

Oral Ulcerative Lesions

Oral Ülsere Lezyonlar

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ABSTRACT

Oral ulcerative lesions have diverse aetiologies, including autoimmune diseases, infections, trauma and malignancies. This review explores the clinical features, diagnostic methods and therapeutic strategies associated with these conditions. The most prevalent type is recurrent aphthous ulcer, which is categorised as minor, major, or herpetiform. Management primarily aims to alleviate pain and prevent recurrence. Autoimmune disorders such as erythema multiforme, oral lichen planus, pemphigus vulgaris and mucous membrane pemphigoid are often associated with chronic, recurrent oral lesions. Diagnosis is based on clinical evaluation and histopathological analysis, with treatment mainly relying on corticosteroids and immunomodulatory agents. Infectious causes, including viral, bacterial and fungal pathogens, also play a significant role in the development of oral ulcerations. Herpes simplex virus, *Treponema pallidum* and *Candida* species are among the most common pathogens. Trauma-induced lesions, whether physical, chemical or mechanical, are particularly prevalent among vulnerable populations. Oral squamous cell carcinoma poses a unique challenge due to its malignant nature and potential for metastasis. Accurate diagnosis usually necessitates a biopsy and advanced molecular techniques. Treatment should be etiology-specific, and complex cases often require a multimodal approach. This review emphasises the importance of early diagnosis, personalised therapeutic planning and regular follow-up to minimise morbidity and optimise patient outcomes.

Keywords: Oral ulcer, Autoimmunity-related Ulcerations, Traumatic oral ulcer, Infection related ulcers, Oral squamous cell carcinoma.

ÖZ

Ağız ülseratif lezyonları; otoimmün hastalıklar, enfeksiyonlar, travma ve maligniteler gibi çeşitli etiyolojilere sahiptir. Bu derleme, söz konusu lezyonların klinik özelliklerini, tanı yöntemlerini ve tedavi yaklaşımlarını ele almaktadır. Tekrarlayan aftöz ülseler en yaygın tür olup; minör, majör ve herpetiform olmak üzere sınıflandırılır. Tedavileri, ağrıyı hafifletmeye ve tekrarları önlemeye odaklanır. Eritema multiforme, oral liken planus, pemfigus vulgaris ve mukoz membran pemfigoidi gibi otoimmün hastalıklarla ilişkili durumlar, genellikle tekrarlayan ağız lezyonlarıyla karakterizedir. Bu hastalıkların tanısı, klinik değerlendirme ve histopatolojik analizlere dayanır; tedavide ise kortikosteroidler ve immünomodülatör ajanlar kullanılır. Viral, bakteriyel ve fungal patojenler de önemli katkıda bulunur. Herpes simplex virüsü, *Treponema pallidum* ve *Candida* türleri en sık görülen etkenlerdir. Fiziksel, kimyasal veya mekanik travmaya bağlı lezyonlar, özellikle savunmasız bireylerde yaygındır. Oral skuamöz hücreli karsinom ise, malign doğası ve metastaz riski nedeniyle özel bir öneme sahiptir. Doğru tanı için biyopsi ve moleküler teknikler gereklidir. Tedavi, etiyolojiye özgüdür ve karmaşık vakalarda genellikle çoklu yaklaşımlar uygulanır. Bu derleme, morbiditeyi en aza indirmek ve hasta sonuçlarını optimize etmek için erken tanının, bireyselleştirilmiş terapötik planlamanın ve düzenli takibin önemini vurgulamaktadır.

Anahtar Kelimeler: Oral ülsere, Otoimmüniteyle ilişkili ülsereasyonlar, Travmatik oral ülsere, Enfeksiyon kaynaklı ülseler, Oral skuamöz hücreli karsinom

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Ulcerations Associated with Autoimmunity:

The primary characteristic of oral ulcers with an immunological etiology is their recurrent nature.¹

Recurrent Aphthous Ulcers: Recurrent aphthous ulcers are the most common lesions observed in the oral mucosa, typically presenting as painful, round or ovoid ulcers surrounded by erythematous haloes. The ulcer bases appear yellow or gray in color and vary in size. recurrent aphthous ulcers are classified into three main types: minor, major, and herpetiform ulcers.²

