

TRAUMATIC ULCER ON THE LATERAL BORDER OF THE TONGUE: CLINICAL FEATURES, DIAGNOSIS, AND MANAGEMENT

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Abstract

Traumatic ulcers on the lateral border of the tongue are among the most common ulcerative lesions of the oral mucosa, primarily caused by repetitive mechanical irritation. This anatomical area is particularly vulnerable due to its constant mobility and proximity to teeth and dental appliances. While most traumatic ulcers are acute and self-limiting, chronic lesions may persist and clinically mimic oral squamous cell carcinoma, posing significant diagnostic challenges. The etiology involves local irritants such as sharp teeth, fractured restorations, or ill-fitting dentures, as well as systemic factors including immunosuppression, nutritional deficiencies, and infections like syphilis, tuberculosis, or HIV. Diagnosis relies on thorough clinical examination and the elimination of local trauma, with biopsy indicated for ulcers persisting beyond two to three weeks. Management focuses on removing causative factors and using adjunctive therapies such as corticosteroids, hyaluronic acid, and CO₂ laser treatment. Early identification and intervention are essential for optimal prognosis and to prevent malignant transformation.

Keywords: traumatic ulcer, tongue, oral mucosa, diagnosis, management

Introduction

Traumatic ulcerations of the oral mucosa represent one of the most common pathological entities encountered in dental and medical practice, with the lateral border of the tongue being a particularly frequent site due to its anatomical and functional characteristics [1,2]. The constant mobility of the tongue, combined with its close relationship to teeth and prosthetic devices, predisposes this area to repetitive mechanical trauma. These lesions typically present as solitary, painful ulcers that can significantly affect oral function, including speech, mastication, and swallowing, thereby impairing a patient's quality of life [3,4]. While most traumatic ulcers are benign and self-limiting, the lateral border of the tongue has a higher risk of developing persistent or recurrent lesions because of continuous exposure to irritants, making accurate diagnosis and appropriate management essential [5,6].

The etiology of traumatic ulcers is multifactorial, with local mechanical factors playing a predominant role. Common sources of injury include sharp tooth edges, fractured restorations, ill-fitting dentures, and orthodontic appliances [2,6]. Parafunctional habits such as bruxism or involuntary tongue biting can exacerbate the mechanical stress on the lateral tongue, especially during periods of psychological stress or sleep disturbances [4]. Systemic factors may also contribute by altering tissue resilience and delaying healing. For instance, conditions such as

diabetes, immunosuppression, and nutritional deficiencies have been linked to increased susceptibility to mucosal breakdown [7,8]. In rare cases, a traumatic ulcer may represent the initial manifestation of an underlying systemic disease, such as tuberculosis, syphilis, or HIV-related immunodeficiency, necessitating thorough clinical evaluation [1,9,10].

From a pathophysiological perspective, repetitive trauma initiates an inflammatory cascade characterized by disruption of the epithelial layer, vascular injury, and recruitment of inflammatory cells, which leads to localized necrosis and ulcer formation [6]. The lateral border of the tongue, composed of thin, highly vascularized mucosa, responds to trauma with heightened inflammatory activity, which explains the pain and sensitivity commonly reported by patients [4,6]. Persistent trauma interferes with the natural healing process, resulting in chronic ulcerations with indurated margins that may clinically resemble premalignant or malignant lesions [8]. This similarity poses a diagnostic challenge for clinicians, as chronic traumatic ulcers can mimic the appearance of oral squamous cell carcinoma, particularly in older patients or those with significant risk factors such as tobacco and alcohol use [5,6,8].

Clinically, traumatic ulcers are usually well-defined, shallow or deep lesions with a yellowish fibrinous center surrounded by an erythematous halo. The lesion is typically painful, especially when subjected to further irritation by speech, mastication, or acidic foods [4,6]. Acute ulcers generally heal within 10 to 14 days once the source of trauma is eliminated, while chronic ulcers may persist for weeks or even months [3,4]. In such cases, the persistence of the lesion raises concerns about other etiologies, including infections or neoplasia, making biopsy and histopathological analysis necessary for definitive diagnosis [6,8]. For example, tuberculosis can manifest as a persistent oral ulcer with a non-healing pattern, often accompanied by systemic symptoms such as cough, fever, and weight loss [1]. Similarly, syphilis may present with painless ulcers in its primary stage, whereas secondary syphilis is associated with multiple mucosal erosions [10]. HIV-positive patients are also at increased risk of atypical oral ulcerations due to immune dysregulation and opportunistic infections [9]. Therefore, clinicians must maintain a broad differential diagnosis when evaluating ulcers that fail to resolve with conservative measures.

