



ORAL CAVITY: A MIRROR TO HIV MANIFESTATIONS

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ABSTRACT

A multitude of oral lesions have been described in individuals infected with the human immunodeficiency virus. Human immunodeficiency virus infection is a major interest and concern to dentists and other health care workers because of the many varieties of oral lesions often associated with Human immunodeficiency virus infection. Individual's tested seropositive for Human immunodeficiency virus infections are more susceptible to occurrence of lesions in oral cavity. This review covers the clinical and therapeutics aspects of Human immunodeficiency virus associated oral diseases. Data was collected from detailed search through research and review articles of scientific journals and magazines as well as through Pubmed and National medical library database.

Keywords: Human Immunodeficiency Virus infection, Oral diseases, Oral manifestations.

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is an infectious disease caused by the Human Immunodeficiency Virus (HIV), and is characterized by profound immunosuppression that leads to opportunistic infections, secondary neoplasm and neurologic manifestations¹.

HIV disease has an effect over the entire body. It is not practical in the present scenario for any health personnel dealing with diagnosis and treatment in humans to not encounter this dreaded disease and its manifestations. Thus it becomes imperative to be aware of the various forms of HIV manifestations.

Oral health is an important component of the overall health status in HIV infection. Awareness of the variety of oral disorders which can develop throughout the course of HIV infection and coordination of health care services between a physician and a dentist may improve the overall health of the patient. The spectrum of oral manifestations is very vast in HIV-AIDS².

Based on standard classification and diagnostic criteria, common HIV-associated oral disorders can be broadly classified into four categories by pathophysiological process: infection, neoplasm, immune-mediated, and others³.

Infection

- Fungal : Candidiasis; Cryptococcus; Histoplasmosis; Aspergillosis
- Viral : Herpes simplex virus; Oral Hairy leukoplakia(Epstein Barr); Human Papilloma virus; Cytomegalovirus, MolluscumContagiosum
- Bacterial: Bacillary angiomatosis; linear erythematous gingivitis; necrotizing ulcerative gingivitis, necrotizing ulcerative periodontitis; necrotizing stomatitis ;syphilis (Treponemappallidum).

Neoplasm

- Fungal : Kaposi's Sarcoma
- Viral : Non-Hodgkin's lymphoma

Immune-Mediated

- Fungal : Major Aphthous

Other Diseases

- Fungal : Xerostomia; Parotid disease
- Viral : Pain syndromes
- Bacterial : Nutritional

There is no particular oral lesion, which is uniquely associated with HIV infection, or more lesions require that HIV infection be considered as a possible underlying cause. Some oral lesions, such as oral candidiasis and oral hairy leukoplakia, are so strongly associated with HIV infection that they have been incorporated into the Centers for Disease Control (CDC) and Prevention clinical classification of HIV disease.

Indeed, the emergence of one or more oral lesions correlates highly with HIV progression. A CD4 lymphocyte counts of less than 200/mm³ is a reliable prognosticator of active disease and probability of shortened lifespan. The spectrum of HIV- associated oral lesions also varies with transmission risk-factor, gender, age, and health-care access^{1,4}.

Oral health is an important component of overall health status in HIV infection. Even common dental diseases such as caries and periodontal disease have greater impact on patients with HIV infection. Painful HIV-associated oral diseases such as necrotizing ulcerative periodontitis and stomatitis, major aphthous ulceration, candidiasis, and Kaposi's sarcoma impair ingestion of food and negatively impact on nutritional health. Therefore, it is essential that the physician and dentist, together, identify and reduce risk factors for oral disease in the patient with HIV infection³.

INFECTION

Candidiasis

The most common HIV-related oral lesion is candidiasis, predominantly due to *Candida albicans*. While *Candida* can be isolated from 30–50% of the oral cavities of healthy adults, making it a constituent of the normal oral flora, clinical oral candidiasis rarely occurs in healthy patients^{3,5}.

In stark contrast, clinical oral candidiasis has been reported to occur in 17–43% of patients with HIV infection and in more than 90% of patients with AIDS. One report found that unexplained oral candidiasis in healthy adults with risk factors for HIV infection predicted the development of clinical signs of AIDS within 3 months³.

