Periodontal Diseases in HIV

S R Menon

Citation

S R Menon. *Periodontal Diseases in HIV*. The Internet Journal of Dental Science. 2017 Volume 14 Number 1.

DOI: 10.5580/IJDS.48158

Abstract

HIV infection in adults is related to various types of periodontal lesions, which include specific forms of gingivitis and necrotizing periodontal diseases, also with possible exacerbation of pre-existing periodontal disease. This article summarizes the treatment of these lesions

INTRODUCTION

In adults, HIV infection is related to a variety of periodontal lesions including necrotizing periodontal diseases and certain forms of gingivitis as well as due to possible exacerbation of pre-existing periodontal conditions. Risk Factors for periodontal diseases in HIV -infected individuals include age, smoking ,poor diet, poor oral hygiene, pre-existing gingivitis, CD4+ cell count (Glick et al, 1994b)5, viral load and certain species of microflora. Periodontal diseases associated with HIV infection are grouped into Linear Gingival Erythema (LGE), Necrotizing ulcerative Gingivitis (NUG) and Necrotizing Ulcerative Periodontitis (NUP). These are included among seven cardinal oral lesions accepted internationally, including Oral hairy leukoplakia, Oral candidiasis, Kaposi sarcoma, LGE, NUG, NUP and Non-Hodgkin lymphoma.

Previously, NUP and LGE were called HIV-P (HIV-associated periodontitis) and HIV-G (HIV-associated gingivitis) respectively. But now, there are indications that HIV-P and HIV-G do occur in HIV-negative immunocompromised individuals and hence makes their original terms unsuitable and hence were renamed as NUP and LGE. HIV-associated periodontal diseases along with other oral infections, are considered as serious complications of HIV infection and hence, have an important diagnostic and prognostic significance as they are one of the early occurring clinical features of infection and can predict the advancement of HIV disease to AIDS.

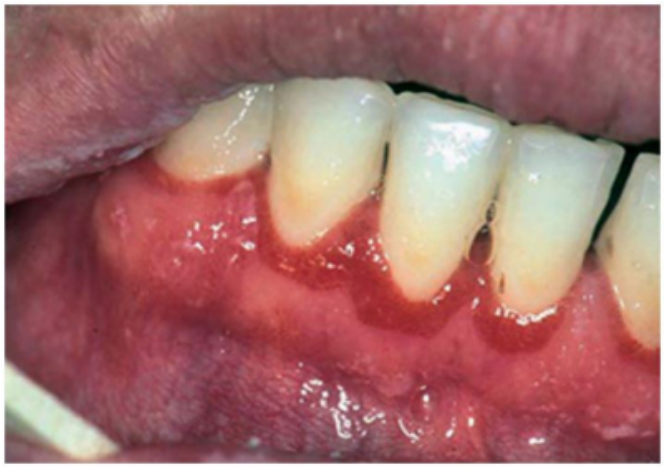
However, HIV-associated periodontal lesions are not included as criteria in CDC (Centers for Disease Control) classification (CDC, 1992) as they are less common than oral candidiasis and oral hairy leukoplakia. HIV associated periodontal infections have a well-defined and characteristic clinical appearance.

LINEAR GINGIVAL ERYTHEMA (LGE)

Linear Gingival Erythema (LGE) is a form of gingivitis characterized by distinct fiery red band along the margin of gingiva (EC-Clearinghouse, 1994) and is limited to the soft tissues of the periodontium. It is usually seen 2-3mm from the free gingival margin in anterior teeth, occasionally extending to the posterior teeth, presenting as a linear erythematous band. (Reznik , 2006)3. In some cases, it presents as petechia -like patches on free or attached gingiva causing bleeding and discomfort. There may be punctate erythema, which extends to the alveolar mucosa.