The global prevalence of traumatic oral ulcers varies depending on population characteristics, with studies reporting higher rates in elderly individuals and in those wearing removable prosthetic appliances [3,12]. The lateral tongue border remains a primary site of involvement due to its functional role and frequent contact with dental structures. Importantly, chronic irritation has been investigated for its potential association with oral carcinogenesis. Although recent systematic reviews suggest limited evidence directly linking chronic trauma to the initiation of oral squamous cell carcinoma, persistent mechanical irritation may act as a cofactor that exacerbates pre-existing mucosal changes in susceptible individuals [8]. This underlines the importance of early recognition and removal of traumatic stimuli to prevent progression to more severe pathologies.

Advances in diagnostic methods have enhanced the clinician's ability to differentiate traumatic ulcers from other causes of mucosal ulceration. Conventional clinical examination remains the cornerstone of diagnosis, but adjunctive tools such as cytological analysis, salivary biomarkers, and molecular techniques have been increasingly explored [7]. Saliva, in particular, has emerged as a promising non-invasive diagnostic medium for detecting early malignant or infectious processes associated with oral ulcerations [7]. Histopathological evaluation remains indispensable in cases of persistent or atypical ulcers, providing definitive information regarding the nature of the lesion and ruling out malignancy or specific infections [6,8,9].

Management of traumatic ulcers focuses on identifying and eliminating the causative factor, which typically results in rapid healing. Adjunctive therapies, such as topical corticosteroids, antiseptic rinses, and agents promoting mucosal repair, can be employed to reduce inflammation and accelerate epithelial regeneration [2,3,4]. Natural substances, including plant-derived compounds, have been investigated for their healing potential, showing promising preliminary results [4]. For ulcers secondary to systemic diseases like tuberculosis or syphilis,

treatment must target the underlying condition, emphasizing the necessity of interdisciplinary collaboration between dentists, physicians, and infectious disease specialists [1,9,10].

Etiology and pathogenesis

The etiology of traumatic ulcers on the lateral border of the tongue is multifactorial, with local mechanical trauma playing the most significant role. The tongue, due to its constant mobility and proximity to teeth, is particularly susceptible to repetitive irritation. The most common local causes include sharp tooth edges, fractured restorations, improperly contoured crowns, ill-fitting dentures, and orthodontic appliances, which can continuously rub against the delicate mucosal lining of the tongue (figure 1) [11-14]. This repeated mechanical stress disrupts the integrity of the epithelial barrier, leading to localized tissue breakdown and ulcer formation. In addition, parafunctional habits such as bruxism, tongue thrusting, or involuntary tongue biting during sleep further exacerbate trauma, especially in individuals with high levels of psychological stress or neuromuscular disorders [12,13].

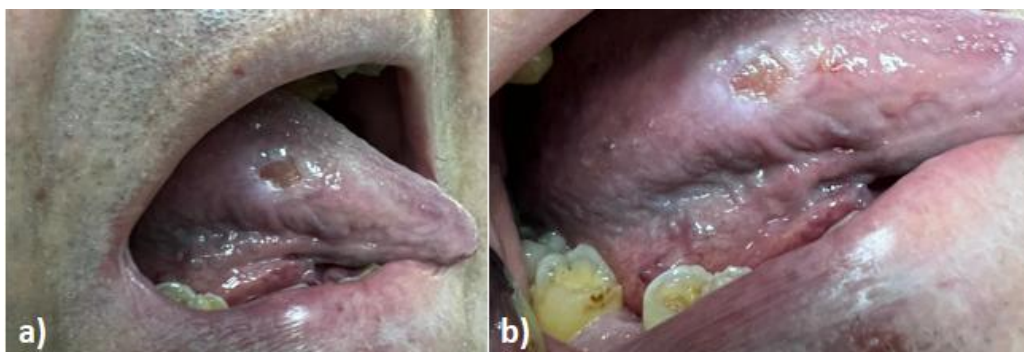


Figure 1. Traumatic ulcer on the lateral border of the tongue (a) Superficial ulcer with irregular margins and a fibrin-covered base located on the lateral border of the tongue. (b) Chronic ulcer with thickened margins and adjacent erythematous areas, associated with mechanical irritation from sharp tooth edges and fractured restorations, indicating the persistence of the traumatic factor.