Based on clinical appearance, oral candidiasis can appear as one of four distinct clinical entities:

- Erythematous or atrophic candidiasis
- Pseudomembranous candidiasis
- Hyperplastic or chronic candidiasis
- Angular cheilitis

In all cases, the infection is superficial. While in most instances the clinical appearance is adequate to arrive at a diagnosis, simple exfoliative cytology will identify the characteristic budding yeast and hyphae when the clinical diagnosis is uncertain.

Treatment of oral candidiasis is determined by the clinical type, distribution, and severity of infection. Topical treatment is effective for limited and accessible lesions³.

- Clotrimazole troches, Nystatin pastilles, and Nystatin oral suspension are effective for mild-to-moderate erythematous and pseudomembranous candidiasis.
- Chlorhexidine 0.12% oral rinses do not contain a cariogenic substrate and may be similarly effective.
- Topical Amphotericin B is also a useful non-cariogenic treatment for resistant candidiasis and can be prepared by dissolving 50 mg in 500 ml of sterile saline (0.1 mg/ml).
- Regular, gentle mechanical debridement with soft gauze soaked with 10% Povidone-Iodine or Chlorhexidine 0.12% solution is very effective in managing focal, limited candidiasis.
- Clotrimazole 1% cream, Miconazole or Ketoconazole 2% cream, Nystatin ointment, and Hydrocortisone iodoquinol 1% cream are useful medications for angular cheilitis and for application to a removable denture base when there is candidal infection involving the underlying mucosa.

When long-term treatment or prophylactic use is required, systemic antifungal therapy is often required. Systemic treatment for oral candidiasis involves the use of Imidazole (Ketoconazole) and Triazole (Fluconazole and Itraconazole) antifungal medications.

Ketoconazole is hepatotoxic and requires gastric acid for absorption, thereby limiting its usefulness in patients with HIV infection who may also have developed gastric achlorhydria.

Fluconazole is an excellent systemic antifungal medication with a favorable therapeutic index, making it the preferred systemic antifungal medication.

Itraconazole (100 mg tablet, 1–2 tabs/day) is another excellent systemic antifungal for use alone, or in combination with Fluconazole (100 mg tablet, 1–2 tabs/day), for resistant candidiasis.

Deep Fungal Infections: Unlike the superficial infection of candidiasis, several systemic fungal infections can infrequently lead to single or multiple, deep oral lesions with the potential for considerable local tissue destruction.

Cryptococcosis, Histoplasmosis, Aspergillosis, and Mucormycosis are uncommon oral deep fungal infections which require histological diagnosis. Treatment typically requires the use of intravenous antifungal therapy with Amphotericin.

Histoplasmosis

It is a granulomatous fungal disease caused by *Histoplasma capsulatum*. The clinical presentation ranges from an asymptomatic or mild lung infection to an acute or chronic disseminated form.

Oral histoplasmosis appears as chronic ulcerated areas located on the dorsum of the tongue, palate, floor of the

mouth, and vestibular mucosa. Focal or multiple sites can be involved. In AIDS patients, histoplasmosis is rarely curable, but it can be controlled with long-term suppressive therapy consisting of the administration of Amphotericin B and Ketoconazole².

Cryptococcosis

Oral manifestations are quite unusual and only two cases have been reported in the literature. The lesions consist of ulcerations of the oral mucosa, but the clinical diagnosis of oral cryptococcus may be difficult since other microbial infections and trauma may show similar appearances. Tissue biopsy may be required for the diagnosis and treatment involves use of Amphotericin B².

Herpesvirus accounts for the majority of HIV-related oral viral infections, most frequently as recurrent oral herpes due to Herpes simplex virus (HSV) or Epstein-Barr virus (EBV)-induced oral hairy leukoplakia (OHL). Less commonly occurring viral infections involving the oral cavity include cytomegalovirus and human papilloma virus³.

Herpes Simplex Viruses

HSV is responsible for both primary and recurrent infections of the oral mucosa. These infections are acquired in childhood and after initial pustular lesions. The virus remains dormant, but in later stages of immunosuppression, the virus can be activated and can lead to various manifestations.

Oral manifestations, represented by diffuse mucosal ulcerations, are accompanied by fever, malaise, and cervical lymphadenopathy. Intraoral herpes in healthy individuals results in multiple, small, shallow ulcerations with irregular raised white borders. Small clusters of lesions usually coalesce to form a larger ulcer, which heals uneventfully in 7–10 days.

