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REVIEW ARTICLE

ORAL MANIFESTATIONS OF HIV/AIDS: CLINICAL PRESENTATION, DIAGNOSTIC FEATURES, AND DIFFERENTIAL DIAGNOSIS: A SCOPING REVIEW

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Abstract

Background: Human immunodeficiency virus (HIV) infection is associated with a broad spectrum of oral manifestations resulting from progressive immune dysfunction, opportunistic infections, inflammatory processes, and neoplastic complications. Although modern antiretroviral therapy (ART) has substantially reduced the frequency and severity of several HIV-associated oral conditions, oral lesions remain clinically important indicators of local and systemic disease.
Objective: This review aims to describe the principal oral manifestations associated with HIV/AIDS, discuss their clinical and diagnostic characteristics, evaluate their relationship with immune status and ART, and summarize relevant differential diagnostic and management considerations.
Materials and Methods: A narrative review approach was used to examine published literature on HIV-associated oral diseases, including clinical reviews, classification studies, epidemiological investigations, and relevant clinical guidelines. Particular attention was given to oral candidiasis, oral hairy leukoplakia, Kaposi sarcoma, periodontal diseases, ulcerative lesions, salivary gland disorders, and other potentially significant oral conditions.
Results: The principal oral manifestations include pseudomembranous and erythematous candidiasis, oral hairy leukoplakia, Kaposi sarcoma, linear gingival erythema, necrotizing ulcerative gingivitis and periodontitis, persistent ulcerative lesions, salivary gland enlargement, and xerostomia. Their occurrence and clinical severity may be influenced by CD4-positive T-cell depletion, viral replication, ART exposure, oral hygiene, and additional systemic or local risk factors. No single oral lesion is diagnostic of HIV infection; clinical findings must be interpreted in conjunction with medical history, laboratory investigations, and appropriate confirmatory procedures.
Conclusion: Systematic oral examination remains an important component of comprehensive HIV care. Early recognition, accurate differential diagnosis, appropriate treatment, and multidisciplinary collaboration may improve oral health, facilitate timely referral, and contribute to better quality of life in people living with HIV.

Keywords: HIV; AIDS; oral manifestations; oral candidiasis; oral hairy leukoplakia; Kaposi sarcoma; periodontal disease; xerostomia; differential diagnosis.

INTRODUCTION

Human immunodeficiency virus (HIV) infection remains a major global public health concern because of its

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progressive effects on the immune system and its broad range of clinical manifestations. Although the introduction of combination antiretroviral therapy (ART) has substantially reduced HIV-associated morbidity and mortality, oral diseases continue to represent an important clinical component of HIV infection. The oral cavity is particularly relevant because several infectious, inflammatory, neoplastic, and immune-mediated conditions may develop during the course of HIV-associated immunosuppression. Some of these lesions may appear early, precede the recognition of systemic disease, or indicate inadequate viral suppression and declining immune function[1–5].

HIV primarily targets CD4-positive T lymphocytes, leading to progressive impairment of cell-mediated immunity. The reduction in CD4-positive T-cell numbers and dysfunction of the immune response facilitate the development of opportunistic infections and other pathological conditions. In the oral cavity, this immunological impairment may manifest as candidiasis, oral hairy leukoplakia, periodontal disease, recurrent or persistent ulceration, salivary gland disorders, and HIV-associated malignancies. The clinical expression of these conditions is influenced by the degree of immunosuppression, plasma viral load, duration of infection, individual susceptibility, oral hygiene, smoking, nutritional status, and access to antiretroviral treatment [2–5].

The relationship between HIV infection and oral pathology has been extensively investigated. The international classification developed by the EC-Clearinghouse and the World Health Organization established standardized diagnostic categories for oral lesions associated with HIV infection. This classification distinguished lesions strongly associated with HIV infection from those less consistently related to the disease, providing a useful framework for clinical examination, epidemiological research, and diagnostic evaluation[1]. Subsequent clinical reviews have confirmed the importance of oral candidiasis, oral hairy leukoplakia, Kaposi sarcoma, and characteristic periodontal diseases as clinically relevant manifestations of HIV-associated immune dysfunction [2,3,7].

Oral candidiasis is among the most frequently reported oral manifestations of HIV infection. It may present in pseudomembranous, erythematous, or hyperplastic forms and may also involve the angles of the mouth. Pseudomembranous candidiasis is characterized by removable white plaques, whereas erythematous candidiasis typically presents as red, atrophic areas of the oral mucosa or tongue. The occurrence of candidiasis is associated with impaired host immune defence and is particularly relevant in patients with reduced CD4-positive T-cell counts. Although candidiasis is not specific to HIV infection, its recurrent, extensive, or treatment-resistant presentation should prompt assessment of possible underlying immunosuppression [2,3,24].

Oral hairy leukoplakia is another important lesion associated with HIV infection. It is related to Epstein–Barr virus reactivation in the setting of impaired cellular immunity and most commonly affects the lateral borders of the tongue. Clinically, it appears as a white, corrugated or vertically folded plaque that cannot be removed by scraping. Unlike pseudomembranous candidiasis, it generally does not disappear after wiping and is usually asymptomatic. Recognition of this lesion is important because it may indicate impaired immune function, although it can also occur in other immunosuppressed individuals [1,3,15].

Kaposi sarcoma is a vascular neoplasm associated with human herpesvirus 8 and is an important AIDS-defining malignancy. Intraoral lesions frequently involve the hard palate, gingiva, and tongue and may appear as reddish, violaceous, or dark-purple macules, plaques, or nodules. Lesions may progress in size, become ulcerated, or interfere with mastication and speech. Histopathological examination and appropriate immunohistochemical testing are essential for confirmation. The presence of suspicious oral lesions requires careful evaluation and coordination with the patient's HIV and oncology care teams [2,8,23].

Periodontal diseases associated with HIV infection have also received considerable attention. Linear gingival erythema, necrotizing ulcerative gingivitis, and necrotizing ulcerative periodontitis have been described among the characteristic periodontal conditions observed in immunocompromised patients. These conditions may present with intense gingival erythema, spontaneous bleeding, severe pain, necrosis of interdental papillae, rapid periodontal tissue destruction, and, in advanced cases, exposure of alveolar bone. Their clinical severity may be influenced by immune status, plaque accumulation, smoking, nutritional deficiencies, and other systemic factors [17–21].

Ulcerative lesions represent another clinically significant group of oral manifestations. Recurrent aphthous-like ulcerations may be unusually large, deep, painful, and persistent in patients with advanced immunosuppression. Viral infections, particularly herpes simplex virus and cytomegalovirus, may also cause oral ulceration. The clinical appearance, distribution, duration, and associated systemic findings are important in determining the appropriate diagnostic approach. Persistent or atypical ulcers require further investigation, including microbiological testing or biopsy when clinically

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indicated [2,5,8].