Sometimes, these areas fuse, creating diffuse erythematous zones from the gingival margin to the vestibule. Linear Gingival Erythema, marked by a marginal band of severe apical focal and/or diffuse areas of erythema, extending beyond the mucogingival line. It is associated with initial stages of HIV infection and CD4+ cell suppression. There are no pockets, ulceration or loss of attachment and is resistant to local treatment. The lesion may be localized generalized. Plaque that is commonly seen in conventional gingivitis is usually not associated with LGE. Bleeding on gentle probing is seen in majority of cases of LGE. Presently, the aetiopathology of LGE and other HIV associated periodontal diseases have been associated with Candida species. The microflora of LGE resembles periodontitis than gingivitis. (Clark et al, 1991)

Figure 1



MANAGEMENT :

LGE lesions undergo abrupt and quick remission and is often refractory to treatment. The treatment success relies on recognizing causative factors like tobacco, plaque, association with candida infection or existence of pathogenic bacteria consistent with those in conventional periodontitis . Treatment usually includes scaling and debridement by a dental professional, rinsing twice a day with 0.12% Chlorhexidine gluconate for 2 weeks and proper home care3.

NECROTIZING ULCERATIVE GINGIVITIS(NUG)

Necrotizing Ulcerative Gingivitis(NUG) is characterized by rapid onset of necrotic and ulcerated papillary and marginal gingiva covered by a yellowish-white or grey coloured slough or “pseudomembrane” , blunting of papillae, spontaneous bleeding, pain, bleeding on probing and fetid breath.2 But, Necrotizing Ulcerative Periodontitis (NUP) is characterized by sharp and intense pain, bleeding with ulcerated marginal gingiva. Rapid and extensive soft tissue necrosis with progressive periodontal attachment loss that results in bone exposure is seen.(Murray, 1994; Reznik,2006;Greenspan and Greenspan, 2008). NUG has been related to HIV infection and has a distinctive ulcerated and necrotic interdental papillae .It usually has a chronic or sub-acute course with spontaneous hemorrhage and characteristic fetor accompanied by severe pain. NUG rapidly progresses to NUP.

Figure 2

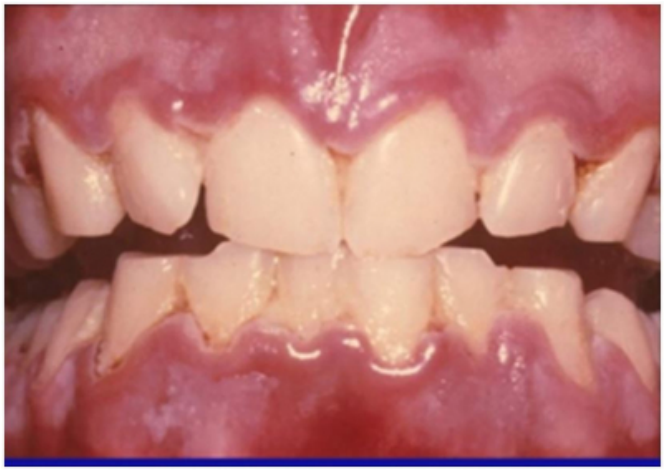
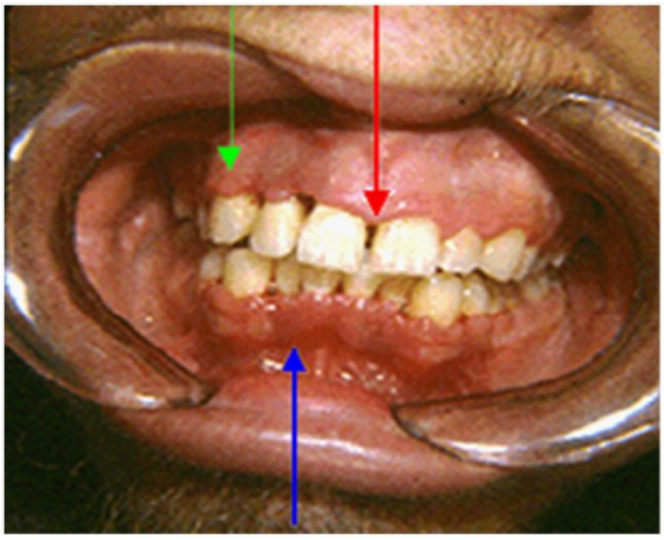


Figure 3



MANAGEMENT:

Cleaning and debridement of affected areas with plaque control is the treatment of choice. The patient should be seen at least every other day or daily for the first week. Debridement of affected areas is repeated at each visit along with precise plaque control methods. Patient should avoid using tobacco, alcohol etc. Antimicrobial mouth rinses such as chlorhexidine gluconate 0.12% must be prescribed. Systemic antibiotics (like amoxicillin or metronidazole) can be prescribed for patients with moderate to severe tissue destruction, localized lymphadenopathy or systemic or both. If required, prophylactic antifungal medication must be considered. One month after resolution of acute symptoms, the periodontium must be evaluated again to conclude treatment results and decide the need for additional treatment.