Ulcerations that follow the rupture of vesicles are painful and may persist for several weeks. Recurrent HSV usually appears in keratinizing oral mucosa (i.e., palate, dorsum of tongue, and gingiva) as ulcerations but in most HIV-seropositive patients, this rule is not followed. In these patients, the lesions may show unusual clinical aspects and persist for many weeks².

While the prevalence of seropositive HSV and the rate of reactivation is similar among both HIV-infected and non-infected populations, estimated to be 60% for those older than 30 years of age, recurrent intraoral HSV in patients with HIV infection often results in ulceration and pain of longer duration^{3,6,7}.

Recurrent intraoral HSV lesions occur more commonly on poorly keratinized tissue like the buccal and labial mucosa, an uncommon site in healthy individuals. The pain associated with persistent herpetic ulceration can result in reduced oral intake of food and significant weight loss. Clinical diagnosis can be assisted by culture and examination of a cytologic smear for the virus. Culture results should be interpreted with caution due to the high HSV seropositivity and the potential for false negative results due to silent shedding of HSV.

Intraoral HSV infection responds well to systemic Acyclovir, 2 grams daily in divided doses. However, the incidence of Acyclovir-resistant HSV has increased among patients with HIV infection. For most of these cases, oral Famciclovir and Valacyclovir and intravenous Foscarnet alone or in combination are effective. Topical Acyclovir is approved for genital HSV infections, but has been found to have little therapeutic effect for oral HSV^{3,8}.

Although Penciclovir was recently released as the first topical

antiviral medication indicated for treatment of herpes simplex labialis, it reduced time to healing by only 0.7 days in healthy individuals compared to vehicle placebo, and no data on its use in patients with HIV infection are available.

Oral Hairy Leukoplakia

Although originally postulated to be pathognomonic for HIV infection, this lesion has subsequently been reported in other immune deficiency states as well as in immunocompetent individuals.

It appears as an asymptomatic adherent white patch with vertical corrugations, most commonly on the lateral borders of the tongue. It may infrequently be confused with hypertrophic candidiasis and is predominantly found in homosexual males².

Oral hairy leukoplakia has since been shown to be associated with a localized Epstein-Barr virus (EBV) infection and occurs most commonly in individuals whose cluster of differentiation 4 (CD4) lymphocyte count is less than 200/mm³.

While the diagnosis is most often clinical, histological inspection will reveal typical epithelial hyperplasia suggestive of EBV infection.

This asymptomatic lesion does not require treatment. However, for cosmetic purposes, some patients may request treatment. Oral Acyclovir (3,200 mg daily in divided doses), topical Podophyllum resin, Retinoids, and surgical removal have all been reported as successful treatments. In most cases, the lesion returns after initial therapy, thus requiring prophylactic treatment with Acyclovir 200 mg daily³.

Human Papilloma Virus (HPV)

In some patients with HIV infection, human papilloma virus (HPV) causes a focal epithelial and connective tissue hyperplasia, forming an oral wart.

In HIV-infected patients, oral HPV-related lesions have a papillomatous appearance, either pedunculated or sessile, and are mainly located on the palate, buccal mucosa, and labial commissure².

The most common genotypes found in the mouth of patients with HIV infection are 2, 6, 11, 13, 16, and 32. Surgical removal, with or without intraoperative irrigation with Podophyllum resin, is the treatment of choice³.

Cytomegalovirus (CMV)

Cytomegalovirus related oral ulcerations, although infrequent, are a recognized complication of HIV infection. The diagnosis of oral CMV is based upon the presence of large intranuclear and smaller cytoplasmic CMV inclusions in the endothelial cells at the base of the ulcerations. These infections usually manifest in stage IV of the infection when there is advanced immunosuppression with a CD4 count below 50².

Disseminated CMV infection must be diagnosed as early as possible because of the serious nature of its sequelae, including retinitis and meningitis. CMV has been detected postmortem in one or more organ systems in as many as 90% of patients with AIDS.

Oral CMV infection typically appears as a solitary, chronic deep ulceration most often involving the buccal and labial mucosa. Clinically, it is indistinguishable from other nonspecific ulcerations such as chronic HSV and major aphthous ulceration.

Thus, biopsy and histological inspections are essential for definitive diagnosis.