The pathogenesis of traumatic ulceration begins with mechanical disruption of the epithelium, followed by an inflammatory cascade. The initial injury causes vascular damage and the release of pro-inflammatory mediators, attracting neutrophils and macrophages to the site. This leads to tissue necrosis, manifested clinically as a yellowish fibrinous base surrounded by erythema [6,14]. When the traumatic factor is removed, the healing process begins rapidly, with re-epithelialization typically occurring within 10–14 days. However, when the trauma persists, chronic inflammation develops, with continuous recruitment of inflammatory cells and fibroblasts, resulting in the formation of indurated margins and granulation tissue [6,15]. These chronic lesions are clinically significant because they may mimic oral squamous cell carcinoma, particularly in older individuals or those with a history of tobacco and alcohol use [8,15].

Systemic factors can significantly influence both the occurrence and persistence of traumatic ulcers. Conditions such as diabetes mellitus, hematological disorders, and nutritional deficiencies impair tissue repair and increase susceptibility to mucosal injury [7,13]. Immunocompromised patients, including those with HIV infection, are at higher risk of developing atypical ulcers with prolonged healing times, as demonstrated by Delgado et al., who identified unique histopathological patterns in oral ulcers among HIV-positive individuals [9]. Furthermore, certain infectious diseases can either mimic traumatic ulcers or predispose patients to trauma-induced ulceration. For instance, syphilis may initially present with solitary, painless oral ulcers, while secondary stages manifest as multiple mucosal erosions, complicating the diagnostic process [10]. Similarly, tuberculosis can present with cavitating oral ulcers as an initial

symptom, emphasizing the importance of a thorough systemic evaluation in non-healing lesions [1].

A study by Zhou et al. [10] highlighted the complexity of distinguishing between traumatic and infectious ulcers, demonstrating that many syphilitic lesions were initially misdiagnosed as trauma-related due to overlapping clinical features. This diagnostic challenge underscores the importance of considering systemic diseases when evaluating ulcers on the lateral border of the tongue, especially when these ulcers persist despite the removal of obvious mechanical irritants [10,13]. Minhas et al. [13] further emphasized the association between systemic conditions and oral ulcerations, noting that autoimmune diseases, gastrointestinal disorders, and hematinic deficiencies may all contribute to mucosal fragility and delayed healing.

Chronic trauma has also been investigated for its potential role in oral carcinogenesis. While mechanical irritation alone is not considered a direct carcinogenic factor, it may act as a promoter by exacerbating epithelial dysplasia caused by other carcinogens, such as tobacco or alcohol [8]. Pantenero et al. [8] concluded that although there is low evidence to establish a causal relationship, persistent trauma should not be overlooked, especially in high-risk individuals. Nelonda and Setiadhi [15] reported a case in which a chronic traumatic ulcer mimicked oral squamous cell carcinoma both clinically and histologically, demonstrating the need for biopsy in lesions that fail to heal within two to three weeks.

The pathogenesis of chronic traumatic ulcers is characterized by the persistent release of inflammatory cytokines and growth factors, which interfere with normal tissue regeneration. Prolonged inflammation can lead to stromal changes, including fibroblast proliferation and collagen deposition, creating a firm, indurated base. In some cases, eosinophilic infiltration is observed, as in traumatic ulcerative granuloma, a rare but important differential diagnosis [20]. This inflammatory milieu creates an environment that not only delays healing but also increases the risk of secondary infection. Bacterial colonization of the ulcer surface further impairs epithelialization and contributes to pain and discomfort [6,14].

Environmental factors and lifestyle habits play an additional role. Poor oral hygiene and the use of tobacco products increase mucosal vulnerability to trauma and delay healing by impairing blood flow and immune response [14,15]. Chen et al. [12] observed that trauma-associated lesions were significantly more common among individuals with poor oral hygiene and those wearing removable dental prostheses, reinforcing the importance of preventive strategies. Moreover, acidic or spicy diets can aggravate existing ulcerations by chemically irritating the exposed nerve endings and inflamed tissue [4].

In summary, the etiology of traumatic ulcers on the lateral tongue border involves a complex interplay of local mechanical factors, systemic health conditions, and environmental influences. The pathogenesis follows a continuum from acute epithelial disruption and inflammation to chronic ulceration with histological changes that may resemble malignancy. A comprehensive understanding of these mechanisms is critical for clinicians to accurately identify the underlying cause, eliminate contributing factors, and initiate appropriate treatment strategies. Persistent or atypical ulcers warrant careful investigation, including biopsy and laboratory testing, to rule out systemic disease or neoplastic transformation [10,13–15].