Salivary gland involvement and xerostomia may further compromise oral health in individuals living with HIV. Patients may experience reduced salivary flow, dry mouth, difficulty swallowing, altered taste, increased caries risk, and recurrent oral candidiasis. Salivary gland enlargement, including parotid involvement, has also been described. These manifestations may result from HIV-associated glandular changes, concomitant conditions, or medication-related adverse effects. Their recognition is important because salivary dysfunction can significantly affect oral comfort, nutrition, and quality of life [3,16].

The introduction of highly active antiretroviral therapy has changed the epidemiological pattern and clinical expression of HIV-associated oral disease. The improved immune function achieved through effective ART has contributed to a reduction in the prevalence and severity of several opportunistic oral lesions. Nevertheless, oral manifestations have not disappeared completely. They may persist in patients with delayed diagnosis, inadequate adherence, virological failure, incomplete immune recovery, or additional risk factors. Furthermore, some oral conditions may be influenced by medication-related adverse effects and changes in the oral microbial environment [9–11].

The clinical importance of oral manifestations extends beyond their local effects. Oral lesions may provide valuable information about the patient's immune status, treatment response, and general health. However, none of the oral manifestations described in HIV infection should be regarded as independently diagnostic of HIV. Similar clinical findings may occur in individuals without HIV infection, and the interpretation of oral lesions must always consider the patient's medical history, clinical examination, laboratory findings, and appropriate confirmatory investigations [1,2,11].

Dentists and oral healthcare professionals therefore have an important role in the early recognition of suspicious oral lesions, the identification of patients who may require further medical assessment, and the maintenance of oral health in individuals receiving HIV treatment. A systematic oral examination, appropriate diagnostic testing, timely referral, and coordinated multidisciplinary care can contribute to improved clinical outcomes and quality of life [2,5,17].

The aim of this narrative review is to provide a comprehensive overview of the principal oral manifestations associated with HIV/AIDS, describe their clinical and diagnostic characteristics, discuss their relationship with immune dysfunction and antiretroviral therapy, and outline the principles of differential diagnosis and clinical management. Particular attention is given to the recognition of characteristic oral lesions and their distinction from other infectious, inflammatory, and neoplastic conditions.

2. MATERIALS AND METHODS

2.1. Review design

This article was prepared as a narrative literature review addressing the clinical presentation, diagnosis, differential diagnosis, and management of oral manifestations associated with HIV/AIDS. The review focused on oral conditions described in clinical studies, systematic reviews, classification documents, specialist reviews, and relevant clinical guidelines.

2.2. Literature sources and search strategy

Relevant literature was identified through searches of PubMed/MEDLINE, PubMed Central, and available scientific and clinical guideline resources. Search terms included combinations of the following: “HIV oral manifestations,” “AIDS oral lesions,” “oral candidiasis HIV,” “oral hairy leukoplakia,” “Kaposi sarcoma oral,” “HIV periodontal disease,” “HIV oral ulceration,” “HIV salivary gland disease,” and “oral manifestations antiretroviral therapy.”

For the differential diagnosis section, additional terms included “hepatitis C oral lichen planus,” “hepatitis B oral manifestations,” and “HPV oral papillary lesions.”

2.3. Selection of literature

Publications were considered relevant if they addressed the clinical characteristics, pathogenesis, diagnosis, epidemiology, or management of oral lesions in people living with HIV. Priority was given to peer-reviewed clinical reviews, recognized classification studies, systematic reviews, and official clinical guidelines. Historical publications were included where they provided foundational diagnostic classifications or important descriptions of HIV-associated oral lesions.

2.4. Data synthesis

The selected literature was organized according to the principal clinical categories of oral disease. The review considered the following domains:

1. Infectious mucosal lesions.

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2. Epstein–Barr virus-associated oral hairy leukoplakia.
3. HIV-associated neoplastic lesions.
4. Periodontal manifestations.
5. Ulcerative oral lesions.
6. Salivary gland disorders and xerostomia.
7. Other oral and orofacial manifestations.
8. Differential diagnosis and clinical management.

As this is a narrative review, no pooled prevalence estimate or formal meta-analysis was performed. The findings were synthesized descriptively.

3. CLINICAL MANIFESTATIONS OF HIV/AIDS IN THE ORAL CAVITY

3.1. Oral candidiasis

Oral candidiasis is one of the most frequently recognized opportunistic fungal infections in people living with HIV. It is predominantly associated with Candida albicans, although non-albicans species may also be involved. The development of candidiasis is facilitated by impaired cell-mediated immunity, disruption of local mucosal defence, changes in the oral microbiota, xerostomia, antibiotic exposure, and other predisposing conditions [2,3,24].

Four principal clinical presentations are relevant in oral practice.

3.1.1. Pseudomembranous candidiasis

Pseudomembranous candidiasis is characterized by multiple white or cream-coloured plaques affecting the tongue, buccal mucosa, palate, and oropharyngeal mucosa. The plaques consist of fungal elements, desquamated epithelial cells, inflammatory cells, and debris. They can usually be removed by gentle scraping, exposing an erythematous or occasionally slightly bleeding mucosal surface.

Patients may report burning, altered taste, discomfort during eating, or difficulty swallowing when the infection extends into the oropharynx. However, some lesions remain asymptomatic.

The clinical diagnosis is generally based on characteristic morphology and distribution. Microscopic examination of a smear may demonstrate yeast cells and pseudohyphae. Fungal culture can be useful in recurrent, atypical, or treatment-resistant cases, although a positive culture must be interpreted in the clinical context because Candida may be present as a commensal organism.



Figure 1. Pseudomembranous Oral Candidiasis. Clinical presentation of pseudomembranous candidiasis characterized by multiple white, creamy plaques distributed over the oral mucosa, particularly the tongue and buccal mucosa. The plaques can usually be removed by gentle scraping, revealing an underlying erythematous surface. This is one of the most common oral fungal manifestations in patients with HIV infection and may indicate immunosuppression.

3.1.2. Erythematous candidiasis

Erythematous candidiasis presents as red, smooth, atrophic areas of the oral mucosa. The dorsal tongue and palate are common sites. Patients may complain of burning or sensitivity to spicy and acidic foods.

Unlike pseudomembranous candidiasis, erythematous candidiasis does not produce removable white plaques. The condition may be overlooked if the oral examination is limited to the presence of white lesions.



Figure 2. Erythematous Oral Candidiasis. Clinical appearance of erythematous candidiasis presenting as red, smooth, and atrophic areas of the oral mucosa, frequently involving the dorsal surface of the tongue and the hard palate. Patients may report burning sensations, discomfort, or altered taste. The condition is associated with Candida overgrowth and may occur in individuals with HIV-related immune dysfunction.

3.1.3. Chronic hyperplastic candidiasis

Chronic hyperplastic candidiasis is characterized by persistent white plaques that cannot be removed by scraping. It may affect the buccal mucosa, tongue, or other mucosal surfaces. Because non-scrapable white

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lesions have a broad differential diagnosis, clinical assessment alone may be insufficient.

Biopsy may be necessary to distinguish chronic hyperplastic candidiasis from epithelial dysplasia, leukoplakia, and other potentially malignant disorders. Histopathological examination may demonstrate epithelial hyperplasia and fungal hyphae within the superficial epithelial layers.