Ganciclovir is the drug of choice, now available in a tablet formulation. Many patients with a history of CMV are placed on a prophylactic regimen (1.0 g Ganciclovir, 3 times daily with food)^{2,3}.

Molluscum Contagiosum

Molluscum contagiosum is caused by an unclassified DNA virus of the poxvirus family. Lesions appear as single or multiple papules on the skin of the buttocks, back, face, and arms. Molluscum contagiosum usually affects children and young adults and is spread by direct and indirect contact. The typical lesion is an umbilicated papule that may itch, leading to autoinoculation. Lesions may persist for years and eventually regress spontaneously. The occurrence of disseminated molluscum contagiosum has been reported in HIV-infected patients. These lesions usually subside with immune reconstitution when patients are started on Highly Active Antiretroviral Therapy (HAART)².

Although isolated cases of oral infection with *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Actinomyces israelii*, *Escherichia coli*, and *Mycobacterium avium-intracellulare* have been reported in patients with HIV infection, the most common oral lesions associated with bacterial infection are linear erythematous gingivitis, necrotizing ulcerative periodontitis, and, much less commonly, bacillary epithelioid angiomatosis and syphilis.

In the case of the periodontal infections, the bacterial flora is no different from that of a healthy individual with periodontal disease. Thus, the clinical lesion is a manifestation of the altered immune response to the pathogens.

Linear Erythematous Gingivitis

It is defined as a distinct erythematous band of marginal gingiva with either diffuse or punctuate erythema of the attached gingiva⁹.

In early studies it was often further characterized by a discrepancy between the amount of plaque and the intensity of the inflammation. The prevalence of Linear Erythematous Gingivitis in HIV infected population varies from 0 to 49%¹⁰. This considerable variation can be due to lack of clear diagnosis standardization and the heterogeneity of the population's studies. The existence of Linear Erythematous Gingivitis as a periodontal disease entity that is strongly associated with HIV has been questioned by some and others suggested that Linear Erythematous Gingivitis is mostly misdiagnosed as gingivitis^{11,12,13}.

Necrotizing Ulcerative Gingivitis (NUG) and Necrotizing Ulcerative Periodontitis (NUP)

- These may be different stages of the same disease. Necrotizing Ulcerative Gingivitis (NUG) results in the destruction of one or more interdental papillae and remains confined to the marginal gingival.
- Necrotizing Ulcerative Periodontitis (NUP) extends to marginal gingival, involves the periodontal ligaments and the alveolar bone, leading to a loss of attachment.

Most studies show a higher prevalence of NUG and NUP in HIV infected patients than in non-HIV infected patients^{14,15}.

NUP may be used as a marker for immune deterioration with a 95% predictive value that CD4⁺ cell counts have decreased below 200 cells μl^{-1} . If untreated, the cumulative probability of death within 24 months is 72.9%¹⁶.

HIV-associated NUG and NUP were initially considered specific disease conditions. Microbiology there is no major

difference between HIV-positive and HIV negative subjects^{17,18,19}.

A study was done to examine by Transmission Electron Microscope and Scanning Electron Microscope the supra-gingival microbial plaque overlying the ulcerated gingival papillae of NUP lesions in HIV-seropositive patients. Microscopic examination revealed a surface biofilm comprised of a mixed microbial flora of various morphotypes in 81.3% of biopsy specimens. The subsurface flora featured dense aggregations of spirochetes in 87.5% of specimens. Zones of aggregated Polymorphonuclear neutrophil's and necrotic cells were also noted. Yeasts were observed in 65.6% of specimens and herpes-like viruses in 56.5% of the specimens. Collectively, except for the presence of yeast and viruses, the results suggested that the microbial flora and possibly the soft tissue lesions of NUP and NUG are very similar¹⁸.

The prevalence of chronic periodontitis in HIV positive subjects varies considerably from 5 to 69%²⁰.

When compared with HIV negative counterparts, HIV-positive patients with chronic periodontitis suffer from a greater loss of attachment over time^{21,22}.

Necrotizing Stomatitis

Necrotizing stomatitis is an uncommon acute, painful ulceration which often exposes underlying bone and leads to considerable tissue destruction. This lesion may be a variant of major aphthous ulceration, but occurs in areas overlying bone and is associated with severe immune deterioration. Unlike NUP, the lesion may occur in edentulous areas. As in major aphthous ulceration, systemic corticosteroid medication or topical steroid rinse is the treatment of choice³.