NECROTIZING ULCERATIVE PERIODONTITIS - NUP

Necrotizing Ulcerative Periodontitis - NUP is necrotizing, ulcerative, rapidly progressive form of periodontitis seen in HIV individuals. NUP may be generalized or localized and manifest as a continuation of NUG in which periodontal attachment loss and bone loss occurs. There is marked necrosis of soft tissue, rapid destruction of periodontium and interproximal bone loss in NUP. The osseous structures of the periodontium are affected in NUP. NUP might be seen after marked CD4+ cell depletion. The clinical features seen are intense pain, interproximal gingival necrosis, and craters in soft tissues. Spontaneous bleeding and joint pain are often complained by the patients. Destruction of the periodontium and bone may be extremely extensive and quick. Untreated NUP may extend into the adjacent tissues and expose the alveolar bone. When this occurs, the condition has been called Necrotizing Stomatitis. Bone is usually exposed resulting in necrosis and sequestration. The necrotizing lesions show spontaneous resolution on treatment, resulting in painless and deep interproximal craters that are difficult to clean and may lead to conventional periodontitis (Glick et al, 2000). Data indicates a similar microbiota in both NUP and chronic periodontitis (Glick et al 1994, Murray et al 1991). Patients with NUP were more likely to have CD4+ lymphocyte counts below 200/mm3 and is agreed that NUP is a predictive marker for severe immunodeficiency.

Figure 4

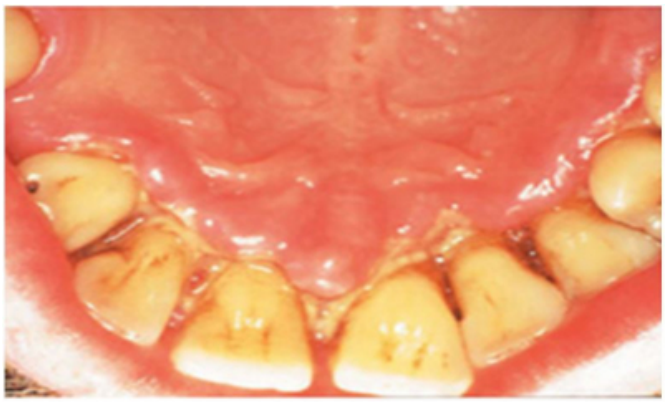
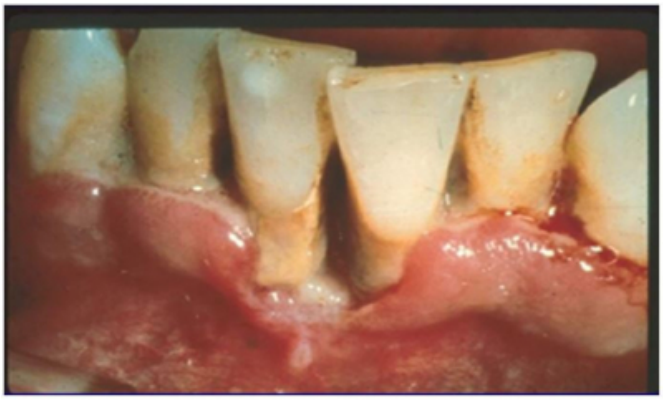


Figure 5



In 1994, Glick et al3,4have described a connection between CD4+ cells count below 200 cells/mm3 and NUP in HIV patients. It is proposed that NUP might be a good marker of immune suppression. The progress of periodontal disease in HIV infected patients need a detailed investigation as the HIV infection get chronic.

