Oral Manifestations of HIV Infection

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ABSTRACT

Acquired Immune Deficiency Syndrome (AIDS) is caused by the Human Immunodeficiency Virus (HIV). It is a critical disorder of the immune system which severely damages the body’s normal defences to infections. Oral lesions in Human Immunodeficiency Virus (HIV) infection are common. Over 30 different types of oral conditions have been reported to occur in patients with HIV disease. It must be understood, however, that the oral lesions seen in HIV positive patients are a result of immunodeficiency caused by the Human Immuno-deficiency virus and not due to the direct effect of the virus on oral tissues. Even common dental diseases such as caries and periodontal disease have greater impact on patients with HIV infection. Therefore, it is essential that the physician and dentist, together, identify and reduce risk factors for oral diseases in the patients with HIV infection.

KEYWORDS: HIV, AIDS, Immunodeficiency, Oral lesions.

INTRODUCTION

AIDS was officially recognized for the first time in June, 1981 at the centres for disease control, USA in previously healthy homosexual men dying with Pneumocystis Carinii pneumonia and Candidiasis. Since then AIDS had been reported from all the continents. (Reichart, 1987) Acquired Immune Deficiency Syndrome (AIDS) is caused by the Human Immunodeficiency Virus (HIV). It is a critical disorder of the immune system which severely damages the body’s normal defences to infections, thereby making it vulnerable to a host of infections/conditions including malignancies which then becomes life-threatening and fatal. (Abbas, 2005) HIV is thought to have originated in non-human primates in sub-Saharan Africa and transferred to humans early in the 20th century. The rapid spread of HIV/AIDS has led to an infringement of the human rights of men, women and children affected by the pandemic in various ways. The impact of HIV/AIDS has permeated the social, cultural and economic fabric of many nations. It has been estimated that 20% to 50% of all patients infected with HIV will develop some oral lesions associated with the disease. These lesions are often important markers in disease progression, and they may be the first manifestation of infection in a previously undiagnosed patient.¹ The oral lesions that are seen in patients infected with HIV can be broadly categorized as follows: (1) infections (fungal, viral, and bacterial); (2) neoplasms; (3) salivary gland disease; and (4) miscellaneous lesions, the last including effects attributable to treatment with Highly Active Anti-Retroviral Therapy (HAART). Due to multiple oral conditions and periodontal involvement, the dentist plays an essential role in early recognition of signs and symptoms of HIV disease or progression of the medical condition. (Narani et al., 2001)

ORAL LESIONS

Various oral manifestations seen in HIV are:

- Candidosis
- Herpes simplex

How to cite this article:
Herpes zoster
- Cytomegalovirus
- Oral hairy leukoplakia
- Kaposi sarcoma
- Non-Hodgkin’s lymphoma
- Necrotizing gingivitis/periodontitis

Candidosis: Oral candidosis which is an infection by Candida albicans is the most common lesion, with 90% of patients with AIDS affected at some point during their disease course. The lesions are more commonly seen when CD4 counts fall below 200/mm³. It was present in 40% to 75% of HIV-infected subjects with CD4 counts less than 200/mL. 65% of subjects with CD4 counts less than 200/mL and the presence of candidosis developed AIDS or died within a 24-month period. According to Ranganathan and Hemelatha, and Ranganathan et al, pseudomembranous candidiasis is the most common type seen in these patients. These authors also mentioned the presence of angular cheilitis as a frequent fungal infection, but in accordance with Noce et al, the type of candidiasis most frequently observed is the erythematous type. Cell mediated immunity plays a role in the pathogenesis of candidiasis in these patients. Both erythematous and pseudomembranous oral candidiasis are associated with increased risk for the subsequent development of opportunistic infections classifying the patient as having AIDS as defined by the Centers for Disease Control (CDC).

Herpes Simplex: Primary and recurrent intraoral and perioral herpes simplex is a relatively frequent viral infection, with a prevalence of 5% to 10%. The clinical features of oral herpes viral infection are usually similar to those seen in non-HIV-infected patients; however, in HIV-infected patients with severe immune suppression (CD4 cell counts usually below 100 cells/mm³) widespread, severe, and prolonged lesions develop which resembles the primary herpetic gingivostomatitis. In addition, recurrences are also common. Oral acyclovir (800 to 1200 mg/day) is usually the treatment of choice.

Herpes Zoster: It is caused by the reactivation of the Varicella virus (virus that causes chicken pox) in the trigeminal ganglion. Herpes Zoster of the oral mucosa and facial skin is a marker of HIV progression in HIV infected persons. (Scully et al., 1991) In immunocompetent patients under the age of 40, shingles is rare, but it is fairly common in patients who have HIV infection. Lesions in HIV patients also follow dermatomes, but are usually longer lasting and more likely to involve more than one dermatome in advanced HIV. Treatment of VZV infection is generally accomplished with acyclovir, with recommended therapy at 800 mg five times/day until resolution of the lesions occurs. Inadequate therapy may result in chronic persistence of the lesions or dissemination.