Bacillary Epithelioid Angiomatosis (BEA)

This recently described lesion appears to be unique to HIV infection and is often clinically indistinguishable from oral Kaposi's sarcoma (KS).

Since both may present as an erythematous, soft mass which may bleed upon gentle manipulation, biopsy and histological examination are required to distinguish Bacillary Epithelioid Angiomatosis from Kaposi's sarcoma. The presumed etiological pathogen, *Rochalimaea henselae*, can be identified using Warthin-Starry staining.

Both Kaposi's sarcoma and Bacillary Epithelioid Angiomatosis are histologically characterized by atypical vascular channels, extravasated red blood cells, and inflammatory cells. However, prominent spindle cells and mitotic figures occur only in Kaposi's sarcoma.

Erythromycin (Erythromycin estolate, 500 mg 4 times daily for at least 10 days) is the treatment of choice for Bacillary Epithelioid Angiomatosis³.

Syphilis

While the prevalence of syphilis infection has risen significantly over the past decade, it is an uncommon cause of intraoral ulceration, even in HIV infection. Its appearance is no different from that observed in healthy individuals; it is a chronic, non-healing, deep, solitary ulceration often clinically indistinguishable from that due to tuberculosis, deep fungal infection, or malignancy.

Dark field examination may demonstrate *Treponema pallidum*. Positive reactive plasma reagin (RPR) and histological demonstration of *Treponema pallidum* is diagnostic.

Patients with newly diagnosed syphilis should be referred to

their physicians for evaluation and treatment; combination treatment with Penicillin, Erythromycin and Tetracycline is the treatment of choice, the dosage and duration of treatment depending on presence or absence of neurosyphilis³.

NEOPLASMS

Kaposi Sarcoma(KS)

Kaposi's sarcoma is the most common intraoral malignancy associated with HIV infection. Recognition of the lesion is essential, since oral Kaposi's sarcoma is often the first manifestation of the disease and is a diagnostic criterion for AIDS.

The lesion may appear as a red-purple macule, an ulcer, or as a nodule or mass. Intraoral Kaposi's sarcoma occurs on the heavily keratinized mucosa, the palate being the site in more than 90% of reported cases.

However, lesions have also been reported on the gingiva, tongue, and buccal mucosa. The skin should also be examined for lesions whenever oral lesions are discovered. Kaposi's sarcoma is especially common among homosexual and bisexual males and is rarely found in HIV-infected women³.

A new human herpes virus (HHV8) has recently been demonstrated to be an important cofactor in the development of Kaposi's sarcoma, and prophylaxis with Foscarnet and Ganciclovir, but not Acyclovir, has been shown to reduce the incidence of Kaposi's sarcoma in a large at-risk cohort²³.

Definitive diagnosis of Kaposi's sarcoma requires histological examination. There is no cure for Kaposi's sarcoma. Therapy for intraoral Kaposi's sarcoma should be instituted at the earliest sign of the lesion, the goal being local control of the size and number of lesions. When only one or a few lesions exist and the lesions are small (<1 cm), intralesional chemotherapy with Vinblastine sulfate (0.2–0.4 mg/ml per cm² of lesion) or sclerotherapy with 3% Sodium Tetradecyl sulfate (0.1–0.2 ml per cm² of lesion) is effective. Radiation therapy (800–2,000 cGy) is effective for larger or multiple lesions; stomatitis and glossitis are common side effect of radiation, but xerostomia is not³.

Non-Hodgkin's Lymphoma (NHL)

NHL is the most common lymphoma associated with HIV infection and is usually seen in late stages with CD4 lymphocyte counts of less than 100/mm³.

It appears as a rapidly enlarging mass, less commonly as an ulcer or plaque, and most commonly on the palate or gingiva. NHL may be indistinguishable from masses caused by Kaposi's sarcoma or other diseases in HIV-infected patients. Histological examination is essential for diagnosis and staging. Prognosis is poor, with mean survival time of less than one year, despite treatment with multi-drug chemotherapy^{3,24}.