The etiology of NUP is undetermined, but a mixed fusiform-spirochete bacterial flora appears to play an important part. Because bacterial pathogens alone are not responsible for causing the disease, some predisposing “host” factors may be necessary. Numerous predisposing factors have been responsible for NUG, including smoking ,stress, poor oral hygiene, immunocompromised status, preexisting periodontal disease, viral infections and malnutrition. NUP is regularly associated with a diagnosis of AIDS or a positive HIV condition. NUP can progress quickly to tooth exfoliation, so treatment should include local debridement local antiplaque agents and systemic antibiotics. Early diagnosis and treatment of NUP are decisive because the osseous defects that occur in later stages are highly difficult to treat, even with extensive surgical procedure.

The pathogenesis of NUP associated with HIV infection is unclear. The subgingival bacterial flora in patients with HIV infections are not significantly different from those in other patients with periodontitis, with the exception that Candida and other pathogens can sometimes be seen in some patients, although it is considered that the imbalance and suppression of the local and systemic immune response results in hyper responsiveness of neutrophils in the lesions and exacerbation of the usual acute inflammatory response14 .There are no conclusive data to indicate that the pathogenesis of periodontal diseases in HIV-positive patients is different from that of HIV-negative patients. According to some studies, the subgingival microflora is almost identical

in HIV-positive and HIV-negative patients with periodontitis6,7,8,9. Further studies have described an increased presence of certain periodontal pathogens in HIV-positive patients compared to HIV-negative patients. These periodontal pathogens include Aggregatibacter actinomycetemcomitans, Fusobacterium nucleatum, Prevotella intermedia, Porphyromonas gingivalis,Treponema denticola and Tannerella forsythia.Murray et al ,1989; Cross and Smith, 1995; Scully et al,1999; Alpagot et al,2004). But, there are other studies that suggest lesser prevalence of putative pathogens in HIV-positive patients. (Tenenbaum et al,1997; Paster et al,2002; Patel et al,2003; Botero et al,2007;

Goncalves de Souza et al 2007). Many authors agree that several microbial species such as Candida spp, Clostridium Clostridiiformae, Enterobacterfaecalis, ,Clostridium difficile, Mycoplasma salivarium, Klebsiella pneumonia, , Pseudomonas aeroginosa, Enterobacter cloacae, Acinetobacter baumanii, which are often found in periodontal diseases of HIV-infected patients are unusual in other individuals. While the function of these microorganisms in the pathogenesis of periodontal disease in HIV positive patients is not yet completely understood,it is indicated that the greater presence of these opportunistic micro-organisms are due to severe immunosuppression in these patients.

In a recent review article, Feller and Lemmer15,14 suggested that spirochetes, herpes viruses, candida and HIV have a potential pathogenic role in NUP lesions in the HIV-seropositive individual. Spirochetes have the ability to modulate host innate and adaptive immune responses and to stimulate host inflammatory reactions, which may reduce local immune competence and facilitate development of necrotizing disease. Activated herpes virus have the capacity to deregulate host immune system, which may lead to an increase in the colonization and activity of other pathogenic microorganisms. Candida albicans has been reported to produce eicosanoids leading to the release of proinflammatory mediators, which may facilitate spirochete colonization and invasion, promoting the development of necrotizing periodontal disease. A compromised immune system in HIV-infected patient is driven by impaired T-cell function and altered T-cell ratios. It is well known that stress increases systemic cortisol levels, and sustained increase in cortisone have a suppressive effect on the immune response.

MANAGEMENT :

Treatment recommendations include systemic antibiotics(such as metronidazole , tetracycline, clindamycin, amoxicillin, and amoxicillin-clavulanate potassium) combined with debridement of necrotic tissues. Simultaneous administration of an antifungal agent should be considered as systemic antibiotics increase the patient's risk for candidiasis. Patient's oral hygiene maintenance and necessary periodontal therapies are done in periodic appointments in the acute and healing stages of NUP. A detailed periodontal examination should be done at each recall session for any patient with a history of NUP. Past severity of disease, plaque control, and case stabilization when determining the frequency of recall visits must be considered as periodontal maintenance programs for HIV patients are individualized. According to Reznik(2006)3, treatment of NUP includes prescription of narrow spectrum antibiotics(metronidazole 500mg, dispensing 14-20 tablets,1 tablet twice daily for 7 to 10 days or clindamycin or amoxicillin) along with pain management and nutritional supplementation or counseling if necessary during the initial visit. Follow up visits include periodontal care like scaling and root planning.

