Anand Induchoodan

Periodontal status in stages of HIV disease – a short-term clinical study

Anand Induchoodan, MDS
Assistant Professor
Department of Periodontics
Amritha School of Dental Science
Edapally, Kerala
India
Tel: +91-484-2801234
Fax: +91-484-2802124
Email: ainduchudan@yahoo.com

KEY WORDS *cell count, gingivitis/epidemiology, HIV infections, periodontal diseases/complications, periodontal diseases/epidemiology, periodontal index*

Aim: The aim of the present study was to investigate the association between clinical periodontal indices and stages of HIV disease as expressed by CD4 cell counts.
Materials and methods: A total of 60 individuals, both males and females, above the age of 18 years who were previously confirmed as HIV-positive were recruited and a detailed medical history was taken. To evaluate periodontal disease, a full-mouth examination was undertaken and, plaque index (PI), gingival index (GI), probing pocket depth and clinical attachment level were recorded. These HIV-positive individuals were classified into three different groups consisting of 20 individuals/group, according to their CD4 count: group A, individuals with CD4 count ≥500 cells/µl (mild immunosuppression); group B, individuals with CD4 count from 200 to 499 cells/µl (moderate immunosuppression); and group C, individuals with CD4 count <200 cells/µl (severe immunosuppression). Associations between the above indices and CD4 counts were examined.
Results: The mean plaque index of group B individuals were higher than the mean plaque index of group C which was statistically significant. The mean gingival index of group A, group B and group C did not show any statistically significant difference. The mean probing pocket depth and mean clinical attachment level in group A and group B individuals were higher when compared with group C individuals, which was statistically significant. However, no statistically significant difference was detected between group A and group B.
Conclusion: The individuals with severe immunosuppression (CD4 cell count <200 cells/µl) showed less periodontal disease progression than expected.

■ Introduction

The human immunodeficiency virus (HIV) is a frequently mutating retrovirus that attacks the human immune system and has shown to cause acquired immunodeficiency syndrome (AIDS). AIDS is a pandemic that significantly affects dental practice, regardless of geographic location. The oral cavity is a frequent site for clinical manifestations of the disease[1]. The ability to recognise and manage oral manifestations of this disease is an important part of dental practice. The clinician should be prepared to assist HIV-infected patients in maintenance of oral health throughout the course of their disease.

Periodontal manifestations in patients with HIV infection were first described in 1987[2]. Four peri-

odontal disease entities were initially associated with HIV infection. They include linear gingival erythema (LGE), necrotising ulcerative gingivitis (NUG), necrotising ulcerative periodontitis (NUP) and necrotising ulcerative stomatitis (NUS). These diseases were considered to be the first clinical expression of HIV-seropositive individuals, or an indicator of severe immune deterioration.

T lymphocytes play a major role in defence against intracellular pathogens such as viruses, protozoa and intracellular bacteria, and are also involved in immunity to extracellular pathogens by providing 'help' for the antibody response (T-helper cells). The primary receptor in humans for HIV is believed to be the characteristic 55 KD protein on the surface of $CD4^+$ lymphocytes, which is responsible for many of the pathological and clinical manifestation of HIV infection[3].

In recent years, the most encouraging news regarding management of periodontal patients with HIV infection comes from new treatment approaches to this disease on a systemic level. The goal of these new approaches, the so-called highly active antiretroviral therapy (HAART), is to reduce the HIV virus to undetectable levels in the bloodstream through the use of a combination of reverse transcriptase inhibitors and protease inhibitors. By controlling the viral activity, reduced T cell counts of the host can rebound leading to immuno-reconstitution.

The aim of the present study was to investigate the association between clinical periodontal indices and the stage of HIV disease, as expressed by CD4 cell counts.

■ Materials and methods

A total of 60 individuals, both males and females, above the age of 18 years were included in this study. Study participants were selected from the South Indian Positive Network (Peravallur, Chennai, Tamil Nadu, India) and from the Community Health Education Society (Kodambakkam, Chennai, Tamil Nadu, India).

■ Study design

This study consisted of individuals who were previously serologically confirmed as HIV-positive. Prior to the examination, the participants were informed regarding the study design and informed consent was gained from each individual. The information gathered from the patients included the subject's age, medical history and medication, and social history of alcohol, tobacco or recreational drug use.