Minor Aphthous Ulcers: This is the most prevalent form of RAS, affecting approximately 80% of patients. The ulcers are typically small, measuring less than 5 mm in diameter, and are characterized by a round or oval shape with a gray-white pseudomembrane surrounded by an erythematous halo. (Fig.1) They commonly appear on non-keratinized surfaces such as the labial and buccal mucosa, but are less frequent on the gingiva or hard palate. Minor ulcers heal without scarring within 10–14 days.^{2,3}



Figure 1. Minor Aphthous Ulcers (*Image adapted from Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Major Aphthous Ulcers: This is a more severe and less common form, affecting around 10% of people with RAS. The ulcers are deep and oval-shaped, and can exceed 1 cm in diameter (Fig. 2). They have a predilection for the lips, soft palate and fauces (see Fig. 2). Major ulcers may persist for up to six weeks and often result in scarring upon healing.^{3,4}

Herpetiform Ulcers: This is the least common type of aphthous ulceration. It is characterised by multiple crops of small ulcers, each measuring 1–3 mm.⁴ These lesions may coalesce to form larger, irregularly shaped ulcers. Despite their name, they are not associated with herpes virus infections. Herpetiform ulcers are more prevalent in women and typically present at a later age compared to other forms.^{2,3,5}

Etiological factors can be categorised as either local or systemic. Local factors include trauma, while systemic

causes include Behçet's disease, MAGIC syndrome, HIV, nutritional deficiencies, gastrointestinal disorders and cyclic neutropenia.³ Treatment focuses on alleviating pain, promoting ulcer healing and preventing recurrence.⁵ Tetracyclines such as doxycycline and minocycline have been shown to suppress collagenases and matrix metalloproteinases, which aids the management of recurrent aphthous ulcers. Topical corticosteroids are the primary treatment and are believed to work by inhibiting interactions between T-lymphocytes and epithelial cells. However, systemic administration has been found to be more effective in reducing recurrence rates. Topical anaesthetics such as benzocaine and lidocaine are effective in controlling pain and reducing inflammation. Additional topical agents include NSAIDs, antiseptics, and hyaluronic acid. Systemic treatment modalities may include immunomodulatory agents, antibiotics/antimicrobials, and corticosteroids. Furthermore, the literature has documented laser therapy as an adjunctive treatment option.⁴



Figure 2. Major Aphthous Ulcers (*Image adapted from Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Erythema Multiforme and Drug-Induced Ulcerations:

Erythema multiforme is a condition characterised by the formation of lesions that are usually self-limiting and isolated. These lesions may affect only the skin or involve mucocutaneous regions. Lesions typically present as acute, symmetrical skin eruptions, which can spread to the oral mucosa. These lesions are characterised by their round morphology, known as 'target' or 'iris' lesions. These consist of three concentric segments: a dark centre surrounded by a lighter pink ring, which is in turn enclosed by a red ring (Fig. 4). (Fig. 4). Atypical lesions may have less defined borders and consist of only two zones of colour. Lesions typically begin as pink or red papules, evolving into plaques and causing a burning or itching sensation. They typically appear symmetrically on the extensor surfaces of the extremities, spreading centrifugally towards the trunk, although they tend to be less numerous on the torso. Mucosal lesions may start as

oedematous, erythematous lesions that can progress to shallow erosions with pseudomembranes.⁶⁻⁸ It can be triggered by viral infections, specifically the herpes simplex virus, and certain medications. Erythema multiforme may be recurrent and persistent.^{7,8} Around 90% of cases are infection-related, primarily caused by the herpes simplex virus, followed by *Mycoplasma pneumoniae*. Medications are implicated in less than 10% of cases. The most commonly encountered medications linked to this condition include non-steroidal anti-inflammatory drugs, antiepileptics and antibiotics. Differential diagnoses include pityriasis rosea, urticaria, viral exanthems, fixed drug eruptions, bullous pemphigoid, Stevens–Johnson syndrome, polymorphic light eruption, paraneoplastic pemphigus and hypersensitivity reactions.⁸ A crucial aspect of treatment is identifying and eliminating the triggering factor. In cases involving drugs, it is essential to identify and discontinue the triggering medication. For cases associated with the herpes simplex virus, the administration of antiviral agents is indicated. For patients with minimal mucosal involvement, topical corticosteroids, antiseptics and anaesthetics may be effective and sufficient. Although rare, there have been reports of an association between erythema multiforme and malignancy, so a comprehensive clinical assessment may be necessary for patients presenting with recurrent, idiopathic erythema multiforme ulcers.⁶

Lichenoid Lesions:

Oral lichen planus (OLP) is a chronic, recurrent autoimmune disease affecting the oral mucosa.⁹ The clinical presentation of OLP can vary significantly and include six distinct subtypes: reticular, plaque-like, atrophic, erosive/ulcerative, papular and bullous. The erosive/ulcerative form of OLP is particularly relevant to discussions about ulcerative lesions, as it is characterised by erythema and ulceration. These painful lesions may cause significant discomfort and interfere with daily activities such as eating and speaking, and lesions on the tongue and red clinical forms are considered risk factors for the development of oral squamous cell carcinoma.^{9,10} Oral lichen planus (OLP) affects approximately 5% of the general population, predominantly women (with a ratio of 2:1), with peak incidence in middle age.¹¹ It primarily affects the buccal mucosa and may present either asymptotically or with discomfort, burning sensations or pain that impairs eating.^{9,11} Although it is a benign lesion, it has been reported in the literature that 1.4% of lesions transform into a malignant form within seven years.¹⁰ The diagnosis of OLP is typically based on a combination of clinical and histopathological criteria. While a clinical diagnosis may be sufficient for classic, bilateral and symmetrical reticular lesions, an oral biopsy is generally recommended to confirm the diagnosis and, more importantly, to rule out dysplasia or malignancy. Histopathological analysis usually shows

liquefactive degeneration of basal cells and a band-like lymphocytic infiltrate in the superficial connective tissue. The absence of epithelial dysplasia is a key criterion for diagnosing OLP. In difficult cases, particularly those affecting the gingiva, direct immunofluorescence (DIF) of the surrounding mucosa can be used to rule out other conditions, such as vesiculobullous diseases. Additionally, patch testing can help to identify the allergens responsible for lichenoid reactions linked to dental materials.¹²⁻¹³ The aetiology includes genetic history, psychological state, trauma, systemic diseases, graft-versus-host disease, dental materials, hepatitis C infection, hypertension, diabetes, hypothyroidism and systemic medications such as antihypertensives and non-steroidal anti-inflammatory drugs.¹² Asymptomatic lesions do not require treatment and annual monitoring is considered adequate. For symptomatic lesions, topical corticosteroids are used as part of the therapeutic approach. Maintaining optimal oral hygiene is essential, and any rough or sharp-edged dental restorations should be corrected if present. It is also important to avoid foods that may contribute to irritation.¹³

Vesiculobullous Lesions:

These lesions are typically characterised by the transient yet recurrent formation of vesicles and bullae, which can be observed on the oral mucosa as well as on other mucosal sites within the body. They may occur alongside various autoimmune disorders, particularly pemphigus vulgaris and mucous membrane pemphigoid¹⁴. Pemphigus vulgaris is a rare but severe mucocutaneous vesiculobullous disorder. It affects both the skin and the oral mucosa, with approximately 80% of cases initially presenting with oral lesions before skin involvement (Fig.5) This condition means that dental professionals are often the first to identify lesions of this disorder, presenting an opportunity for early diagnosis and clinical recognition.¹⁵ Mucous membrane pemphigoid (MMP) is a group of autoimmune disorders characterised by subepithelial/subepidermal vesicle formation. It primarily affects the oral cavity and conjunctiva, followed by the nasopharynx and genital region. Less commonly, it can also affect the larynx, oesophagus and respiratory tract. The oral mucosa is often the initial site of appearance of the first signs of MMP, with gingival lesions being the most common oral symptom and being observed in approximately 80% of cases. Vesiculobullous lesions are associated with autoimmune diseases such as PV and MMP. Gingival lesions may manifest as 'desquamative gingivitis', whereby the outer layer of the gums sloughs off, leaving a raw and painful surface. Furthermore, the Nikolsky sign, which is characterised by the formation of a bulla when the skin is gently rubbed, can be observed in lesions on the gingiva caused by both pemphigus vulgaris and mucous membrane pemphigoid (Fig.4)



Figure 3. Oral pemphigus vulgaris (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

an 'intraepithelial split' can be observed within the surface cells, which can make the basal cell layer appear as if it is made up of 'tombstones'. By contrast, mucous membrane pemphigoid is characterised by a 'subepithelial split', whereby the entire epithelial surface separates from the underlying connective tissue layer. Direct immunofluorescence (DIF) reveals a 'fishnet pattern' on the cell surfaces in PV and a linear band of fluorescence along the basement membrane in MMP. Indirect immunofluorescence (IIF) is used to detect circulating autoantibodies, which is particularly helpful in defining prognosis and guiding treatment for PV. All intraoral vesicular disorders are characterised by an autoimmune pathophysiology. Using immunosuppressants with anti-inflammatory properties helps to mitigate pain and reduce the morbidity associated with these lesions by controlling inflammation. Although the causes may vary, the treatment protocols are usually similar and involve corticosteroids. These are available in multiple formulations, facilitating various methods of administration. These include topical ointments/gels, syrups, systemic medications and injectable solutions. It is important to note that topical creams designed for cutaneous use should not be applied to the inside of the mouth.^{15 14}



Figure 4. Eritema multiforme (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

A definitive diagnosis of these conditions relies on histopathological and immunofluorescence examinations of biopsy specimens. In the case of pemphigus vulgaris,

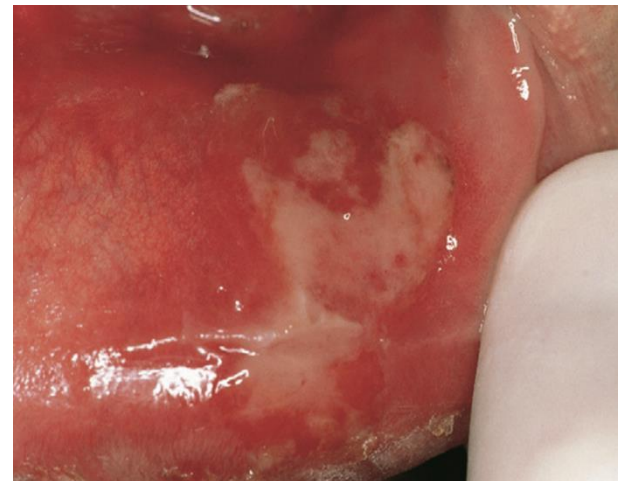


Figure 5. Pemphigus vulgaris lesion (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Trauma-related ulcerative lesions:

Traumatic lesions can occur as a result of physical, chemical, thermal or mechanical injury. Inappropriate orthodontic or prosthetic materials, malpositioned or fractured teeth with sharp edges, chronic cheek biting and the improper topical application of aspirin can contribute to the formation of traumatic ulcers¹⁶. (Fig.6) Trauma caused by the patient can be observed in children and individuals with mental health issues. Incorrect and aggressive tooth brushing can also lead to traumatic lesions. Biting the lips while under the influence of anaesthesia from a dental examination is also a common cause.¹⁷.(Table 1).

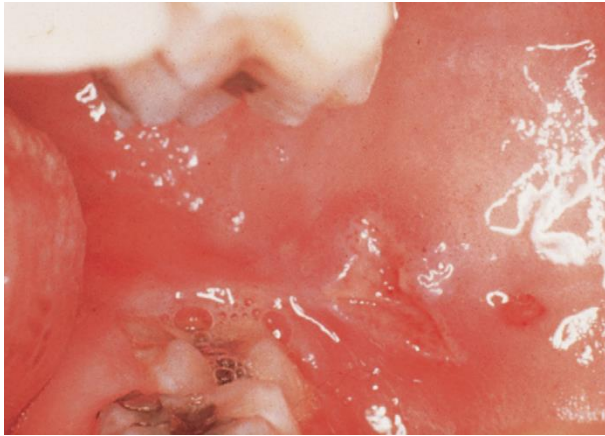


Figure 6. Acute traumatic ulcer (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

usually resolves spontaneously within seven to ten days.¹⁷



Figure 7. Herpes simplex (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Table 1. Ulcerative Lesions Caused by Trauma

Chronic	Acute
Small and painless	Painful
Most commonly observed on the tongue and may mimic carcinoma or infectious lesions	Yellow base with a surrounding red halo
Base may appear elevated and yellowish	History of trauma is usually present
Healing is delayed if irritation persists	Heals within 7–10 days if the causative factor is not repeated

Ulcers caused by infections:

Viral infections can be categorised, with herpes simplex virus (HSV-1 and HSV-2) being the most common pathogens¹⁸ Both HSV-1 and HSV-2 can cause ulcerative lesions, but HSV-1 is more frequently observed in the oral mucosa.¹ The term 'primary HSV-1 infection' refers to the initial encounter with the viral pathogen. This occurs due to a deficiency in antibodies and T lymphocytes. Often presenting with subclinical features, it can be difficult to recognise; the most prominent manifestation is herpes gingivostomatitis.¹⁹ The initial manifestation of herpes gingivostomatitis is hyperaemia of the perioral and oral mucosa (Fig.7) It typically progresses to rapidly disseminating vesicular lesions. These lesions may subsequently rupture and convert into an ulcerative form. They usually resolve within two weeks without leaving scarring.²⁰ Secondary HSV-1 infections lead to recurrent ulcers. Following the primary infection, latent HSV-1 can reactivate due to triggers such as colds, menstruation, trauma, fever and immunosuppression.¹⁹ It manifests as herpes labialis, presenting as painful, recurrent ulcerative lesions on the lips and perioral area. In the oral cavity, it is typically observed on keratinised mucosa and the hard palate. It

The varicella-zoster virus (VZV) is responsible for the primary infection, which manifests as varicella. Following a period of latency, the virus can reactivate under conditions of immunosuppression, leading to herpes zoster.²¹ Varicella primarily affects the skin; however, in severe cases, lesions can also be observed in the oral region.¹ (Fig.8)



Figure 8. Varicella zoster (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Cytomegalovirus (CMV) is a virus belonging to the Herpesviridae family.²² The infection is often asymptomatic. However, in immunocompromised individuals, manifestations such as hepatomegaly, splenomegaly, and central nervous system involvement may occur. Oral lesions are typically observed in HIV-positive patients when co-infected with HSV.²³ Valganciclovir and ganciclovir are first-line medications used in treatment.²⁴

Fungal Infections: The most prevalent fungal pathogen in the oral cavity is *Candida* species, which typically produce non-ulcerative lesions. In contrast, oral ulcerations may be caused by other fungal pathogens, such as *Cryptococcus neoformans*, *Histoplasma capsulatum*, *Aspergillus fumigatus*, *Absidia*, *Rhizopus*,

Rhizomucor and Mucor species. Oral ulcerations due to these fungi are strongly associated with underlying immunosuppression or immunodeficiency.²⁵

Blastomycosis can manifest as oral ulcerations, which may mimic squamous cell carcinoma. Definitive diagnosis relies on histopathological examination with the use of specific staining methods. The most commonly employed treatment is intravenous amphotericin B.¹ In the differential diagnosis of fungal infections, it is important to note that Actinomyces species exhibit characteristics resembling both bacteria and fungi, leading to their historical classification as transitional organisms. However, their core biological properties unequivocally classify Actinomyces as bacteria. Unlike pathogenic fungi, Actinomyces are obligate or facultative anaerobes.²⁶

Bacterial Infections: Syphilis is caused by *Treponema pallidum*, a spirochete bacterium with a twisted spiral shape. It is transmitted through unprotected sexual contact, congenital transmission and, less commonly, exposure to contaminated blood products²⁷. Oral manifestations can be observed in primary, secondary and tertiary syphilis. (Fig.9) (Fig10) (Fig11). These signs are typically the initial indicators of the infection.²⁸ The defining feature of primary syphilis is the chancre. It is typically an asymptomatic, hard ulcerative lesion.²⁸ The most frequent site of manifestation is the lips. The tongue, palate, and tonsils are less commonly involved. It may present with associated cervical lymphadenopathy. Diagnosis can be difficult as it typically regresses within 3-8 weeks.¹ Penicillin G is the first-line treatment for all stages of syphilis, and no resistance has been observed to date. Long-acting penicillin should be administered once or three times daily, or oral antibiotics should be given multiple times a day for a period of ten days. If the patient is HIV positive, treatment can be administered similarly to that of HIV-negative individuals.²⁷



Figure 9. Primary syphilis chancre
(Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)



Figure 10. Condyloma latum (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

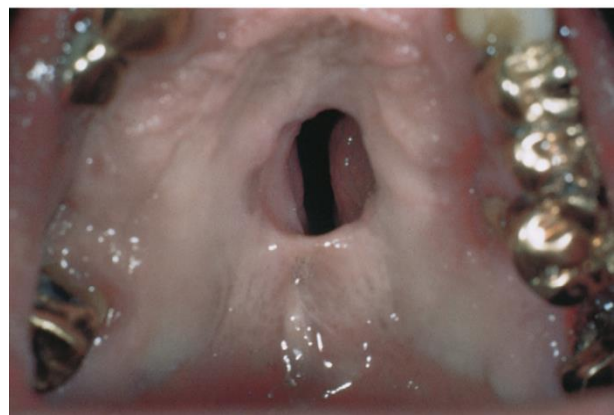


Figure 11. Palatal bone fistula caused by gumma due to tertiary syphilis (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Tuberculosis is a granulomatous disease caused by the bacterium *Mycobacterium tuberculosis*. While the pulmonary system is most commonly affected, other organ systems may also be involved. Primary tuberculosis lesions are usually painless, whereas secondary lesions may be painful. Both forms may be associated with lymphadenopathy. Primary tuberculosis lesions are commonly observed in paediatric patients, whereas secondary tuberculosis lesions are more frequently seen in adults. Ulcerations are generally singular, though multiple lesions have also been documented. These ulcers may be either deep or superficial, with an irregular, centrally necrotic appearance (Fig.12). They grow slowly and do not spontaneously resolve.¹ Oral lesions are most frequently found on the tongue and often manifest as painful ulcers. These lesions tend to resolve following antituberculous therapy.²⁹ There is no single diagnostic test; multiple techniques are employed to achieve rapid and accurate results. The results should always be correlated with the clinical presentation to ensure consistency. Molecular diagnostic methods such as microscopic examination of sputum, transcription amplification, ligase chain reaction

and strand displacement amplification are included among PCR-based diagnostic techniques.³⁰ In treatment, isoniazid, rifampicin, pyrazinamide and ethambutol are administered together for the first two months, followed by continued treatment with rifampicin and isoniazid for the next four months.³¹

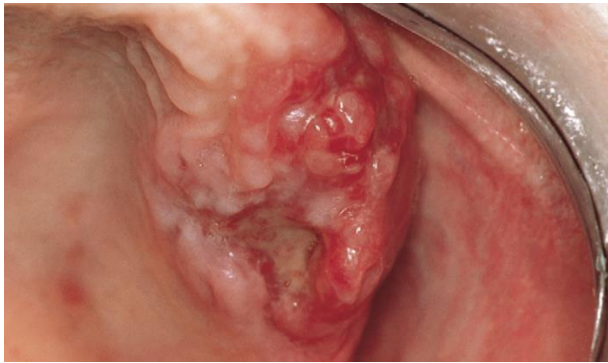


Figure 12. Tuberculosis lesion of the maxillary alveolar crest (Image adapted from *Oral Pathology: Clinical Pathological Correlations, 6th Edition.*)

Oral squamous cell carcinoma:

Oral squamous cell carcinoma (SCC) accounts for over 90% of all malignancies of the oral cavity and is the eighth most common cancer worldwide.³²

Oral malignant transformation typically progresses from normal epithelial cells to a thin, white lesion, and then to hyperplasia. This lesion frequently develops an erythematous component that can result in ulceration on the surface.¹ The posterior lateral border of the tongue is the most common site of occurrence, followed by the floor of the mouth, the soft palate, the gingiva, the buccal mucosa, and the hard palate.³³ Metastasis primarily occurs via lymphatic drainage to the ipsilateral cervical lymph nodes, though tumours can also metastasise to the contralateral or bilateral lymph nodes. Distant metastases most frequently involve the lungs, bones and liver. Treatment strategies depend on tumour staging, regional lymph node involvement and distant metastases. These may include wide local excision, neck dissection, radiotherapy, chemotherapy or, more commonly, a multimodal approach combining surgical intervention and adjunctive therapies.³³

Conclusion: Oral ulcerative lesions, which may result from various etiological factors, can typically be diagnosed through clinical evaluation and detailed patient anamnesis. However, in certain cases, a biopsy and subsequent histopathological examination may be necessary.

REFERENCES

1. Fitzpatrick SG, Cohen DM, Clark AN. Ulcerated Lesions of the Oral Mucosa: Clinical and Histologic Review. *Head Neck Pathol.* 2019;13(1):91-102. doi:10.1007/s12105-018-0981-8
2. Giannetti L, Murri dello Diago A, Lo Muzio L. Recurrent aphthous stomatitis. *Minerva Dental and Oral Science.* 2018;67(3). doi:10.23736/S0026-4970.18.04137-7
3. Jurge S, Kuffer R, Scully C, Porter SR. Number VI: Recurrent aphthous stomatitis. *Oral Dis.* 2006;12(1):1-21. doi:10.1111/j.1601-0825.2005.01143.x
4. Lau CB, Smith GP. Recurrent aphthous stomatitis: A comprehensive review and recommendations on therapeutic options. *Dermatol Ther.* 2022;35(6). doi:10.1111/DTH.15500
5. Plewa MC, Chatterjee K. Recurrent Aphthous Stomatitis. *European Handbook of Dermatological Treatments, Third Edition.* Published online November 13, 2023:67-71. doi:10.1007/978-3-662-45139-7_6
6. Sokumbi O, Wetter DA. Clinical features, diagnosis, and treatment of erythema multiforme: A review for the practicing dermatologist. *Int J Dermatol.* 2012;51(8):889-902. doi:10.1111/J.1365-4632.2011.05348.X
7. Soares A, Sokumbi O. Recent Updates in the Treatment of Erythema Multiforme. *Medicina (B Aires).* 2021;57(9):921. doi:10.3390/medicina57090921
8. Traves KP, Love G, Studdiford JS. Erythema Multiforme: Recognition and Management. *Am Fam Physician.* 2019;100(2):82-88.
9. González-Moles MÁ, Warnakulasuriya S, González-Ruiz I, et al. Worldwide prevalence of oral lichen planus: A systematic review and meta-analysis. *Oral Dis.* 2021;27(4):813-828. doi:10.1111/ODI.13323
10. Giuliani M, Troiano G, Cordaro M, et al. Rate of malignant transformation of oral lichen planus: A systematic review. *Oral Dis.* 2019;25(3):693-709. doi:10.1111/ODI.12885
11. Gupta S, Ghosh S. 4 Biopsy is reserved for noncharacteristic lesions Oral lichen planus Interventions for the management of oral lichen planus: a review of the conventional and novel therapies. *J Oral Pathol Med.* 2020;192:1029-1071. doi:10.1503/cmaj.200309/-/DC1
12. Alrashdan MS, Cirillo N, McCullough M. Oral lichen planus: a literature review and update. *Arch Dermatol Res.* 2016;308(8):539-551. doi:10.1007/s00403-016-1667-2