A favorable treatment response is observed when HIV-associated periodontal disease is in the earliest stages. Repeated episodes of the disease is seen in patients who have been treated for NUP, especially with poor oral hygiene levels. NUP can be localized, insidious, and not always related to plaque. After clinical stabilization, recall visits are usually scheduled every 3 months to prevent recurrence.

NECROTIZING ULCERATIVE STOMATITIS - NUS

Necrotizing Ulcerative Stomatitis - NUS is destructive and very painful. There is marked necrosis of oral soft tissue and underlying bone. NUS might occur separately or as continuation of NUP. It is related to low CD4+ immune cells and higher viral load.(GRASSI et al 1988)10.

MANAGEMENT:

Oral antibiotics and antimicrobial mouth rinse. Removal of the affected bone is necessary when osseous necrosis is seen.

References

1. Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS: Michael Glick DMD, Brian C. Muzyka DMD,Leslie M Salkin DDS,Med, Deborah Lurie PhD
2. American Academy of Periodontology,1999;Barnes, et al. 1973, Falker, et al. 1987, Horning and Cohen,1995 Johnson and Engel,1986; Kinane 2001, Maruyama et al,1994 ; Rowland,1999.

3. Reznik DA(2006) Oral Manifestation of HIV Disease Top HIV Med 2005(13)
4. Glick M, Muzyka BC, Lurie D, Salkin LM (1994a). Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS. Oral Surg Oral Med Oral Pathol 77
5. Glick M, Muzyka BC, Salkin LM, Lurie D (1994b) Necrotising ulcerative periodontitis: A marker for immune deterioration and a predictor for the diagnosis of AIDS. J Periodontol 65
6. Brady LJ, Walker C, Oxford GE, Stewart C, Magnusson I, Mc Arthur W (1996). Oral diseases, mycology and periodontal microbiologyof HIV-1- infected women. Oral Microbiol Immunol 11
7. Tsang CS, Samaranayake LP(2001). Predominant cultivable subgingival microbiota of healthy and HIV-infected ethnic Chinese. APMIS 109
8. Zambon JJ, Reynolds HS, Genco RJ,(1990).Studies of the subgingival microflora in patients with acquired immunodeficiency syndrome. J Periodontol 61
9. Murray PA (1994). Periodontal diseases in patients infected by human immunodeficiency virus. Periodontol 2000(6).
10. Murray PA, Grassi M, Winkler JR (1989). The microbiology of HIV-associated periodontal lesions. J Clin periodontal 16
11. Greenspan J, Greenspan D(2008). Oral complications in HIv infection. In: Volberding PA, Sande MA, Lange J, Greene WC eds Global HIV/AIDS medicine. WC. Saunders, Elsevier B.V. Inc. Publishing: Philadelphia, USA.
12. Winkler JR, Robertson PB(1992). Periodontal disease associated with HIV infection. Oral Surg Oral Pathol Oral Radiol Endod 73.
13. EC Clearinghouse(1993). EC Clearinghouse on oral problems related to HIV infection and WHO Collaborating Centre on oral manifestation of immunodeficiency virus classification and diagnostic criteria for oral lesions in HIV infection. J Oral Pathol Med 22
14. Review article: HIV infection and Periodontal disease: An overview of the post-HAART era. M Mataftsi, L skoura, D. Sakellari
15. Feller L, Wood N, lemmer J (2007). Herpes zoster infection as an immune reconstitution inflammatory syndrome in HIV seropositive subjects: a review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 104
16. Carranza's clinical periodontology
All Images courtesy of Google Images

Author Information

Shilpa R Menon, BDS, Clinical Observer
River Oaks Dental
San Jose, CA
drsmenonbds@gmail.com