

Prevalence of periodontitis in HIV-positive patients on highly active antiretroviral therapy: an observational study

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Abstract

Introduction: The study aimed to evaluate the severity and prevalence of periodontitis in HIV-positive patients on highly active antiretroviral therapy (HAART).

Material and methods: Twenty seven individuals aged between 25 and 55 years, with more than fifteen permanent teeth excluding third molars were included in the study, and periodontal status of patients was assessed.

Results: The average GI was 0.76 ± 0.53 , and the average PI was 0.69 ± 0.36 . The average bleeding index recorded according to Mombelli sulcular bleeding index was 0.46 ± 0.43 , the average normal sulcus/pocket depth was 1.61 ± 0.53 , and the average recession was 0.48 ± 0.42 . The most common therapy administered was tenofovir, lamivudine, and dolutegravir (TLD) 30 in 10 patients (37%), followed by 5 patients on TLD 90 (18.5%), 5 patients on tenofovir, lamivudine + atazanavir/ritonavir (TL + ATV/r) (30 + 30), and 4 patients on TLD 60 (14.8%).

Conclusions: Periodontitis was observed in 26 patients (96.3%) with the absence of periodontitis in 1 of the 27 patients examined (3.7%). The current investigation demonstrates that HIV-positive individuals under HAART present a high prevalence of mild to moderate periodontitis. According to the study's conclusions, tooth loss was consistently affected by the administration of cART. Therefore, treating physicians should routinely recommend patients to oral health professionals to maintain dental health and provide higher quality of life for HIV-infected patients.

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Key words: HIV, HAART therapy, periodontal disease, AIDS.

Introduction

Human immunodeficiency virus (HIV) continues to be a universal health issue, affecting lives of a large population [1]. Even though COVID-19 has garnered much of the world's attention, the HIV/acquired immunodeficiency

syndrome (AIDS) pandemic, which is now in its 5th decade, is far from being controlled regardless of the effectiveness of antiretroviral medications [2]. The first reported case of HIV was documented in 1981, and the virus is being spread primarily through sexual contact or by intravenous needle and

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syringe sharing [3]. Since the early 1990s, significant changes have been made in HIV infection and global AIDS epidemic, while in less developed countries, HIV/AIDS continues to be one of the leading causes of premature death [4].

HIV in its advanced stage leads to AIDS, which results in a decreased host response that makes a person more susceptible to opportunistic infections; it is also associated with the most prevalent type of HIV, i.e., HIV-1 [1]. HIV selectively infects and kills T helper cells, reducing CD4 (cluster of differentiation 4) cell count in an individual [5]. It weakens the acquired immune response of the body, making the individual more susceptible to several oral diseases and lesions [1]. As HIV makes the person immunocompromised, the affected individual becomes more vulnerable to periodontal diseases with chronic inflammations [2], such as necrotizing ulcerative gingivitis, necrotizing ulcerative periodontitis, linear gingival erythema, candidiasis, salivary gland diseases, Kaposi's sarcoma, etc. [3].

Periodontitis and HIV infection are strongly interconnected. Patients with HIV infection who already have periodontitis appear to be at an increased risk of the condition worsening. However, it is still unclear how exactly HIV infection contributes to the development of periodontitis. Patients with HIV are also more likely to develop age-related illnesses. It is unknown if a link exists between periodontitis and HIV infection that raises the risk [6].

With the use of highly active antiretroviral therapy (HAART), quality of life has improved and better survival rate of HIV-infected patients has been observed [1]. Before the development of combination antiretroviral therapy (cART), periodontitis was prevalent in HIV-infected patients with rates ranging from 51% in a British study to 62% in an American research. Following the development of cART, the reported incidence of periodontitis in patients with HIV differed even more [6]. HAART was developed in 1995, and has notably altered the course of HIV. HAART is a combination of 3 or more drugs, including a dual nucleoside analogue reverse transcriptase inhibitor backbone and a third cornerstone drug, such as a non-nucleoside inhibitor or a protease inhibitor [1].

This combination regimen is commonly used to treat patients infected with HIV type 1. Due to the decrease in CD4 cell count, there is a disruption of oral symbiosis, which makes the individual more susceptible to pathologic microbial growth [2]. With the emergence of ART, oral manifestations have reduced considerably [3], as ART decreases the plasma load of the virus to an appreciable range, thus increasing CD4 count [5].

Nowadays, HIV infection has become a chronic illness with a nearly normal life expectancy. There is a lack of data regarding the prevalence and severity of periodontitis among HIV-positive patients in resource-rich environments. Understanding the prevalence of periodontitis, its consequences and modifiable risk factors could have a significant impact on the oral and general health of HIV-positive patients [6]. Studies related to ART and periodontitis in this part of the population are insufficient, so the present

study aimed to fill this gap by evaluating the severity and prevalence of periodontitis in HIV-positive patients on HAART therapy.

The objectives of the study were to evaluate clinically the periodontal conditions of HIV-positive patients, assess the reduced CD4 count, and find a correlation between the severity of periodontal conditions associated with reduced CD4 count and HIV.

Material and methods

The study was conducted in a recognized private medical and dental hospital, and included patients aged above 25 years, with more than fifteen permanent teeth, without the 3rd molars.

Excluded from the study were patients with an inflammatory periodontal condition that required immediate medical attention, those requiring antibiotic prophylaxis before dental procedures, pregnant women, nursing mothers, diabetics, patients with autoimmune diseases and necrotizing periodontal diseases, persons who were recently on antibiotics and/or anti-inflammatory drugs, and those who had recently undergone periodontal therapy. A mouth mirror and William's periodontal probe were utilized to assess the periodontal status of participants.

Statistical analysis

Data were analyzed using statistical software Statistical Package For The Social Sciences (SPSS) version 20.0, with results presented as mean, standard deviation, median, and percentages. An independent *t*-test was employed to compare periodontal findings between two groups of patients.

Results

In total in the study, there were 27 participants (16 males and 11 females) on ART for longer than 3 months. The mean age in years was 47.0 ± 9.25 , with drivers comprising the majority of participants. Fourteen patients had a CD4 count < 500 (51.9%), and 13 subjects had a CD4 count > 500 (48.1%), with a mean of 491.26 ± 246.02 (Figure 1, Table 1).

Adult respondents had an average of 25.44 ± 3.07 teeth, excluding third molars. The average plaque index was 0.69 ± 0.36 , the average gingival index was 0.76 ± 0.53 , and the average bleeding index recorded according to Mombelli sulcular bleeding index was 0.46 ± 0.43 . Moreover, the average normal sulcus/pocket depth was 1.61 ± 0.53 , the average recession was 0.48 ± 0.42 , and the average clinical attachment loss was 0.94 ± 0.82 , which was statistically insignificant (Table 2).

The most common regimen was tenofovir, lamivudine, and dolutegravir (TLD) 30 in 10 patients (37%), followed by 5 patients on TLD 90 (18.5%), 5 patients on tenofovir, lamivudine + atazanavir/ritonavir (TL + ATV/r) 30 + 30 (18.5%), and 4 patients on TLD 60 (14.8%). The least common treat-

ment plans were AL-600 mg, tenofovir disoproxil fumarate + lamivudine (TDF + 3TC), and TL + ATV/r 60 + 60 in one patient each, accounting for 3.7% of the study participants (Table 3, Figure 2).

Mobility of teeth was present in 7 of the 27 patients (25.9%) and absent in 20 patients (74.1%) (Table 4, Figure 3).

The gingival index (GI) (Table 5, Figure 4) was mild in 22 patients (81.5%) and moderate in 5 patients (18.5%).

The plaque index (PI) (Table 6, Figure 5) was found to be good in 18 patients (66.7%) and fair in 9 patients (33.3%).

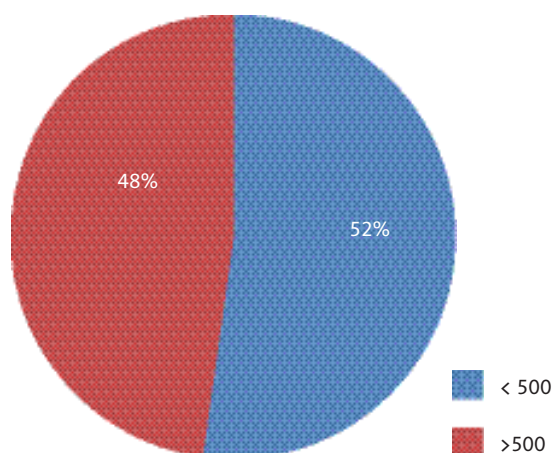


Figure 1. Percentage distribution of CD4 count in HIV-positive patients on highly active antiretroviral therapy

Table 1. Distribution of CD4 count in HIV-positive patients on highly active antiretroviral therapy

CD4 count	n	Valid percentage
< 500	14	51.9
> 500	13	48.1
Total	27	100.0

Of the 27 patients examined, periodontitis (Table 7, Figure 6) was observed in 26 patients (96.3%), whereas one patient was periodontitis-free (3.7%).

Comparing findings among participants with CD4 counts less than 500 and greater than 500 (Table 8, Figures 7-9) with age, revealed that age was higher in the < 500 group with a *t*-value of 0.576 and non-significant *p*-value of 0.57.

Comparison of the number of teeth present excluding third molars between the two groups (i.e., patients with CD4 cell count < 500 and those with CD4 cell count > 500) showed that the number of teeth present excluding third molars was higher in the < 500 group with a *t*-value of 0.096, and was statistically non-significant (*p*-value of 0.924). The average gingival index between the two groups was higher in the > 500 group with a *t*-value of -0.271, and was statistically non-significant (*p*-value of 0.789).

The plaque index between the two groups was higher in the > 500 group with a *t*-value of -0.044, and was statistically non-significant (*p*-value of 0.966). The average bleeding index between the two groups showed it was higher in the > 500 group with a *t*-value of -0.149 and was statistically non-significant (*p*-value of 0.883). The normal sulcus/pocket depth between the two groups was higher in the > 500 group with a *t*-value of -1.397, and was statistically non-significant (*p*-value of 0.175).

Comparison of the average recession between the two groups showed that it was higher in the < 500 group with a *t*-value of 0.289, and was statistically non-significant (*p*-value of 0.775). Also, the clinical attachment loss between the two groups was higher in the < 500 group with a *t*-value of 0.221, and was statistically non-significant (*p*-value of 0.827).

Discussion

HIV as one of the greatest epidemics in our nation, has had a significant negative influence on socio-economic conditions of the population. HIV is a persistent infection that begins asymptotically, progresses to early symptoms

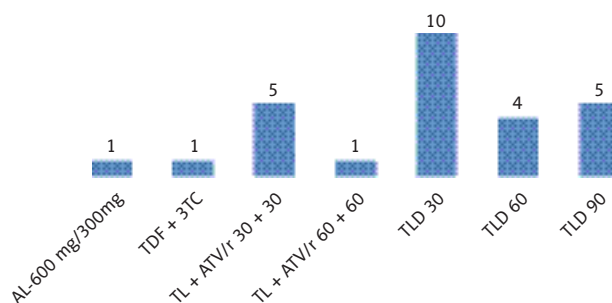
Table 2. Evaluation of periodontal parameters

Parameter	n	Mean $\pm$ SD	Median (IQR)	Range
Age (years)	27	47.0 $\pm$ 9.25	50 (43, 54)	25-55
CD4 count	27	491.26 $\pm$ 246.02	495 (268, 659)	71-923
Number of teeth present (excluding third molars)	27	25.44 $\pm$ 3.07	26 (25, 28)	16-28
Average gingival index	27	0.76 $\pm$ 0.53	0.7 (0.44, 0.99)	0-1.98
Average plaque index	27	0.69 $\pm$ 0.36	0.74 (0.35, 0.96)	0.04-1.39
Average bleeding index	27	0.46 $\pm$ 0.43	0.36 (0.17, 0.67)	0-1.63
Average normal sulcus/pocket depth	27	1.61 $\pm$ 0.53	1.68 (1.23, 1.78)	0-2.82
Average recession	27	0.48 $\pm$ 0.42	0.38 (0.12, 0.73)	0-1.57
Average clinical attachment loss	27	0.94 $\pm$ 0.82	0.65 (0.32, 1.27)	0-2.75

Table 3. Highly active antiretroviral therapy (HAART) regimens of participants

HAART regiments	No. of patients	Valid percent
AL-600 mg	1	3.7
TDF + 3TC	1	3.7
TL + ATV/r 30 + 30	5	18.5
TL + ATV/r 60 + 60	1	3.7
TLD 30	10	37.0
TLD 60	4	14.8
TLD 90	5	18.5
Total	27	100.0

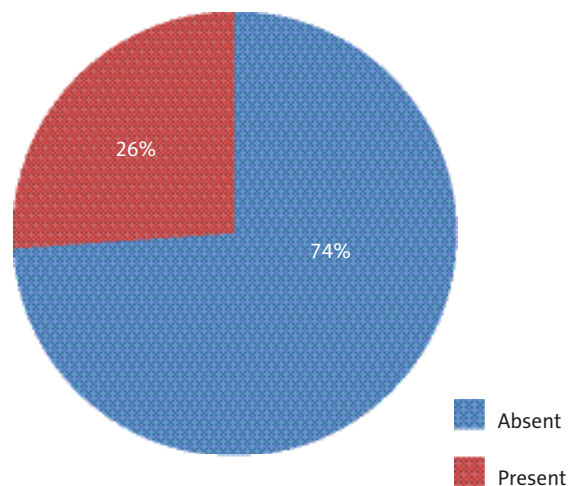
TDF + 3TC – tenofovir disoproxil fumarate + lamivudine, TL + ATV/r – tenofovir, lamivudine + atazanavir/ritonavir, TLD – tenofovir, lamivudine, dolutegravir.



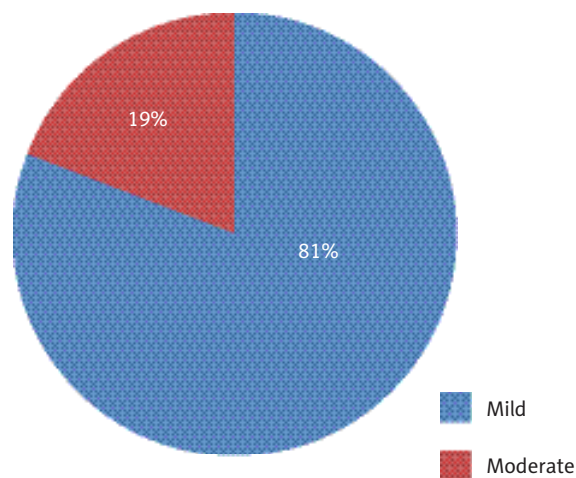
TDF + 3TC – tenofovir disoproxil fumarate + lamivudine, TL + ATV/r – tenofovir, lamivudine + atazanavir/ritonavir, TLD – tenofovir, lamivudine, dolutegravir.

Figure 2. Data distribution of highly active antiretroviral therapy regimens of patients**Table 4.** Presence and absence of mobility of teeth in participants

Absence	Mobility	Valid percent
Absent	20	74.1
Present	7	25.9
Total	27	100.0

**Figure 3.** Percentage distribution of presence and absence of mobility**Table 5.** Full-mouth gingival index (GI) of participants

GI categories	n	Valid percent
Mild	22	81.5
Moderate	5	18.5
Total	27	100.0

**Figure 4.** Percentage distribution of full-mouth gingival index (GI)

resulting in immunosuppression, and eventually leads to AIDS.

The virus drastically reduces a person's immunity by focusing on T helper cells. A microbiota imbalance in the oral cavity causes a variety of opportunistic infections, leading to the development of oral lesions in addition to systemic illnesses. The individual's quality of life, nutrition, and psycho-social behavior are all negatively impacted by declining dental health, which also negatively influence treatment of many co-existing systemic diseases [7].

HAART treatment plan, also known as cART and anti-retroviral therapy (ART), consists of three or more antiviral medication combinations. The basis of HAART is the administration of various drugs that inhibit the reproduction of viral particles through different mechanisms. This approach has the advantage of using different pharmacological agents to prevent treatment resistance [8].

It is still debatable how common periodontal disease is among people living with HIV infection [7]. Due to several

Table 6. Full-mouth plaque index (PI) of participants

PI category	n	Valid percent
Good	18	66.7
Fair	9	33.3
Total	27	100.0

Table 7. Prevalence of periodontitis among participants

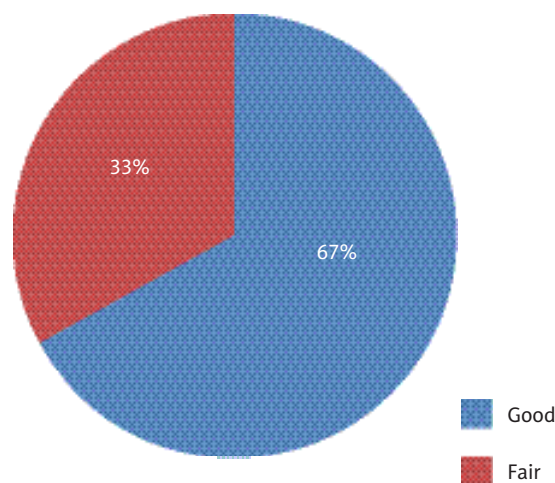
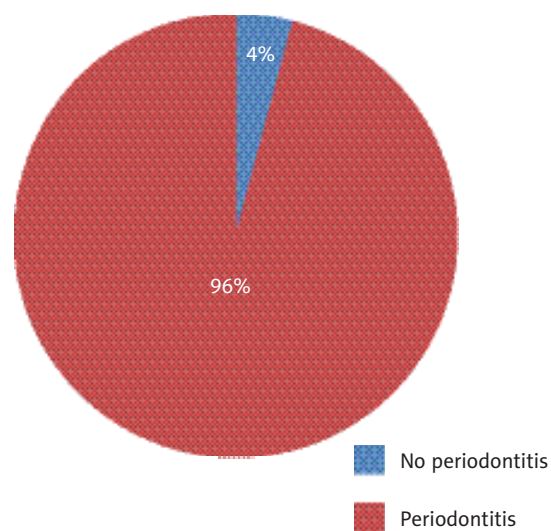
Periodontitis	n	Valid percent
No periodontitis	1	3.7
Periodontitis	26	96.3
Total	27	100.0

variable factors, including the stage of AIDS and cART regimen, data from relevant research vary greatly. Most studies do not usually consider other significant aspects, such as age, smoking habits, immune system capacity, and levels of oral hygiene [9]. In the current study, the prevalence of periodontal disease was 96%, which was higher compared with studies by Brady *et al.* [10] and Alpagot *et al.* [11], who found prevalence rates of 52% and 73%, respectively. The study by Ranganathan *et al.* [12] in India showed a prevalence rate of 33%, while Akmatov *et al.* [13] found a 30% prevalence rate in Germany.

Most of the patients in this study were in their fourth decade of life, which is consistent with the findings of Bravo *et al.* [14]. The conventional belief that severe periodontitis is progressively more common as people get older is in direct opposition to this. Tooth loss may occur because of severe periodontitis as people age, concealing a rise in the prevalence of severe periodontitis [15]. Furthermore, the present study revealed a male predominance, supporting the findings of Ranganathan *et al.* [12] and Bodhade *et al.* [16], who both demonstrated a male preponderance in South India and other regions of the world. However, reports suggest that women outnumber men in Africa [17].

Compared with HIV-positive women, HIV-infected men tend to be more prone to developing severe periodontitis. This outcome is in line with a previous study conducted among Brazilian HIV-positive patients, where periodontitis was found to be frequent in 30% of men and 20% of women [18]. Our study found that gingivitis was more common than periodontitis, which is consistent with the research by Masouredis *et al.* [19], who postulated that poor dental hygiene might be the cause.

The categorization of periodontal diseases has undergone substantial changes, resulting in repeated adjustments and updates [20]. The variation in periodontitis clinical symptoms and disease progression from the 1999 to the 2017 classifications proved challenging and exacerbated the expansion of collaborative research both before and after the introduction of HAART. Findings from relevant research

**Figure 5.** Percentage distribution of full-mouth plaque index (PI)**Figure 6.** Percentage distribution of periodontitis prevalence

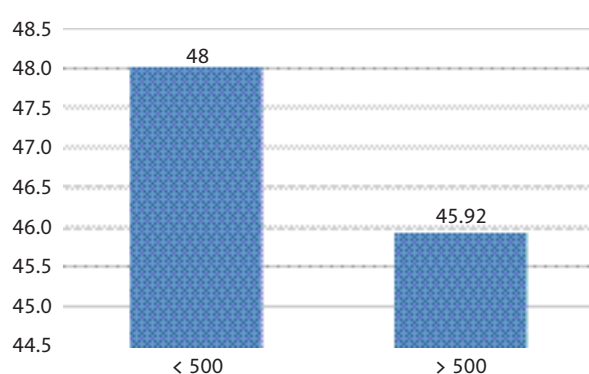
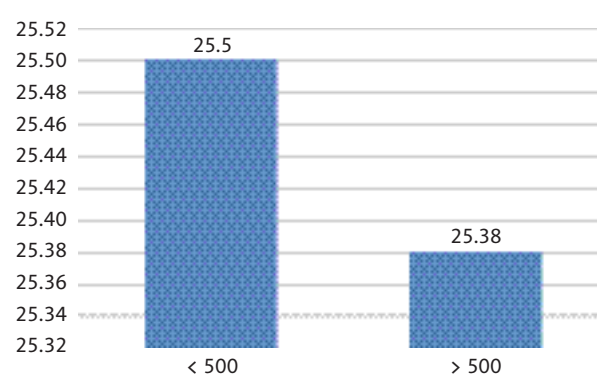
also differ, and cannot be compared due to the diverse modalities of therapy of participating patients [21].

The pattern of HIV-1 infection has changed from a deadly sickness to a manageable chronic illness following HAART therapy. Consequently, the connection between HIV-1 infection and the symptoms of oral diseases, particularly periodontal disease, has changed [22]. As previously supported by Fricke *et al.*, the ART regimen did not specifically modify the occurrence of periodontitis. Additionally, the authors discovered a connection between periodontitis and the time of infection [23].

Patients with a CD4 count of less than 500 demonstrated clinical attachment loss, while patients with CD4 counts below 200 had a greater clinical attachment loss, with a *t*-value of 0.221 and a *p*-value of 0.827 indicating

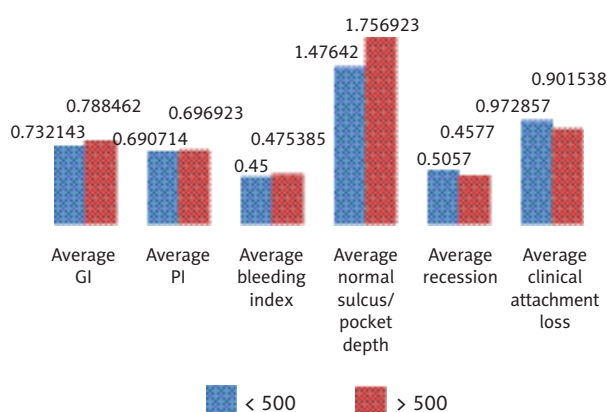
Table 8. Independent *t*-test comparing CD4 count groups

Parameter	< 500 (<i>n</i> = 14) Mean ± SD	> 500 (<i>n</i> = 13) Mean ± SD	<i>t</i> -value	<i>p</i> -value
Age in years	48.00 ± 9.86	45.92 ± 8.80	0.576	0.570
Number of teeth present, excluding third molars	25.50 ± 3.44	25.38 ± 2.76	0.096	0.924
Average gingival index	0.73 ± 0.59	0.79 ± 0.47	−0.271	0.789
Average plaque index	0.69 ± 0.42	0.70 ± 0.31	−0.044	0.966
Average bleeding index	0.45 ± 0.50	0.48 ± 0.37	−0.149	0.883
Average normal sulcus/pocket depth	1.48 ± 0.63	1.76 ± 0.36	−1.397	0.175
Average recession	0.51 ± 0.40	0.46 ± 0.46	0.289	0.775
Average clinical attachment loss	0.97 ± 0.80	0.90 ± 0.88	0.221	0.827

**Figure 7.** Frequency distribution of participants' age**Figure 8.** Frequency distribution of the number of teeth present

statistical insignificance. Our findings are in line with the study by Umezudike *et al.* [24], who also found no significant correlation ($p = 0.623$). This is further comparable to the findings of Vernon *et al.* [25], who discovered that patients with CD4 counts under 200 had a two-fold increased chance of developing periodontitis compared with people with CD4 cells over 200 ($p = 0.001$). This consistent evidence suggests that, in the group of people living with HIV, having CD4 cells below 200 is a strong predilection for periodontitis; in our study, six patients had CD4 counts below 200. This finding contrasts with a previous investigation that found no correlation between CD4 cells below 200/mm³ and clinical periodontal markers [25].

Antiretroviral therapy has altered the trajectory of HIV disease and enhanced the general well-being of HIV-infected individuals. There are conflicting findings regarding the link between CD4 levels and periodontal disease in HIV-positive patients, with some research reporting severe damage of periodontal tissues and others showing no difference between HIV-positive and healthy people. According to several studies, HIV-positive individuals with high immunosuppression had lesser periodontal damage than HIV-positives with mild immunosuppression. Additional systematic research is needed before periodontal disease may be considered a significant HIV consequence.

**Figure 9.** Frequency distribution of average gingival index (GI), plaque index (PI), normal sulcus/pocket depth, recession, and clinical attachment loss

The limitations of the current study are the sample size and lack of follow-up to determine the progression of periodontal disease. Examining the relationship between CD4 count and diverse oral symptoms could be the subject of future research.

Conclusions

Periodontitis, also called gum disease, is a serious gum infection that damages the soft tissue around teeth, and is caused by a particular pathogen or collection of particular micro-organism through the development of periodontal pockets, gingival recession, or both. HIV infection and periodontitis are correlated. Periodontitis is more common in people with HIV infection and older male patients, who tend to suffer from more severe forms of the condition. The dental health and general well-being of HIV-infected people may be considerably improved if patients and healthcare professionals are made aware of the rising frequency of periodontitis linked to HIV infection.

The invention of HAART, at least in the developed world, substantially changed the trajectory of HIV disease, converting it from deadly into serious illness that can be treated. HIV-infected individuals have now an extended survival time and better quality of life. Therefore, the treating physician should routinely recommend patients to oral health professionals to maintain oral health and high quality of life of HIV patients. According to the study's findings, the administration of HAART consistently affect periodontal diseases and tooth loss. Even if the study's goals are met, more research with larger sample size and long-term follow-up are required.

Disclosures

1. Institutional review board statement: The study was carried out according to the protocol approved by the Institutional Ethics Committee on October 17, 2022 (approval number: MCODS/22065/2022). Written informed consent was obtained from each participant before their enrollment in the study.
2. Assistance with the article: This research was conducted in the Kasturba Medical College and Manipal College of Dental Sciences, Mangalore, Manipal Academy of Higher Education, Manipal, Karnataka, India. The authors would like to thank the participants who generously shared their time and information for this research.
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4. Conflicts of interest: None.

References

1. Mataftsi M, Skoura L, Sakellari D. HIV infection and periodontal diseases: an overview of the post-HAART era. *Oral Dis* 2011; 17: 13-25.
2. Ravi JR, Rao TR. Estimation of prevalence of periodontal disease and oral lesions and their relation to CD4 counts in HIV seropositive patients on antiretroviral therapy regimen reporting at District General Hospital, Raichur. *J Indian Soc Periodontol* 2015; 19: 435-439.
3. Fokam J, Geh BKN, Sosso SM, Takou D, Ngufack ES, Nka AD, et al. Determinants of periodontitis according to the immunological and virological profiles of HIV-infected patients in Yaoundé, Cameroon. *BMC Oral Health* 2020; 20: 359. DOI: 10.1186/s12903-020-01353-7.
4. Ryder MI, Nittayananta W, Coogan M, Greenspan D, Greenspan JS. Periodontal disease in HIV/AIDS. *Periodontol* 2000 2012; 60: 78-97.
5. Gonçalves LS, de Carvalho Ferreira D, Vidal F, Souza RC, Gonçalves C, Pavan P, et al. Stage II and stage III periodontitis clinical burdens of HIV-1 undergoing antiretroviral therapy. *Clin Oral Investig* 2022; 26: 2187-2195. Erratum: *Clin Oral Investig* 2022; 26: 4239.
6. Groenewegen H, Bierman WFW, Delli K, Dijkstra PU, Nesse W, Vissink A, et al. Severe periodontitis is more common in HIV-infected patients. *J Infect* 2019; 78: 171-177.
7. Lauritano D, Moreo G, Oberti L, Lucchese A, Di Stasio D, