapy tailored to the specific pathogen. Acute bacterial, fungal, viral, or parasitic infections require appropriate pharmacologic management to eliminate the organism and reduce mucosal inflammation. In cases where glossitis occurs in the context of immunosuppression—such as HIV infection or uncontrolled diabetes—management must also

address the underlying immune dysfunction. An immunologic workup may be necessary to identify opportunistic pathogens, and optimal glycemic control is essential to improving oral mucosal health and reducing recurrent infections. Ultimately, successful management of glossitis requires a comprehensive, individualized, and etiology-driven approach. By combining targeted therapy with supportive care, clinicians can effectively alleviate symptoms, correct underlying causes, and prevent recurrence, ensuring both oral comfort and overall health [1][12][13][14].

Differential Diagnosis

The differential diagnosis of glossitis is notably extensive, reflecting the wide range of systemic, nutritional, infectious, immunologic, and idiopathic conditions that can manifest with inflammation or alterations of the tongue. Because glossitis is a descriptive clinical finding rather than a discrete disease, identifying the correct underlying cause requires careful correlation of history, clinical examination, and, when appropriate, laboratory or histopathological studies. The physical examination provides an important framework for narrowing the possibilities, as characteristic patterns—such as atrophy, migratory lesions, papillary hypertrophy, or midline plaques—tend to align with specific diagnostic categories. Nonetheless, considerable overlap exists among many conditions, and clinicians must approach the differential diagnosis with both breadth and precision. A normal-appearing tongue accompanied by burning, stinging, or dysesthetic sensations presents a distinct diagnostic challenge. In such cases, burning mouth syndrome is an important consideration, particularly in postmenopausal women or individuals experiencing psychological stress, as the condition involves neuropathic pain without overt mucosal abnormalities. Diabetic neuropathy can similarly produce burning or dysesthetic oral pain, often in the context of poorly controlled diabetes mellitus. Post-herpetic glossitis, occurring after herpes zoster or HSV infections, may present with persistent sensitivity in the absence of visible lesions. Acid reflux, particularly laryngopharyngeal reflux, may also cause burning sensations or inflammation of the posterior tongue despite an otherwise normal appearance. In each of these scenarios, the absence of visible tongue pathology necessitates heightened attention to systemic and neurologic factors in the differential diagnosis [15][16].

When the tongue displays atrophic changes—characterized by smoothness, loss of papillae, and erythema—several key etiologies must be considered. Protein-calorie malnutrition and vitamin B12 deficiency remain classic causes of atrophic glossitis, owing to their impact on cellular turnover and epithelial integrity. Bullous diseases, such as pemphigus vulgaris or mucous membrane pemphigoid, may initially present with erosions or

atrophy following blister rupture. Candidiasis can produce erythematous depapillated regions, especially in immunocompromised individuals or those using inhaled corticosteroids. Xerostomia, resulting from medications, Sjögren syndrome, or dehydration, can lead to mucosal dryness and secondary atrophic changes.[16] The presence of atrophy requires careful evaluation for nutritional, autoimmune, or infectious contributors. Lesions consistent with median rhomboid glossitis—classically a midline, depapillated plaque on the dorsal tongue—necessitate differentiation from several conditions. Haemangioma, though less common, may appear as a vascular midline lesion and must be distinguished by color and compressibility. Geographic tongue can occasionally involve the midline, producing overlapping features, although its migratory nature typically sets it apart. Amyloidosis, with its characteristic mucosal deposits, may present firm, waxy plaques on the tongue and carries significant systemic implications. Candidiasis remains a central consideration, given its strong association with median rhomboid glossitis. Importantly, squamous cell carcinoma must always be excluded when a persistent, non-migratory midline lesion is present, especially in patients with risk factors such as tobacco or alcohol use.[17]