Figure 3. Chronic Hyperplastic Oral Candidiasis.

Clinical presentation of chronic hyperplastic candidiasis characterized by persistent, adherent white plaques that cannot be easily removed by scraping. Lesions commonly affect the buccal mucosa and may demonstrate a heterogeneous or slightly nodular surface. Histopathological examination may be necessary to confirm the diagnosis and exclude epithelial dysplasia or other potentially malignant disorders.

3.1.4. Angular cheilitis

Angular cheilitis is characterized by erythema, fissuring, maceration, and discomfort at one or both commissures of the mouth. Candida species may contribute to the condition, sometimes in association with Staphylococcus aureus, nutritional deficiencies, reduced vertical dimension, or local moisture accumulation.



Figure 4. Angular Cheilitis Associated with Oral Candidiasis. Clinical appearance of angular cheilitis characterized by erythema, fissuring, maceration, and occasional crust formation at one or both corners of the mouth.

Candida species, sometimes in combination with bacterial microorganisms, may contribute to the development of these lesions. Angular cheilitis can occur in patients with HIV infection, particularly in the presence of additional predisposing factors such as xerostomia or nutritional deficiencies.

Clinical significance: Recurrent or extensive candidiasis may be an indicator of impaired immune function, but it is not specific to HIV. Assessment should include local predisposing factors, medication history, diabetes status where appropriate, and consideration of HIV testing when clinically indicated and performed with appropriate consent [2,3,24].

3.2. Oral hairy leukoplakia

Oral hairy leukoplakia is an Epstein–Barr virus-associated epithelial lesion most commonly observed in immunocompromised individuals. It is classically located on the lateral borders of the tongue and may be unilateral or bilateral.

Clinically, it appears as a white, non-removable plaque with a corrugated, vertically folded, or shaggy surface. The lesion is usually asymptomatic and does not generally cause significant functional impairment.

The principal diagnostic distinction is between oral hairy leukoplakia and other white oral lesions, particularly pseudomembranous candidiasis, frictional keratosis, and leukoplakia. Unlike candidiasis, oral hairy leukoplakia cannot be removed by scraping. Unlike many frictional lesions, it is not necessarily associated with a clear mechanical irritant.

Histopathological examination may demonstrate epithelial hyperplasia, hyperparakeratosis, and characteristic changes in the superficial epithelial layers. EBV-associated testing, including in situ hybridization for EBV-encoded RNA where available, may support the diagnosis in uncertain cases [1,3,15].

Oral hairy leukoplakia is clinically important because it may be associated with immune suppression. Nevertheless, it is not exclusive to HIV infection and should not be used as a standalone diagnostic marker.

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Figure 5. Oral Hairy Leukoplakia. Clinical presentation of oral hairy leukoplakia characterized by non-removable, vertically corrugated white plaques, predominantly affecting the lateral borders of the tongue. The lesion is associated with Epstein–Barr virus (EBV) infection and is particularly relevant in patients with impaired cellular immunity. Unlike pseudomembranous candidiasis, the white plaques cannot be removed by scraping.

3.3. Oral Kaposi sarcoma

Kaposi sarcoma is a vascular neoplasm associated with human herpesvirus 8 (HHV-8). It is one of the classical AIDS-associated malignancies and may involve the skin, oral mucosa, lymph nodes, and internal organs.

The oral cavity is a clinically important site of involvement. The hard palate is frequently affected, although lesions may also occur on the gingiva, tongue, buccal mucosa, and oropharyngeal tissues.

Early lesions may appear as flat red, purple, or brown macules. With progression, they may develop into raised plaques or nodules. Lesions can become ulcerated, bleed, or interfere with mastication, speech, and oral hygiene.

The clinical differential diagnosis includes vascular malformations, hemangioma, pyogenic granuloma, melanoma, and other pigmented or vascular lesions. A biopsy is generally required for confirmation when clinically appropriate. Histopathological findings may include spindle-shaped cells, slit-like vascular spaces, and extravasated erythrocytes. Immunohistochemical demonstration of HHV-8 latent nuclear antigen (LANA-1) supports the diagnosis. Management requires coordination with infectious disease and oncology specialists. Treatment depends on the extent of disease, symptoms, immune status, and systemic involvement [2,8,23].



Figure 6. Oral Kaposi Sarcoma. Clinical appearance of oral Kaposi sarcoma presenting as reddish-purple, violaceous, or dark-brown macular, nodular, or plaque-like lesions.

The hard palate and gingiva are commonly affected, although lesions may develop at other intraoral sites. Kaposi sarcoma is associated with HHV-8 and represents an important AIDS-defining malignancy. Histopathological examination is required for definitive diagnosis.

3.4. Periodontal manifestations

Periodontal conditions associated with HIV infection include linear gingival erythema, necrotizing ulcerative gingivitis, and necrotizing ulcerative periodontitis. These lesions may be more severe in patients with impaired immune function, although their occurrence is influenced by multiple local and systemic factors [17–21].

3.4.1. Linear gingival erythema

Linear gingival erythema is characterized by a distinct red band along the marginal gingiva. It may extend to the attached gingiva and is sometimes accompanied by petechiae or spontaneous bleeding.

The condition may be disproportionate to the amount of visible plaque. However, the diagnosis should not be made solely on the basis of gingival redness. Plaque-induced gingivitis, medication-related gingival changes, and other inflammatory conditions should be considered.



Figure 7. Linear Gingival Erythema. Clinical presentation of linear gingival erythema characterized by a distinct, red band along the marginal gingiva, sometimes

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extending into the attached gingiva. The erythema may be disproportionate to the amount of dental plaque and may persist despite conventional oral hygiene measures. This condition is recognized as an HIV-associated periodontal manifestation and may be related to altered host immune responses and microbial interactions.

3.4.2. Necrotizing ulcerative gingivitis

Necrotizing ulcerative gingivitis is characterized by acute gingival pain, spontaneous bleeding, necrosis of interdental papillae, and the presence of a greyish pseudomembrane. Halitosis and metallic taste may also occur. The interdental papillae may appear punched out, with rapid tissue destruction in severe cases. Fever, malaise, and regional lymphadenopathy may occur in more extensive disease.



Figure 8. Necrotizing Ulcerative Gingivitis in a Patient with HIV Infection.

Clinical appearance of necrotizing ulcerative gingivitis characterized by painful gingival inflammation, interdental papillary necrosis, spontaneous bleeding, and the presence of a grayish pseudomembrane. Patients may also experience halitosis and marked gingival tenderness. The condition is associated with impaired host defences and may progress rapidly without appropriate treatment.

3.4.3. Necrotizing ulcerative periodontitis

Necrotizing ulcerative periodontitis involves periodontal attachment and supporting tissues. Clinical findings may include severe pain, necrosis of gingival tissues, rapid attachment loss, tooth mobility, and alveolar bone destruction.