IMMUNE MEDIATED ORAL LESIONS

While HIV infection and progression is characterized by progressive immune deterioration, it is equally well characterized by an abnormally activated immune system. In other words, the immune system activation itself leads to tissue injury and the worsening health of the patient.

Major Aphthous Ulceration

It is the most common immune-mediated HIV-related oral disorder, with a prevalence of approximately 2–3%. The large solitary or multiple, chronic, deep, painful ulcerations of major aphthae appear identical to those in non-infected

patients, but they often last much longer and are less responsive to therapy.

The diagnosis must include the possibility of a primarily infectious entity, which can be determined by histological examination of biopsy material. Treatment requires the use of a potent topical steroid such as Clobetasol (0.05% ointment applied for 45 seconds 3 times daily) when the lesion is accessible or Dexamethasone oral rinse (0.5 mg/5 ml Dexamethasone elixir 3 times daily, rinse for 45 seconds and expectorate) when inaccessible³.

When multiple ulcers are present or response to topical treatment is incomplete, systemic glucocorticosteroid therapy is required (Prednisone 1 mg/kg). Since lesions often return after discontinuing medication, the use of prophylactic medication is not uncommon.

Major aphthae is associated with advanced HIV infection (typically with CD4 lymphocyte counts of less than 50/mm³). Long-term use of systemic Prednisone may lead to complications such as oral candidiasis, reactivation of tuberculosis, or worsening of Kaposi's sarcoma.

Alternative therapies such as Dapsone 50–100 mg daily and Thalidomide 200 mg daily for 4 weeks should be considered²⁵.

When immunosuppressant or modulating drugs are used, concurrent antifungal medications such as Fluconazole 100 mg tablet 1–2 daily, Itraconazole 100 mg tablet 1–2 times daily, and antibacterial medications such as Chlorhexidine gluconate oral rinse 0.12% 3 times daily, may be required to prevent superinfection or opportunistic overgrowth³.

Xerostomia

Xerostomia is common in HIV disease, most often as a side effect of antiviral medications or of the other antihypertensive, antidepressant, anxiolytic or analgesic medications commonly prescribed for patients with HIV infection.

The oral dryness presents a significant risk factor for caries and can lead to rapid dental deterioration. Xerostomia also contributes to oral candidiasis, mucosal injury and dysphagia, and is often associated with pain and reduced oral intake of food.

Although several saliva substitutes exist, compliance is often poor and really inadequate. For patients with residual salivary gland function, determined by gustatory challenge, oral Pilocarpine (5 mg up to 3 times daily) often provides improved salivary flow and consistency. Oral hygiene instruction, regular maintenance, and the use of prescription-strength, fluoridated dentifrice is essential³.

OTHER DISEASES

Parotid Gland Disease

HIV infection is associated with parotid gland disease, characterized clinically by gland enlargement and diminished flow, and histologically by lymphoepithelial infiltration and benign cyst formation³.

The enlargement typically involves the tail of the parotid gland or, less commonly, the submandibular gland, and it may present uni- or bi-laterally with periods of increased or decreased size. While the appearance may raise suspicion of malignancy (salivary gland or lymphoma) or infection, aspiration of a yellow mucinous secretion supports the diagnosis of HIV-related salivary gland disease, thus avoiding unnecessary biopsy or imaging diagnostics.

Occasional swelling can be managed simply by repeated

aspiration and rarely is radical removal of the gland necessary. The pathophysiological mechanism is not known, though cytomegalovirus has been suggested to play a role²⁶.

Pain Syndromes

Pain is a common symptom experienced by patients with HIV infection. Pain may result from a wide variety of disease processes, including direct effects of HIV on the central or peripheral nervous system, infection, malignancy, and antiretroviral therapy³.

Headache is a common symptom, occurring in approximately 46% of patients with HIV infection and accounting for approximately 17% of all pains in patients with HIV infection²⁷.

Neuropathic pain is common among patients with HIV infection (19%), the most common diagnosis being painful peripheral sensory neuropathy. A complete discussion of pain syndromes in HIV infection is found elsewhere²⁸.

CONCLUSION

Potentially, all HIV infected individuals are equally at risk of presenting with oral HIV lesions at some time or the other during the disease process. A thorough examination of the oral cavity can easily detect most of the common lesions. An understanding of the recognition, significance, and treatment of said lesions by primary healthcare providers is essential for the health and well-being of people living with HIV disease.

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