A strawberry tongue, featuring erythema with prominent, hypertrophic fungiform papillae, is classically associated with several systemic inflammatory or infectious conditions. Kawasaki disease, particularly in children, often presents with strawberry tongue in the context of fever, conjunctivitis, and lymphadenopathy. Toxic shock syndrome produces similar changes due to toxin-mediated mucosal inflammation. Historically, yellow fever has also been described in association with strawberry tongue, although its relevance depends on epidemiologic exposure.[15] Recognition of a strawberry tongue should prompt urgent evaluation for underlying systemic illness. For patients displaying geographic tongue, also known as benign migratory glossitis, the differential diagnosis includes several inflammatory, infectious, and reactive disorders. Oral lichen planus may exhibit white, reticulated borders or erythematous patches that mimic some features of geographic tongue but tend to be more stable over time. Exposure to chemical or inhalational irritants, particularly tobacco smoke, can cause inflammatory changes that resemble migratory erythematous patches. Dehydration, due to reduced salivary flow, may exacerbate mucosal sensitivity or produce irregular patterns of papillary loss. Candidiasis can produce erythematous or depapillated areas but typically lacks the sharply demarcated migratory borders seen in geographic tongue. Connective tissue diseases, including psoriasis and reactive arthritis, have associations with geographic tongue and may influence its

presentation. Bullous diseases may produce irregular patches following rupture of mucosal blisters, though these lesions generally lack the migratory nature of geographic tongue. Finally, leukoplakia, a potentially premalignant lesion, must be carefully excluded, as its stable, non-migratory white plaques differ from the shifting patterns seen in benign migratory glossitis but may be confused with its borders.[18] In summary, the differential diagnosis of glossitis is inherently wide-ranging and depends heavily on clinical context and examination findings. By categorizing glossitis according to characteristic patterns—normal-appearing but symptomatic tongue, atrophic changes, midline plaques, papillary hypertrophy, or migratory lesions—clinicians can narrow the diagnostic possibilities and identify appropriate evaluative pathways. A systematic, pattern-based approach ensures accurate diagnosis, timely management, and the exclusion of serious underlying disease, including autoimmune disorders, infections, nutritional deficiencies, and malignancy [18].

Prognosis

The prognosis of glossitis is largely determined by its underlying etiology, as the condition itself represents a clinical sign rather than a distinct disease entity. In many cases, glossitis is benign and self-limiting, resolving completely once precipitating factors—such as nutritional deficiencies, medication-induced irritation, or localized infections—are addressed. Nutritional causes, including deficiencies of iron, folate, or vitamin B12, typically respond well to appropriate supplementation, with gradual restoration of lingual papillae and improvement in symptoms. Similarly, infectious forms of glossitis often resolve following eradication of the causative organism, whether fungal, viral, or bacterial. Medication-related glossitis generally carries an excellent prognosis once the offending agent is discontinued, underscoring the importance of a thorough medication review in evaluation. However, certain variants of glossitis may follow a more chronic or recurrent course. Geographic tongue, for instance, often persists throughout life with periodic flare-ups characterized by migratory, erythematous patches and variable discomfort. Although benign, its unpredictable course may cause ongoing distress for some patients, particularly those highly sensitive to changes in appearance or function. Emotional distress is not uncommon, and anxiolytic therapy may be beneficial when anxiety significantly exacerbates symptom perception or interferes with quality of life. Importantly, the presence of persistent, non-healing lesions must always raise the possibility of premalignant or malignant transformation. In such cases, prompt biopsy is essential for timely diagnosis and treatment, as delayed evaluation can adversely affect outcomes. Overall, while most causes of glossitis have a favorable prognosis, accurate

identification of the etiology and vigilance for malignancy are key determinants of long-term outcomes [17][18].