The condition requires prompt periodontal assessment and treatment. Management includes gentle removal of necrotic debris, plaque control, pain management, and treatment of associated systemic or local factors. Systemic antimicrobial therapy may be considered in selected patients according to the severity and clinical circumstances.

Clinical significance: Necrotizing periodontal diseases should prompt assessment of immune status and systemic health, but they are not pathognomonic for HIV infection [17–21].



Figure 9. Necrotizing Ulcerative Periodontitis. Clinical presentation of necrotizing ulcerative periodontitis characterized by extensive soft-tissue necrosis, severe periodontal inflammation, interdental tissue destruction, and rapid loss of periodontal attachment. In advanced cases, exposed alveolar bone and significant gingival recession may be observed. This severe periodontal condition is particularly important in patients with advanced HIV-associated immunosuppression.

3.5. Oral ulcerative lesions

Oral ulceration in people living with HIV may be caused by opportunistic viral infections, aphthous-like disease, trauma, medication-related effects, immune-mediated disorders, or malignancy.

3.5.1. Major aphthous-like ulcers

Major aphthous-like ulcers may be larger, deeper, more painful, and more persistent than conventional minor aphthous ulcers. They may affect the tongue, buccal mucosa, soft palate, or other non-keratinized mucosal surfaces.

The ulcers are often round or oval, with a fibrin-covered base and an erythematous margin. Severe lesions may interfere with eating, swallowing, and speaking.

A persistent or unusually large ulcer should not automatically be classified as aphthous disease. Biopsy and additional investigations may be required to exclude infection and malignancy.

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Figure 10. Aphthous-Like Oral Ulceration. Clinical appearance of an aphthous-like ulcer characterized by a well-defined, painful ulcer with a yellowish-white fibrinous base and an erythematous peripheral halo. Lesions may occur on the non-keratinized oral mucosa, including the labial and buccal mucosa, ventral tongue, and soft palate. In patients with HIV infection, ulcers may be larger, more persistent, and more painful than conventional recurrent aphthous lesions.

3.5.2. Herpes simplex virus-associated lesions

Herpes simplex virus may cause recurrent oral vesicles and ulcers. In immunocompromised individuals, lesions may be extensive, prolonged, atypical, and more painful than in immunocompetent patients.

The clinical presentation depends on the site of infection. Lesions may involve the lips, gingiva, tongue, palate, and other oral surfaces. Intraoral recurrent herpes classically affects keratinized mucosa, although atypical presentations can occur in immunocompromised patients.

When the clinical diagnosis is uncertain, viral PCR testing from an appropriate lesion sample may be useful.



Figure 11. Herpes Simplex Virus-Associated Oral Lesions.

Clinical presentation of oral herpes simplex virus infection, demonstrating grouped vesicles and/or multiple painful erosions and ulcers involving the oral mucosa. In immunocompromised patients, lesions may be extensive, persistent, and atypical in appearance. The diagnosis may be supported by lesion-based molecular testing, particularly when the clinical presentation is not characteristic.

3.5.3. Cytomegalovirus-associated ulceration

Cytomegalovirus may cause large, deep, persistent oral ulcers, particularly in patients with advanced immunosuppression. The ulcers may have irregular margins and a necrotic or fibrin-covered base.

Because clinical findings are not specific, histopathological examination and appropriate immunohistochemical or molecular investigations may be necessary. CMV-associated oral ulceration should be considered in the differential diagnosis of persistent ulcers in severely immunocompromised patients.



Figure 12. Cytomegalovirus-Associated Oral Ulceration.

Clinical appearance of a deep, persistent oral ulcer with irregular margins and an ulcerated base, potentially associated with cytomegalovirus infection in patients with advanced immunosuppression. CMV-related oral ulcers are uncommon but may be clinically significant because of their prolonged course and potential resemblance to other infectious or neoplastic lesions. Histopathological examination, including appropriate immunohistochemical testing, may be required to establish the diagnosis.

3.6. Salivary gland disorders and xerostomia

Salivary gland involvement in HIV infection may include gland enlargement, reduced salivary flow, and symptoms of xerostomia. Parotid enlargement may be unilateral or bilateral and may be associated with lymphoepithelial cystic changes.

Xerostomia may cause:

- Difficulty swallowing dry foods.
- Burning sensation of the oral mucosa.

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- Altered taste.
- Increased dental caries.
- Increased susceptibility to candidiasis.
- Difficulty wearing removable prostheses.
- Reduced oral comfort and quality of life.

The evaluation should include medication history, assessment of hydration, examination of salivary gland regions, and measurement of salivary flow when indicated. Other causes of dry mouth, including medication adverse effects and systemic disease, should be considered [3,16].



Figure 13. HIV-Associated Salivary Gland Enlargement.

Clinical presentation of salivary gland enlargement in a patient with HIV infection, typically involving the parotid glands and presenting as unilateral or bilateral swelling in the preauricular region. The enlargement may be associated with lymphoepithelial cystic changes and may occur together with xerostomia or reduced salivary flow. Clinical examination and appropriate imaging may help characterize the underlying glandular changes.

3.7. Other oral manifestations

Additional oral findings reported in people living with HIV include:

- Oral melanotic pigmentation.
- Recurrent herpes zoster involving the trigeminal distribution.
- Non-Hodgkin lymphoma involving the oral cavity or oropharynx.
- Oral squamous cell carcinoma.
- Recurrent bacterial infections.
- Delayed healing of oral lesions in selected immunocompromised patients.

These conditions are not necessarily caused directly by HIV. Their occurrence may be influenced by immune dysfunction, coexisting infections, medication exposure, smoking, alcohol use, and other risk factors.

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Any persistent, indurated, enlarging, or unexplained oral lesion should be evaluated according to standard oral medicine and oncology principles.



Figure 14. Oral Melanotic Pigmentation. Clinical appearance of multiple brown to dark-brown macular areas of pigmentation involving the oral mucosa.

Oral pigmentation in patients with HIV infection may be associated with several factors, including antiretroviral medication, post-inflammatory changes, or other local and systemic conditions. A careful clinical history and examination are necessary to establish the likely cause and exclude alternative diagnoses.

Additional clinical consideration: Non-Hodgkin lymphoma and oral squamous cell carcinoma should remain in the clinical differential diagnosis of persistent oral masses, indurated ulcers, or unexplained tissue enlargement. These lesions require timely specialist assessment and histopathological confirmation. They should not be diagnosed solely on the basis of clinical appearance.



Figure 15. Oral Non-Hodgkin Lymphoma.

Clinical presentation of an intraoral mass involving the oral mucosa, potentially appearing as a rapidly enlarging, painless or painful swelling with or without surface ulceration. Oral non-Hodgkin lymphoma is an important HIV-associated malignancy and may involve the gingiva, palate, tongue, or other oral sites. Histopathological examination and immunohistochemical analysis are essential for definitive diagnosis and classification.

4. CLASSIFICATION OF HIV-ASSOCIATED ORAL MANIFESTATIONS

The classification of oral lesions associated with HIV infection provides a practical framework for clinical examination and research [1].

Table 1. Classification of oral manifestations associated with HIV infection

Clinical category	Representative conditions	Typical clinical presentation
Fungal infections	Pseudomembranous candidiasis	Removable white plaques
Fungal infections	Erythematous candidiasis	Red, atrophic mucosal areas
Viral-associated lesions	Oral hairy leukoplakia	Non-removable corrugated white plaques, usually on lateral tongue
Neoplastic lesions	Kaposi sarcoma	Red, purple, or brown macules, plaques, or nodules
Periodontal conditions	Linear gingival erythema	Marginal gingival erythema
Periodontal conditions	Necrotizing ulcerative gingivitis	Pain, bleeding, necrotic interdental papillae
Periodontal conditions	Necrotizing ulcerative periodontitis	Necrosis and rapid periodontal destruction
Ulcerative conditions	Major aphthous-like ulcers	Large, deep, persistent ulcers
Viral infections	HSV-associated lesions	Vesicles and ulcers
Viral infections	CMV-associated ulcers	Large, persistent ulcers
Salivary disorders	HIV-associated salivary gland disease	Gland enlargement and/or reduced salivary flow
Other conditions	Oral pigmentation	Brown or dark mucosal macules
Neoplastic conditions	Non-Hodgkin lymphoma	Persistent mass or ulcerative lesion

5. DIFFERENTIAL DIAGNOSIS

The diagnosis of oral lesions in people living with HIV requires careful consideration of conditions with similar clinical appearances. Differential diagnosis is particularly important because many oral lesions are not specific to HIV infection.

Hepatitis B and C and human papillomavirus infection are considered in this section only as potential alternative or coexisting diagnoses. They should not be classified as primary HIV-associated oral manifestations.

5.1. Differential diagnosis of white lesions

White oral lesions may result from fungal infection, viral-associated epithelial changes, frictional keratosis, leukoplakia, lichenoid disorders, and other mucosal conditions.

Pseudomembranous candidiasis is usually characterized by removable plaques. Oral hairy leukoplakia is typically non-removable and affects the lateral tongue. Frictional keratosis is usually located at sites exposed to chronic mechanical irritation. Persistent non-removable white lesions may require biopsy to exclude epithelial dysplasia or malignancy.

5.2. Differential diagnosis of red and erosive lesions

Red oral lesions may be associated with erythematous candidiasis, inflammatory mucosal disorders, trauma, vascular lesions, and epithelial dysplasia.

Oral lichen planus may present with bilateral reticular white striae, erythematous areas, or erosive lesions. In some populations, hepatitis C virus infection has been associated with oral lichen planus; however, the strength of this association varies geographically and between studies. The presence of oral lichen planus should therefore not be interpreted as evidence of HIV infection or hepatitis C infection without appropriate investigations [26].

5.3. Hepatitis B and C in the differential diagnosis

Hepatitis B and C are systemic viral infections that may coexist with HIV infection. Their oral implications are less specific than the classical HIV-associated lesions.

Hepatitis C has been investigated in relation to oral lichen planus, oral sicca symptoms, and other oral findings. The association between hepatitis C and oral lichen planus is not uniform across all populations, and geographical variation has been reported. Therefore, oral lichen planus should be regarded as a possible associated condition rather than a diagnostic marker of hepatitis C [26].

Hepatitis B does not have a single characteristic oral

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lesion that can reliably establish the diagnosis. Oral findings in patients with hepatitis B may be related to coexisting conditions, systemic illness, medication exposure, or other factors. Diagnosis requires appropriate serological testing rather than clinical examination of the oral cavity.

5.4. Human papillomavirus-associated oral lesions

HPV may cause several papillary or verruciform lesions of the oral mucosa, including:

- Squamous papilloma.
- Verruca vulgaris.
- Condyloma acuminatum.
- Multifocal epithelial hyperplasia.

These lesions may present as exophytic growths with a papillary, cauliflower-like, or verrucous surface. Their appearance may overlap with other benign and malignant oral lesions.

Clinical examination, histopathological assessment, and selected molecular tests may be required to establish the diagnosis. HPV-associated lesions should not automatically be considered HIV-associated lesions, although HPV infection and HIV infection may coexist in the same patient [27,28].

5.5. Comparative differential diagnostic table

Table 2. Differential diagnosis of oral lesions potentially encountered in people living with HIV

Clinical finding	HIV-associated condition to consider	Important differential diagnoses	Diagnostic approach
Removable white plaques	Pseudomembranous candidiasis	Chemical burns, debris, other superficial infections	Clinical examination, microscopy when indicated
Non-removable white plaques on lateral tongue	Oral hairy leukoplakia	Frictional keratosis, leukoplakia, lichenoid lesion	Clinical examination, biopsy or EBV testing if uncertain
Persistent white plaque	Chronic hyperplastic candidiasis	Leukoplakia, epithelial dysplasia, carcinoma	Biopsy and fungal assessment
Red atrophic mucosal areas	Erythematous candidiasis	Erythroplakia, trauma, inflammatory disease	Clinical examination, microbiological testing where appropriate
Reticular white striae and erosions	Not specific to HIV	Oral lichen planus, lichenoid reaction	Clinical and histopathological assessment; consider HCV testing where indicated
Purple or brown palatal lesion	Kaposi sarcoma	Hemangioma, vascular malformation, melanoma	Biopsy and HHV-8 immunohistochemistry
Papillary exophytic lesion	Not specific to HIV	HPV-associated papilloma, verruca, condyloma, other neoplasms	Histopathology; HPV testing in selected cases
Large persistent ulcer	HSV, CMV, aphthous-like ulceration	Trauma, medication reaction, lymphoma, squamous cell carcinoma	Microbiological testing and/or biopsy
Gingival erythema and necrosis	HIV-associated periodontal disease	Conventional plaque-induced disease, necrotizing periodontal disease in other immunocompromised states	Periodontal examination and systemic assessment
Xerostomia and gland enlargement	HIV-associated salivary gland disease	Medication effects, Sjögren syndrome, dehydration, other salivary disorders	Medication review, salivary assessment, imaging or laboratory tests when indicated

6. DIAGNOSTIC APPROACH

6.1. Clinical examination

A systematic oral examination is essential when evaluating a patient with known or suspected HIV

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infection. The examination should include:

1. Inspection of the lips and oral commissures.
2. Examination of the buccal mucosa and vestibules.
3. Inspection of the dorsal, lateral, and ventral surfaces of the tongue.
4. Examination of the hard and soft palate.
5. Assessment of the gingiva and periodontal tissues.
6. Inspection of the floor of the mouth.
7. Examination of the oropharyngeal region where clinically appropriate.
8. Palpation of suspicious lesions and regional lymph nodes when indicated.

Each lesion should be documented according to site, size, colour, surface characteristics, consistency, number, duration, symptoms, and progression.

6.2. Laboratory and histopathological investigations

Investigations should be selected according to the suspected diagnosis.

Table 3. Diagnostic investigations for oral lesions associated with HIV infection

Suspected condition	Investigation	Diagnostic purpose
Oral candidiasis	Direct microscopy and fungal culture in selected cases	Demonstration and identification of Candida
Oral hairy leukoplakia	Biopsy and EBV-associated testing when needed	Support diagnosis in atypical cases
Kaposi sarcoma	Incisional or excisional biopsy, HHV-8 LANA-1 immunohistochemistry	Histopathological confirmation
HSV ulceration	PCR from lesion swab where available	Detection of HSV DNA
CMV ulceration	Biopsy and appropriate immunohistochemistry	Confirmation of tissue-invasive CMV
Persistent white lesion	Biopsy	Exclusion of dysplasia and malignancy
Persistent unexplained ulcer	Biopsy and selected microbiological tests	Exclusion of infection and neoplasia
Suspected immune suppression	HIV testing with informed consent and appropriate counselling	Establishment of HIV status
Known HIV infection	CD4-positive T-cell count and viral load	Assessment of immune status and virological control

6.3. Diagnostic Algorithm 1: Evaluation of a patient presenting with an oral lesion

Algorithm text (for inclusion in the manuscript)

1. Initial clinical assessment

- Patient presents with an oral lesion or mucosal abnormality.
- Record the chief complaint, onset, duration, progression, pain, bleeding, and associated symptoms.

2. Medical history and risk assessment

- Establish known HIV status, antiretroviral therapy (ART), adherence, and history of opportunistic infections.
- Review CD4+ T-cell count, HIV viral load, medications, and relevant systemic conditions.
- If HIV status is unknown and clinically appropriate, offer confidential HIV testing with informed consent.

3. Comprehensive oral examination

- Assess the lips, buccal mucosa, tongue, gingiva, palate, floor of the mouth, and oropharynx.
- Document lesion location, size, color, morphology, distribution, surface characteristics, and clinical appearance.
- Identify single or multiple lesions and assess associated lymphadenopathy.

4. Clinical characterization and preliminary differential diagnosis

- White lesions: pseudomembranous or erythematous candidiasis, oral hairy leukoplakia, frictional keratosis, and other keratotic disorders.
- Red lesions: erythematous candidiasis, Kaposi sarcoma, inflammatory lesions, and erythroplakia.
- Ulcerative lesions: recurrent aphthous-like ulceration, HSV, CMV, traumatic ulcers, and squamous cell carcinoma.

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- Pigmented or proliferative lesions: Kaposi sarcoma, lymphoma, melanotic lesions, and other neoplasms.
- Periodontal lesions: linear gingival erythema, necrotizing ulcerative gingivitis, and necrotizing periodontitis.

5. Targeted diagnostic investigations

- Cytology, direct microscopy, or fungal culture when candidiasis is suspected.
- HSV or CMV PCR when clinically indicated.
- Biopsy and histopathological examination for persistent, atypical, suspicious, or unexplained lesions.
- Immunohistochemistry and additional molecular investigations when indicated.
- HIV testing and immunological assessment when HIV infection is suspected or not previously established.

6. Final diagnosis and clinical correlation

- Integrate clinical findings, medical history, laboratory results, and histopathological evidence.
- Determine whether the lesion is HIV-associated, opportunistic infection-related, neoplastic, inflammatory, traumatic, or unrelated to HIV.

7. Management and follow-up

- Initiate lesion-specific treatment.
- Refer for specialist assessment when malignancy, severe opportunistic infection, or advanced immunosuppression is suspected.
- Coordinate care with the HIV/infectious disease specialist when appropriate.
- Reassess treatment response and investigate lesions that persist, recur, or progress.

Important diagnostic principle: A suspicious lesion should not be attributed to HIV infection solely on clinical appearance. Persistent, indurated, unexplained, or potentially malignant lesions require timely biopsy and histopathological evaluation.



Figure 15. Diagnostic algorithm for the clinical evaluation of a patient presenting with an oral lesion.

6.4. Diagnostic Algorithm 2: Differential diagnosis according to lesion morphology

Algorithm text

The diagnostic approach is based on the predominant clinical morphology of the oral lesion, followed by targeted investigations to establish the differential and final diagnosis.

1. White lesions

- Removable white plaques → pseudomembranous oral candidiasis.
- Non-removable white plaques, particularly on the lateral tongue → oral hairy leukoplakia (EBV-associated).
- Persistent white keratotic lesions → frictional keratosis, leukoplakia, or epithelial dysplasia.
- Confirmatory investigations: clinical assessment, fungal microscopy/culture when indicated, and biopsy for atypical or persistent lesions.

2. Red lesions

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- Diffuse erythematous mucosal changes → erythematous candidiasis.
- Linear erythema along the gingival margin → linear gingival erythema.
- Red-purple macules, plaques, or nodules → Kaposi sarcoma.
- Persistent red patches → erythroplakia or epithelial dysplasia.
- Investigations: fungal testing, clinical assessment, and biopsy with histopathology when indicated.

3. Ulcerative lesions

- Multiple recurrent shallow ulcers → recurrent aphthous-like ulceration.
- Painful vesicles or ulcers → herpes simplex virus infection.
- Large, persistent, deep ulcers → CMV-associated ulceration, particularly in advanced immunosuppression.
- Non-healing, indurated ulcers → squamous cell carcinoma until excluded.
- Investigations: HSV/CMV PCR when indicated, biopsy, and histopathological examination.

4. Nodular, proliferative, or pigmented lesions

- Red-purple vascular lesions → Kaposi sarcoma.
- Firm nodules or rapidly enlarging masses → lymphoma or other neoplasms.
- Brown or black macules → oral melanotic pigmentation, medication-related pigmentation, or other pigmented disorders.
- Investigations: incisional or excisional biopsy, histopathology, and immunohistochemistry when appropriate.

5. Gingival and periodontal lesions

- Linear gingival erythema → HIV-associated periodontal manifestation.
- Painful necrosis of interdental papillae → necrotizing ulcerative gingivitis.
- Extensive periodontal tissue destruction → necrotizing periodontitis.
- Investigations: periodontal examination, probing, radiographic assessment, and evaluation of systemic immune status.

6. Final diagnostic classification

Following morphological assessment and targeted investigations, lesions are classified as:

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- HIV-associated oral manifestations;
- Opportunistic infections;
- HIV-associated neoplastic lesions;
- Inflammatory or immune-mediated conditions;
- Traumatic, medication-related, or unrelated oral disorders.

Clinical diagnostic note: Morphology provides an initial diagnostic direction but is not sufficient for definitive diagnosis. Histopathological confirmation is particularly important for suspected Kaposi sarcoma, lymphoma, epithelial dysplasia, and oral squamous cell carcinoma.

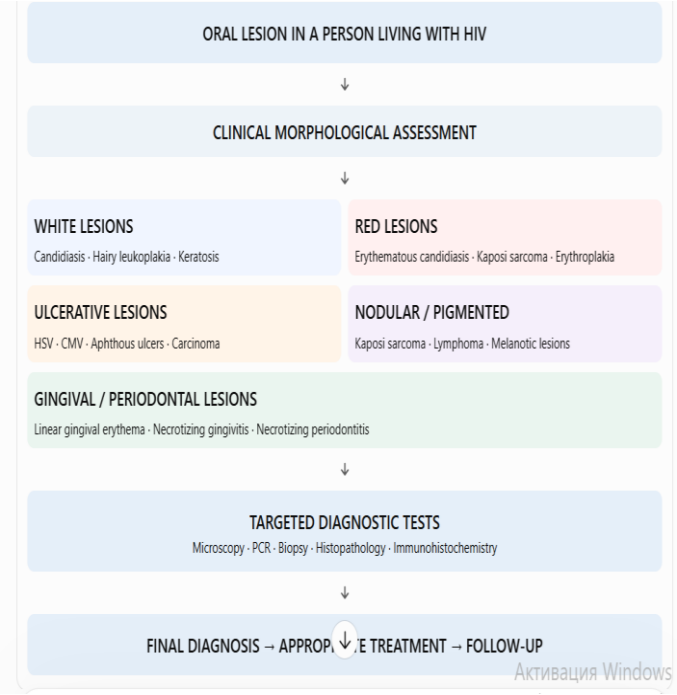


Figure 16. Morphology-based diagnostic algorithm for oral lesions in people living with HIV.

7. MANAGEMENT AND TREATMENT

Management of oral manifestations in people living with HIV should be individualized according to the underlying diagnosis, severity, immune status, ART regimen, drug interactions, and general health.

7.1. General principles

The general principles of management include:

- Establishing an accurate diagnosis.
- Identifying local and systemic predisposing factors.
- Assessing the patient's current ART and medication history.
- Treating the specific oral condition.
- Maintaining effective oral hygiene.
- Managing pain and functional impairment.

- Monitoring response to treatment.
- Referring patients to infectious disease or oncology specialists when appropriate.

The dentist should not independently discontinue or modify ART. Decisions concerning antiretroviral treatment should be made by the treating HIV specialist.

7.2. Management of oral candidiasis

Treatment depends on the clinical extent of infection, severity of symptoms, recurrence, and immune status. Topical antifungal agents may be appropriate for selected mild oropharyngeal disease, while systemic antifungal treatment may be required for more extensive or severe infection.

The NIH opportunistic infection guidelines provide recommendations for the treatment of mucocutaneous candidiasis in adults and adolescents with HIV [24].

In recurrent or refractory disease, clinicians should reassess the diagnosis, adherence, possible antifungal resistance, and contributing factors such as xerostomia, diabetes, and medication exposure.

7.3. Management of oral hairy leukoplakia

Oral hairy leukoplakia is frequently asymptomatic and may not require direct local treatment. Management should focus on assessment of immune status and optimization of HIV treatment in coordination with the treating specialist.

Atypical or diagnostically uncertain lesions should undergo further evaluation to exclude other white mucosal disorders.

7.4. Management of Kaposi sarcoma

Treatment depends on the extent of disease, symptoms, immune status, and systemic involvement. Management may include optimization of ART, local treatment for selected lesions, radiotherapy, or systemic oncological therapy.

Oral lesions should be assessed by clinicians experienced in oral pathology and oncology. Biopsy confirmation is important before definitive treatment when clinically appropriate.

7.5. Management of periodontal disease

Periodontal treatment includes:

- Professional removal of plaque and calculus.
- Gentle debridement of necrotic tissue where indicated.
- Effective daily oral hygiene.
- Periodontal maintenance.
- Pain control.
- Correction of contributing local factors.
- Assessment for systemic involvement in severe disease.

Systemic antibiotics are not required for every periodontal lesion. Their use should be determined by the severity of infection, systemic signs, and individual clinical circumstances [17,18,25].

7.6. Management of oral ulceration

Treatment should be directed at the underlying cause. HSV and CMV infections require appropriate medical assessment and pathogen-specific treatment.

Aphthous-like ulcers may require local symptomatic therapy and evaluation for associated systemic or immune abnormalities.

Persistent or atypical ulcers should be biopsied when indicated. Empirical treatment should not delay investigation of lesions suspicious for malignancy.

7.7. Management of xerostomia and salivary gland disorders

Management may include:

- Adequate hydration.
- Regular use of saliva substitutes where needed.
- Sugar-free salivary stimulants.
- Avoidance of tobacco and irritating oral products.
- Review of medications associated with dry mouth.
- Fluoride-based caries prevention.
- Regular dental assessment.
- Management of associated candidiasis.

Patients with persistent salivary gland enlargement, pain, or asymmetry may require imaging and specialist assessment [16].

7.8. Prevention and long-term oral care

Preventive care should include regular dental examinations, professional plaque control, caries prevention, periodontal maintenance, smoking cessation support, and education regarding oral symptoms that require early assessment.

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People living with HIV should have access to routine dental care without discrimination. Standard infection prevention and control precautions should be applied to all patients, regardless of known HIV status.

8. DISCUSSION

Oral manifestations remain an important clinical component of HIV infection despite the substantial progress achieved through modern antiretroviral therapy. Their clinical relevance is related not only to local symptoms and functional impairment but also to their potential association with systemic immune dysfunction. The literature consistently identifies oral candidiasis, oral hairy leukoplakia, Kaposi sarcoma, and characteristic periodontal diseases among the most clinically significant oral conditions associated with HIV infection [1–5].

The findings discussed in this review support the importance of interpreting oral lesions within the broader clinical context of HIV infection. Oral candidiasis, for example, is frequently encountered in patients with impaired cellular immunity and may be associated with reduced CD4-positive T-cell counts. However, candidiasis is also common in patients with diabetes mellitus, those receiving corticosteroids or antibiotics, and individuals with local predisposing factors. Therefore, its presence alone cannot establish an HIV diagnosis. Recurrent, extensive, or treatment-resistant candidiasis should instead be regarded as a reason for further clinical evaluation when the patient's medical history or risk profile supports such investigation [2,3,24].

Oral hairy leukoplakia has particular diagnostic value because of its characteristic clinical appearance and association with Epstein–Barr virus reactivation. Its typical localization along the lateral borders of the tongue and its non-removable, corrugated white surface help distinguish it from pseudomembranous candidiasis. Nevertheless, clinical examination may not always be sufficient, particularly when lesions are atypical or when other white mucosal disorders are suspected. In such cases, histopathological examination and appropriate EBV-associated investigations may be useful [1,3,15].

Kaposi sarcoma represents a different diagnostic and clinical challenge. Unlike superficial infectious lesions, it is a vascular neoplasm that may initially appear as a painless red, purple, or brown macule. The palate and gingiva are frequently affected, although other oral sites may also be involved. Because several vascular and pigmented lesions may have a similar appearance, clinical suspicion must be followed by histopathological confirmation and, where appropriate, HHV-8 immunohistochemical assessment. Early recognition is important because oral involvement may be part of disseminated disease and requires multidisciplinary management [2,8,23].

The periodontal manifestations of HIV infection also deserve particular attention. Linear gingival erythema, necrotizing ulcerative gingivitis, and necrotizing ulcerative periodontitis may be associated with severe inflammation, tissue necrosis, pain, and rapid periodontal destruction. However, these conditions are not exclusive to HIV infection. Their occurrence and severity are influenced by several interacting factors, including oral hygiene, smoking, nutritional status, microbial composition, systemic health, and immune function. Consequently, the diagnosis should be based on a comprehensive periodontal examination and assessment of relevant systemic factors rather than on HIV status alone [17–21].

Ulcerative oral lesions present a broad differential diagnostic spectrum. Major aphthous-like ulcers may be unusually large and persistent in immunocompromised patients, while HSV and CMV infections may produce painful or extensive ulceration. Traumatic ulcers, medication-related lesions, immune-mediated disorders, and malignant neoplasms must also be considered. Persistent ulcers, particularly those with induration, irregular borders, progressive enlargement, or unexplained bleeding, require timely investigation and often biopsy. This approach reduces the risk of attributing a potentially serious lesion to HIV-associated immunosuppression without adequate diagnostic evidence [2,5,8].

Salivary gland disorders and xerostomia are additional factors that may substantially affect oral health. Reduced salivary flow can contribute to dental caries, mucosal discomfort, difficulty swallowing, taste disturbances, and increased susceptibility to opportunistic infections. These problems may reflect HIV-associated glandular changes, concomitant systemic disease, or adverse effects of medication. Assessment of salivary function and review of the patient's medication history are therefore important components of comprehensive oral care [3,16].

The influence of ART on oral manifestations is particularly important when interpreting the contemporary clinical presentation of HIV infection. Effective treatment improves immune function and has reduced the frequency of several opportunistic oral conditions. Nevertheless, oral lesions continue to occur, particularly in individuals with delayed diagnosis, incomplete immune recovery, poor adherence, virological failure, or additional predisposing conditions. The persistence of oral disease in the ART era indicates that oral examination remains clinically relevant and should

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be incorporated into routine HIV care [9–11].

A further consideration is the diagnostic overlap between HIV-associated lesions and conditions caused by other infectious agents. In the differential diagnosis of oral white, red, ulcerative, pigmented, and papillary lesions, clinicians should consider a broad range of conditions. Hepatitis C-associated oral lichen planus and HPV-related papillary lesions are relevant examples, but they should not be classified as primary oral manifestations of HIV. Their inclusion in the differential diagnosis helps prevent incorrect attribution of a lesion to HIV and emphasizes the need for appropriate clinical and histopathological assessment [26–28].

The clinical value of oral examination should therefore be understood as supportive rather than independently diagnostic. Certain lesions may raise suspicion of underlying immunosuppression, but no single oral finding can confirm HIV infection or reliably determine disease stage. HIV diagnosis requires appropriate laboratory testing, while assessment of immune status depends on relevant clinical and laboratory parameters, including CD4-positive T-cell count and viral load. Oral findings should be interpreted as part of an integrated diagnostic process [1,2,11].

From a practical perspective, dentists and oral medicine specialists can contribute to early recognition, referral, and ongoing management. Their responsibilities include documenting lesion morphology and distribution, identifying signs of opportunistic infection or neoplasia, obtaining appropriate diagnostic samples, providing local treatment when indicated, and coordinating care with infectious disease specialists. Preventive oral healthcare, periodontal maintenance, management of xerostomia, and regular follow-up are also important components of long-term care [2,17,24,25].

Several limitations should be considered when interpreting the available literature. Studies differ in their populations, geographical settings, definitions of oral lesions, diagnostic criteria, ART exposure, and immune status of participants. Changes in treatment availability and HIV epidemiology over time may also influence the reported prevalence of individual lesions. Consequently, prevalence estimates from older studies may not accurately represent contemporary populations receiving effective ART. Further prospective studies using standardized diagnostic criteria and clearly defined immunological parameters are needed to clarify the current epidemiological pattern of HIV-associated oral disease [1,10,11,17].

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Overall, the evidence supports the continued importance of systematic oral examination in people living with HIV. Although the prevalence of some classical lesions has declined with effective ART, oral candidiasis, hairy leukoplakia, periodontal disease, ulcerative lesions, salivary dysfunction, and HIV-associated malignancies remain clinically relevant. Accurate diagnosis requires recognition of characteristic clinical features, consideration of alternative causes, and confirmation through appropriate investigations. A multidisciplinary approach integrating dental, oral medicine, infectious disease, and oncological expertise remains essential for optimal patient care.

9. CONCLUSION

Oral manifestations represent an important clinical component of HIV/AIDS and may reflect opportunistic infection, immune dysfunction, inflammatory disease, salivary gland involvement, or neoplastic complications. Oral candidiasis, oral hairy leukoplakia, Kaposi sarcoma, linear gingival erythema, necrotizing periodontal diseases, persistent ulcerative lesions, and salivary dysfunction are among the principal conditions requiring recognition by dental and medical professionals.

Although effective ART has reduced the frequency and severity of several classical oral manifestations, these conditions remain clinically relevant. Their presence should prompt appropriate assessment, but no oral lesion alone can establish an HIV diagnosis or determine the stage of infection.

Accurate diagnosis requires a systematic oral examination, careful consideration of alternative causes, and the use of microbiological, histopathological, and laboratory investigations when indicated. Hepatitis B/C-associated conditions and HPV-related oral lesions should be considered within the differential diagnosis rather than classified as primary oral manifestations of HIV.

Dentists and oral medicine specialists play an important role in early recognition, prevention, treatment, and referral. Integration of oral healthcare into comprehensive HIV management is essential for reducing oral morbidity and improving patients' quality of life.

10. CLINICAL IMPLICATIONS

1. Recurrent or extensive oral candidiasis should prompt evaluation of possible underlying immune dysfunction.
2. Non-removable corrugated white plaques on the lateral tongue should raise suspicion of oral hairy leukoplakia.

3. Red, purple, or brown palatal lesions require assessment for Kaposi sarcoma and other vascular or pigmented lesions.
4. Necrotizing periodontal disease warrants prompt treatment and consideration of systemic risk factors.
5. Persistent or atypical oral ulcers require investigation and possible biopsy.
6. Xerostomia should be assessed because it increases the risk of caries, candidiasis, and oral discomfort.
7. Oral lesions should never be used as the sole basis for diagnosing HIV infection.
8. Suspected HIV infection should be addressed through confidential, appropriate testing and referral.
9. Hepatitis B/C and HPV should be considered in the differential diagnosis of relevant oral lesions.
10. Dental care should be integrated into the multidisciplinary management of people living with HIV.

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Competing and Conflicting Interests

The authors declare that they have no competing or conflicting interests.

Ethical Approval

Not applicable.

Informed Consent

Written informed consent for publication of the clinical data and images relating to the illustrative case was obtained from the patient. The signed consent form is retained and archived by the authors and is available to the editorial office on request.

Data Availability

All data discussed in this review are contained within the cited published sources. No new dataset was generated or analysed for this article.

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