Inclusion criteria:

- patients whose HIV status was confirmed with ELISA (enzyme-linked immunosorbant assay)
- individuals above the age of 18.

Exclusion criteria:

- patients who were unable to tolerate a periodontal examination
- patients whose CD4 count was not updated within the last 4 months
- patients who had undergone recent tooth extraction or any oral surgical procedures within 3 weeks of the study.

■ Stratification of periodontal disease

A single examiner conducted the full-mouth periodontal assessment. The periodontal status of the HIV-seropositive individuals was assessed using the following clinical parameters:

- plaque index[4]
- gingival index[5]
- probing pocket depth[4]
- clinical attachment level[4].

The patients were examined using a type III[6] study design given by the American Dental Association (1970). A type III design consists of examining the patients with a mouth mirror, a Williams graduated periodontal probe and an explorer under proper illumination. The CD4 cell count was extracted from patient's recent medical records. These parameters along with the $CD4^+$ counts were recorded on a previously prepared form for this study. A verbal explanation of examination techniques was done and written consent forms were obtained.

The HIV-seropositive individuals were classified into three different groups consisting of 20 individuals/group, according to their CD4 count.

- Group A: individuals with a CD4 count ≥ 500 cells/µl (mild immunosuppression).

Variable	Group	Mean ± SD	P Value*	Groups at 5% significance level#
	A	1.54 ± 0.24		
PI	B	1.59 ± 0.39		B vs. C
	C	1.34 ± 0.22	0.02	
	A	1.67 ± 0.29		
GI	B	1.77 ± 0.28		
	C	1.60 ± 0.23	0.14	
	A	4.52 ± 0.80		A vs. C
PPD	B	4.38 ± 0.81		B vs. C
	C	3.61 ± 0.50	0.0003	
CAL	A	4.58 ± 0.81		A vs. C
	B	4.46 ± 0.79		B vs. C
	C	3.83 ± 0.63	0.0047	

Table 1 Mean, standard deviation and test of significance of mean values for plaque index score, gingival index score, probing pocket depth and clinical attachment level between different study groups.

One-way ANOVA was used to calculate the *P* value, where $P<0.05$ was considered as the level of statistical significance.
#Multiple comparisons by Tukey's HSD test were employed to identify the significant groups at 5% level.
PI, plaque index; GI, gingival index; PPD, probing pocket depth; CAL, clinical attachment level.

- Group B: individuals with a CD4 count from 200 to 499 cells/µl (moderate immunosuppression).
- Group C: individuals with a CD4 count < 200 cells/µl (severe immunosuppression).

Statistical analysis

Mean and standard deviation were estimated from the sample for each study group. Mean values were compared by one-way ANOVA. Multiple comparisons by Tukey's HSD test were used at 5% level to identify the significant groups. In the present study, $P<0.05$ was considered as the level of statistical significance.

Results

The results of the present study have been summarised in Table 1. The plaque index score was compared between the different groups. The results showed that the mean plaque index score in group B (1.59±0.39) was significantly higher than the mean plaque index score in group C (1.34±0.22). However, no statistically significant differences were detected between group A (1.54±0.24) and group B (1.59±0.39) and also between group A (1.54±0.24) and group C (1.34±0.22). When the gingival index score was compared between different groups, there was no statistically significant difference in mean gingival index score between different study groups.

When the probing pocket depth and the loss of attachment were compared between the three different study groups, the mean probing pocket depth in group A (4.52±0.80) and in group B (4.38±0.81) were statistically significantly higher than the mean pocket depth in group C (3.61±0.50); however, no statistically significant differences were detected between group A (4.52±0.80) and group B (4.38±0.81). Similarly, when the loss of attachment was compared, the mean loss of attachment in group A (4.58±0.81) and in group B (4.46±0.79) were both significantly higher than the loss of attachment in group C (3.83±0.63). However, no statistically significant differences were detected between group A (4.58±0.81) and group B (4.46±0.79).

Discussion

HIV is a retrovirus that, by preferentially attacking and destroying the T4 lymphocyte, causes an immunocompromised state and its associated pathogenic sequelae which is AIDS. In studying these patients, several oral diseases have been related to HIV infection and the development of AIDS. AIDS is the end stage of the natural history of the infection and was first described by Gottelib et al.[7].