Complications

Although glossitis is frequently benign, several complications may arise depending on the underlying cause and severity of symptoms. The most common and often underestimated complication is patient anxiety related to the appearance of the tongue. For many individuals, the sudden onset of erythema, depapillation, or ulcer-like lesions on a highly visible and functionally important structure can be alarming. This anxiety may exacerbate subjective discomfort, heighten awareness of symptoms, and worsen the overall disease experience. In cases where reassurance proves challenging, a biopsy may be warranted—not only to rule out malignancy but also to provide psychological reassurance for both patient and clinician. Other complications relate to the functional impact of glossitis. Pain, burning sensations, and mucosal sensitivity can interfere with eating, drinking, and speaking. Chronic glossitis may lead to reduced nutritional intake due to discomfort with acidic, spicy, or hot foods. In severe or prolonged cases, secondary complications such as oral candidiasis may develop, particularly in patients with xerostomia, diabetes, or immunosuppression. Additionally, glossitis associated with systemic disease—such as autoimmune disorders or endocrine imbalance—may reflect broader disease activity, necessitating broader medical management. Although rare, failure to identify malignant lesions mimicking glossitis can delay diagnosis of oral squamous cell carcinoma, underscoring the need for careful assessment of persistent or atypical presentations [18].

Patient Education

Patient education is central to the successful management of glossitis, as many cases require no treatment beyond reassurance and supportive care. Patients should be informed that glossitis is often benign and self-limiting, particularly in forms such as benign migratory glossitis or mild inflammatory variants. Educating patients about the role of good oral hygiene—including regular brushing, avoidance of irritants, and maintenance of adequate hydration—can significantly reduce symptoms and prevent exacerbations. The use of anti-inflammatory or anesthetic mouth rinses may provide symptomatic relief during acute episodes, allowing patients to maintain comfort while the condition resolves. It is crucial to inform patients that any chronic, stable, or progressively worsening lesion warrants further evaluation, including possible biopsy, to exclude malignancy. Patients should understand the importance of monitoring changes in tongue appearance and reporting any persistent abnormalities, such as leukoplakic or erythroplakic patches, which carry a higher risk of dysplastic progression. Those with nutritional deficiencies or

systemic disease should be counseled regarding adherence to supplementation and disease management strategies to prevent recurrence. Ultimately, empowering patients with knowledge about the benign nature of most glossitis cases and the signs that warrant medical attention fosters proactive engagement in their care and reduces unnecessary anxiety.[1]

Enhancing Healthcare Team Outcomes

Optimal management of glossitis is best achieved through an interprofessional approach, as its etiologies span multiple medical disciplines. Physicians play a central role in identifying systemic or nutritional causes, ordering appropriate laboratory testing, and initiating medical management. Dentists and oral health specialists are critical in evaluating local irritants, dental trauma, oral hygiene, and mucosal abnormalities, and in distinguishing benign lesions from those that require biopsy. Nurses contribute by reinforcing oral care instructions, addressing patient anxiety, and monitoring treatment adherence and symptom progression. Pathologists provide essential diagnostic clarification through histopathological evaluation when biopsies are performed, helping to differentiate inflammatory, infectious, autoimmune, and neoplastic causes. Oncologists may become involved when malignant or premalignant lesions are identified, ensuring timely evaluation and comprehensive treatment planning. Through effective communication, shared decision-making, and coordinated care, interprofessional teams can markedly improve diagnostic accuracy, expedite treatment, and enhance the overall patient experience. This collaborative approach ensures that glossitis—whether benign or indicative of systemic disease—is managed efficiently and comprehensively to achieve the best possible outcomes [18].

Conclusion:

In conclusion, glossitis is a clinically significant inflammatory condition of the tongue that frequently signals underlying systemic or local pathology rather than representing a disease in itself. Its diverse etiologies—spanning nutritional deficiencies, infections, pharmacological reactions, and autoimmune disorders—necessitate a meticulous and systematic diagnostic approach. A thorough clinical history and detailed oral examination are paramount, often revealing characteristic patterns like the smooth atrophy of nutritional deficiency or the migratory lesions of geographic tongue. This initial assessment must be complemented by targeted laboratory investigations and, for persistent or atypical lesions, biopsy to exclude premalignant or malignant change. Effective management is fundamentally etiology-driven, requiring collaboration between dental and medical professionals. Treatment may involve nutritional repletion, antimicrobial therapy, discontinuation of

causative medications, or management of underlying systemic diseases. For many benign variants, patient education and reassurance are central components of care. Ultimately, recognizing glossitis as a potential marker of broader health issues underscores the importance of an integrated, interprofessional strategy to ensure accurate diagnosis, appropriate intervention, and optimal patient outcomes.

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