Cytomegalovirus: Oral CMV lesions seen as ulcers may occur in patients with advanced HIV disease. Generally these lesions are signs of underlying systemic disease involving gastrointestinal tract or the eye. Oral lesions due to CMV infection can occur anywhere on the oral mucosa. There are no characteristic appearances of CMV ulcers. Ulcers generally exhibit a white halo around the necrotic surface. Often these are confused with major aphthous ulcers or ulcers seen in necrotizing ulcerative periodontitis, necrotizing ulcerative stomatitis or lymphomas. (Eversole, 1992) Intravenous (IV) ganciclovir and acyclovir in high doses can be used for treatment.

Oral Hairy Leukoplakia: Oral hairy leukoplakia (OHL) is caused by Epstein-Barr virus in those with immune deterioration. It occurs in about 20 percent persons with asymptomatic HIV infection and becomes more common as the CD4+T-cell count drops. Presence of OHL is an indication of HIV infection and immunodeficiency. Though not common, in non-HIV population OHL is known to occur in those who have received bone, renal and heart transplants. Diagnosis of HL is an indication of both HIV infection and immunodeficiency. Also, it is an indication for a work-up to evaluate and treat HIV disease. HL correlates with a statistical risk for more rapid progression of HIV disease. Oral hairy leukoplakia is nearly always asymptomatic and self-limiting, and it requires no treatment. Antifungal therapy may resolve the symptoms of a superimposed candidosis but will not resolve the
Kaposi Sarcoma: Kaposi sarcoma (KS) is the single most common neoplasm occurring in patients with AIDS, but there has been a dramatic decline in its incidence since the advent of HAART. It occurs in approximately 15% to 20% of the patients with acquired immunodeficiency syndrome (AIDS). Human Herpes Virus-8 (HHV-8), a sexually transmitted virus has been implicated to be the causative organism of KS. Low CD4 counts, homosexuality and CMV disease are known to increase the probability of occurrence of KS. It has also been reported among non-HIV infected homosexual males. In HIV it may present as the first sign of AIDS. It presents as a flat, nodular or ulcerated mass depending on the stage of the tumour development and time of diagnosis. Lesions may be multifocal and skin involvement may be seen in association with oral KS. Most common in HIV infected male adults and less common in females and children infected with HIV. Palate, gingiva and tongue are the most commonly involved sites. Three clinical stages can be distinguished: patch, plaque, nodular. Occasionally, yellowish mucosa surrounds the KS lesion. Oral KS lesions may enlarge, ulcerate, and become infected. Good oral hygiene is essential to minimize these complications. Early lesions are asymptomatic, flat and red or purple in color. Advanced lesions show nodular appearance, become ulcerated and painful and may destroy bone. Treatment includes surgical (or carbon dioxide laser) excision. Radiation therapy is indicated for large and multiple lesions.

Non–Hodgkin’s Lymphoma: Non-Hodgkin’s lymphoma (NHL) is the second most prevalent intraoral neoplasm in patients with AIDS. HIV-positive patients are at 60 to 70 times greater risk of developing NHL than healthy individuals. Unlike KS, the incidence of NHL has not decreased with the advent of HAART, and it is estimated the incidence may actually increase as patients survive longer. It can occur anywhere in the oral cavity and is more common in males. It present as a painless soft tissue swelling with or without ulceration. Palate and gingiva are common sites. Lesion is generally single and extremely painful. Prognosis is poor, with most patients dying within the first year after diagnosis. Therapy depends on the stage of the disease: Radiation for regional disease and systemic chemotherapy for extra nodal disease. Treatment is given and monitored by the oncologist.

Necrotizing Ulcerative Gingivitis / Periodontitis: Necrotizing ulcerative periodontitis (NUP) is the most severe form of periodontal disease associated with HIV infection, and it develops in patients with marked immunosuppression. The prevalence of NUP is 5% or less. Necrotizing ulcerative gingivitis (NUG) is a fusospirochetal infection which is often associated with severe nutritional and/or immunodeficiency. NUG is characterized by destruction of one or more interdental papillae accompanied by necrosis, ulceration, and/or sloughing that is limited to the marginal gingival tissues. In the acute stage (ANUG), the gingival tissues appear fiery red and swollen, and are accompanied by yellowish-grey necrotic tissue that bleeds easily. Halitosis is a major feature. Regional lymph nodes may be enlarged and the constitutional findings such as fever and malaise are also present. Ulcers heal leaving the gingival papillae with a characteristic cratered appearance. Treatment includes thorough plaque removal, mechanical debridement of necrotic tissue, irrigation with povidone-iodine and systemic use of antibiotics. (Metronidazole 250 mg four times daily. Augmentin 250mg four times daily).

REFERENCES


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