Clinical features

Traumatic ulcers on the lateral border of the tongue exhibit characteristic clinical patterns that reflect both the acute and chronic stages of the lesion. Typically, they present as well-defined ulcers with a yellowish fibrinous base surrounded by an erythematous halo, often accompanied by moderate to severe pain, especially during speech, mastication, or contact with acidic foods [6,12,14]. The lateral border of the tongue is particularly vulnerable due to its constant mobility and frequent contact with sharp tooth edges, fractured restorations, or prosthetic appliances [12,15]. Acute ulcers are usually solitary, shallow, and irregularly shaped, with soft

margins that are non-indurated, and they tend to resolve within 10–14 days once the source of trauma is eliminated [14].

When the traumatic factor persists, the lesion may progress to a chronic ulcer, characterized by thickened, rolled, and indurated margins with a deeper ulcer base covered by yellow-gray necrotic tissue [15-19]. These chronic presentations often raise concern for malignancy, as they can mimic oral squamous cell carcinoma both clinically and histologically [15]. Nelonda and Setiadhi [15] described a case in which a long-standing traumatic ulcer on the tongue was initially suspected to be malignant, highlighting the importance of biopsy for any ulcer persisting beyond two to three weeks. Chronic traumatic ulcers are frequently associated with regional lymphadenopathy, further complicating the clinical distinction from neoplastic lesions [6,15].

Compilato et al. [19] proposed the S-C-D classification system for chronic oral ulcers, dividing them into:

- Specific ulcers (S), linked to identifiable systemic diseases or infections,
- Chronic ulcers (C), including those caused by persistent trauma, and
- Defective healing ulcers (D), associated with systemic or local factors that delay repair.

This system underscores the clinical heterogeneity of ulcerative lesions and aids in differential diagnosis.

In some cases, traumatic ulcers may manifest with granulomatous features, such as traumatic ulcerative granuloma with stromal eosinophilia, which presents as a persistent, firm ulcer with elevated margins and intense inflammatory infiltration [20]. These lesions are rare but clinically relevant due to their resemblance to malignant tumors.

Overall, the clinical features of traumatic ulcers range from transient, self-limiting lesions to persistent, indurated ulcers that require histopathological evaluation. The key indicators suggesting a traumatic etiology are the presence of a local irritant, lesion location on commonly traumatized areas like the lateral tongue, and rapid healing after removal of the causative factor [12,14,15]. Conversely, non-healing ulcers, especially those with firm margins and progressive enlargement, must prompt further investigation to exclude infectious, autoimmune, or malignant conditions [10,13,15]. Recognizing these patterns is essential for timely and accurate diagnosis, preventing unnecessary morbidity, and ensuring optimal patient outcomes.

Diagnosis and differential diagnosis

The diagnosis of a traumatic ulcer on the lateral border of the tongue begins with a thorough patient history and clinical examination to identify potential sources of local trauma, such as sharp teeth, fractured restorations, or ill-fitting dentures [12,14]. An essential diagnostic criterion is the resolution of the lesion within 10–14 days after eliminating the irritant factor. If the ulcer persists beyond this period, further investigation is warranted [14,15].

The differential diagnosis includes a wide spectrum of conditions. Infectious causes must be considered: syphilis, which may present with solitary painless ulcers or multiple mucosal erosions [10]; tuberculosis, where oral ulcers may precede pulmonary symptoms [1]; and HIV-associated lesions, which are often atypical and slow-healing [9]. Autoimmune and systemic diseases, such as pemphigus vulgaris or hematinic deficiencies, can also manifest as recurrent or persistent ulcers [13].

Malignancies, particularly oral squamous cell carcinoma, represent the most critical differential consideration due to their similar appearance to chronic traumatic ulcers [8,15]. Nelonda and Setiadhi [15] emphasized the importance of biopsy in ulcers with indurated margins, progressive growth, or regional lymphadenopathy.

Mortazavi et al. [14] proposed a decision tree approach: initial assessment focuses on ulcer duration and presence of local trauma, followed by evaluation for systemic symptoms or

risk factors. When trauma is excluded or the lesion remains unresolved, histopathological examination becomes mandatory to confirm diagnosis and guide management [14,15,20].