Associations between the plaque index and CD4 counts in this study did not correlate with the studies done by Robinson[8] and Lucht et al[9], where the progression of periodontal disease in HIV-seropositive patients showed no difference in plaque retention between controls and subjects. This was consistent with the current understanding that gingival inflammation is plaque related, whereas the rate of attachment loss may or may not be directly linked with the accumulation of plaque. Other host factors may play a major role in the mechanism of tissue destruction[10].

Associations between the gingival index and CD4 counts in the present study correlated well with the studies done by Martinez-Canut et al[11] and Barr et al[12] In the Martinez study, a lack of association was shown between periodontal pathology and CD4 counts. Barr did not find any relationship between inflammation, necrosis and CD4 cell counts.

Associations between the pocket depth and clinical attachment level with CD4 counts of the present study correlated well with the studies done by Vastradis et al[13], Been et al[14,] and Persson and Hollender[15]. Vastradis found that patients with severe immunosuppression had significantly fewer areas of attachment loss and probing depth than patients with mild or moderate immunosuppression. Been et al[14] reported that immunosuppressed individuals, such as those who receive high doses of immunosuppressive drugs after renal transplants, showed lower levels of periodontal disease compared to matched controls. Persson did not demonstrate any difference in alveolar bone loss between an HIV-seropositive group and controls and when the two HIV infected groups were combined to increase statistical power.

■ Conclusion

From the present study, it can be concluded that individuals with severe immunosuppression (CD4 count less than 200cells/µl) have shown less periodontal disease progression than expected. Further studies utilising larger sample sizes are recommended for evaluating the correlation and understanding of the pathogenesis of periodontitis in HIV-infected individuals.

■ References

1. Winkler JR, Murray PA,Greenspan D. AIDS-virus associated periodontal disease. J Dent Res 1986;65(spec issue):741-744
2. Winkler J.R, Murray P.A. Periodontal disease: A potential intraoral expression of AIDS may be rapidly progressive periodontitis. J Calif Dent Assoc 1987;15:20-24.
3. CDC. Current Trends Acquired Immunodeficiency Syndrome (AIDS) Update - -United States. MMWR Weekly 1983;32:309-211.
4. Sillness J, Loe H. Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 1964;2:112-135
5. Loe H, Silness J. Periodontal disease in pregnancy I. Prevalence and severity. Acta Odontol Scand 1963;21: 533-551.
6. Hunt R. Virology Human Immuno Deficiency virus and AIDS. University of South Carolina, 2001.
7. Gottelib MS, Schroff R, Schanker HM. Pneumocystis carinii pneumonia and mucosal candidiasis in previously healthy homosexual men: evidence of a new acquired cellular immunodeficiency. N Engl J Med 1981;305:1425-1431.
8. Robinson P.G. The significance and management of periodontal lesions in HIV infection. Oral Dis 2002;8(suppl 2):91-97.
9. Lucht E, Heimdahl A, Nord CE. Periodontal disease in HIV-infected patients in relation to lymphocyte subsets and specific microorganisms. J Clin Periodontol 1991;18:252-256.
10. Yeung SC, Stewart GJ, Cooper DA, Sindhusake D. Progression of periodontal disease in HIV seropositive patients. J Periodontol 1993;64:651-657.
11. Martínez-Canut P, Guarinos J, Bagán JV. Periodontal disease in HIV seropositive patients and its relation to lymphocyte subsets. J Periodontol 1996;67;33-36.
12. Barr C, Lopez MR, Rua-Dobles A. Periodontal changes by HIV serostatus in a cohort of homosexual and bisexual men. J Clin Periodontol 1992;19:794-801.
13. Vastradis SA, Yukna RA, Fidel PL Jr, Leigh JE, Mercante DE. Periodontal disease in HIV-positive individuals: Association of periodontal indices with stages of HIV disease. J Periodontol 2003;74:1336-1341.
14. Been V, Engel D. The effects of immunosuppressive drugs on periodontal inflammation in human renal allograft patients. J Periodontol 1982;53:245-248
15. Persson RE, Hollender LG, Persson GR. Alveolar bone levels in AIDS and HIV seropositive patients and in control subjects. J Periodontol 1998;69:1056-1061.