13. Hamour AF, Klieb H, Eskander A. Oral lichen planus. *CMAJ*. 2020;192(31):E892. doi:10.1503/CMAJ.200309/-/DC1
14. Beaty CS, Short AG, Mewar P. Oral Mucosal Lesions, Immunologic Diseases. *StatPearls*. Published online November 14, 2023. Accessed February 7, 2024. <https://www.ncbi.nlm.nih.gov/books/NBK597383/>
15. Hargitai IA. Painful Oral Lesions. *Dent Clin North Am*. 2018;62(4):597-609. doi:10.1016/j.cden.2018.06.002
16. Madani FM, Kuperstein AS. Normal Variations of Oral Anatomy and Common Oral Soft Tissue Lesions. *Medical Clinics of North America*. 2014;98(6):1281-1298. doi:10.1016/j.mcna.2014.08.004
17. Muñoz-Corcuera M, Esparza-Gómez G, González-Moles MA, Bascones-Martínez A. Oral ulcers: clinical aspects. A tool for dermatologists. Part I. Acute ulcers. *Clin Exp Dermatol*. 2009;34(3):289-294. doi:10.1111/j.1365-2230.2009.03220.x
18. Huang CW, Hsieh CH, Lin MR, Huang YC. Clinical features of gingivostomatitis due to primary infection of herpes simplex virus in children. doi:10.1186/s12879-020-05509-2
19. Westley S, Seymour R, Staines K. Recurrent intra-oral herpes simplex 1 infection. *Dent Update*. 2011;38(6). doi:10.12968/DENU.2011.38.6.368
20. Aslanova M, Ali R, Zito PM. Herpetic Gingivostomatitis. *StatPearls*. Published online June 12, 2023. Accessed February 12, 2024. <https://www.ncbi.nlm.nih.gov/books/NBK526068/>
21. Clarkson E, Mashkoo F, Abdulateef S. Oral Viral Infections: Diagnosis and Management. *Dent Clin North Am*. 2017;61(2):351-363. doi:10.1016/J.CDEN.2016.12.005
22. Santosh AR, Muddana K. Viral infections of oral cavity. *J Family Med Prim Care*. 2020;9(1):36. doi:10.4103/jfmpe.jfmpe_807_19
23. Clarkson E, Mashkoo F, Abdulateef S. Oral Viral Infections. *Dent Clin North Am*. 2017;61(2):351-363. doi:10.1016/j.cden.2016.12.005
24. Kotton CN. CMV: Prevention, Diagnosis and Therapy. *American Journal of Transplantation*. 2013;13:24-40. doi:10.1111/ajt.12006
25. Muñoz-Corcuera M, Esparza-Gómez G, González-Moles MA, Bascones-Martínez A. Oral ulcers: clinical aspects. A tool for dermatologists. Part II. Chronic ulcers. *Clin Exp Dermatol*. 2009;34(4):456-461. doi:10.1111/J.1365-2230.2009.03219.X
26. Sezer B, Akdeniz BG, Günbay S, Hilmioğlu-Polat S, Başdemir G. Actinomycosis osteomyelitis of the jaws: Report of four cases and a review of the literature. *J Dent Sci*. 2017;12(3):301. doi:10.1016/J.JDS.2013.02.031
27. Sadoghi B, Stary G, Wolf P. Syphilis. *JDDG - Journal of the German Society of Dermatology*. 2023;21(5):504-517. doi:10.1111/DDG.14999
28. Smith MH, Vargo RJ, Bilodeau EA, et al. Oral Manifestations of Syphilis: a Review of the Clinical and Histopathologic Characteristics of a Reemerging Entity with Report of 19 New Cases. *Head Neck Pathol*. 2021;15(3):787-795. doi:10.1007/s12105-020-01283-4
29. Barragán YA, Murillo Cerda F de J, Vera MH, et al. Diagnosis of disseminated tuberculosis from a lesion in the oral cavity. *IDCases*. 2022;30:e01618. doi:10.1016/J.IDCR.2022.E01618
30. Yepes JF, Sullivan J, Pinto A. Tuberculosis: Medical management update. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*. 2004;98(3):267-273. doi:10.1016/j.tripleo.2004.05.012
31. Jain P, Jain I. Oral Manifestations of Tuberculosis: Step towards Early Diagnosis. *J Clin Diagn Res*. 2014;8(12):ZE18. doi:10.7860/JCDR/2014/10080.5281
32. Merry R, Belfield L, McArdle P, McLennan A, Crean S, Foey A. Oral health and pathology: A macrophage account. *British Journal of Oral and Maxillofacial Surgery*. 2012;50(1):2-7. doi:10.1016/J.BJOMS.2010.10.020
33. Maymone MBC, Greer RO, Kesecker J, et al. Premalignant and malignant oral mucosal lesions: Clinical and pathological findings. *J Am Acad Dermatol*. 2019;81(1):59-71. doi:10.1016/J.JAAD.2018.09.060