Management and prognosis

The management of traumatic ulcers on the lateral border of the tongue primarily focuses on identifying and eliminating the causative factor, which usually results in rapid healing. Local irritants such as sharp teeth, fractured restorations, defective prostheses, or orthodontic appliances should be corrected or removed [12,15]. Once the traumatic source is eliminated, most ulcers heal spontaneously within 10–14 days, without the need for complex interventions [14]. Regular follow-up is essential to monitor lesion progression and ensure complete resolution. Persistent ulcers beyond three weeks require further evaluation, including biopsy, to rule out malignancy or underlying systemic disease [6,15].

Symptomatic treatment plays a vital role in reducing pain and inflammation during the healing process. Topical corticosteroids are considered the gold standard for reducing local inflammation and accelerating epithelial regeneration [2]. These agents, available as gels, ointments, or mouth rinses, must be applied carefully to avoid systemic absorption, particularly in immunocompromised patients. In addition to corticosteroids, antiseptic mouth rinses, such as chlorhexidine, can prevent secondary infections and promote mucosal healing [14].

Emerging evidence supports the use of hyaluronic acid-based formulations to enhance mucosal repair. Casale et al. [3] demonstrated that hyaluronic acid promotes tissue hydration, angiogenesis, and epithelial migration, offering an adjunctive benefit in traumatic ulcer management. Similarly, herbal products and natural compounds have been investigated for their wound-healing potential. Gani et al. [4] reported positive outcomes using *Jatropha multifida* sap in accelerating ulcer resolution, suggesting a promising direction for alternative therapies, especially in patients seeking natural treatment options.

For chronic or non-healing ulcers, advanced therapeutic techniques may be considered. CO₂ laser therapy has shown significant efficacy in promoting rapid wound healing by stimulating the expression of heat shock protein-70 and tenascin C, both of which are critical for tissue regeneration [11]. This minimally invasive approach not only accelerates healing but also provides superior patient comfort by reducing pain and edema. Laser therapy is particularly useful in recurrent cases or in patients with systemic conditions that delay mucosal repair [11].

When an ulcer is associated with an infectious or systemic cause, management must target the underlying condition. Syphilitic ulcers require systemic antibiotic therapy, typically with penicillin, in accordance with established treatment guidelines [10]. Tuberculosis-associated oral ulcers necessitate referral to a medical specialist for comprehensive anti-tubercular therapy [1]. In HIV-positive individuals, antiretroviral therapy plays a crucial role in reducing the incidence and severity of oral ulcerations [9]. This highlights the importance of a multidisciplinary approach, involving dentists, physicians, and infectious disease specialists, to ensure comprehensive patient care.

Patient education and prevention are critical components of long-term management. Individuals should be informed about maintaining optimal oral hygiene, undergoing regular dental check-ups, and avoiding habits such as cheek or tongue biting, as well as minimizing exposure to chemical irritants like tobacco and alcohol [12,14]. Dietary modifications, including the avoidance of acidic and spicy foods, can also reduce discomfort and promote healing [4]. For denture wearers, regular adjustments and relining procedures help prevent chronic mucosal irritation.

The prognosis for traumatic ulcers is generally excellent when the causative factor is identified early and appropriately managed. Most lesions heal completely without recurrence once the trauma is eliminated [12,14]. However, chronic ulcers that persist despite intervention pose a greater diagnostic and therapeutic challenge. Persistent irritation has been suggested as a

potential cofactor in oral carcinogenesis, particularly in individuals at high risk [8]. Therefore, non-healing ulcers must always be treated with suspicion and closely monitored to prevent a late diagnosis of oral squamous cell carcinoma. Nelonda and Setiadhi [15] emphasized that timely biopsy and histopathological examination are essential in such cases to distinguish between benign chronic trauma and malignant transformation.

Conclusions

Traumatic ulcers of the lateral border of the tongue are among the most common oral mucosal lesions, primarily caused by repetitive mechanical irritation from sharp teeth, fractured restorations, or poorly fitting dental appliances.

Early identification and prompt elimination of the traumatic factor are essential to promote rapid mucosal healing and to prevent the transition of an acute ulcer into a chronic, persistent lesion.

When an ulcer fails to heal within two to three weeks or presents atypical features, a biopsy and additional diagnostic tests are mandatory to rule out systemic diseases such as syphilis, tuberculosis, or HIV, as well as malignant conditions like oral squamous cell carcinoma.

A comprehensive, multidisciplinary approach that integrates preventive measures, modern therapeutic options such as CO₂ laser and hyaluronic acid, and thorough patient education provides the best outcomes, minimizes recurrence, and ensures long-term oral health and functional rehabilitation.

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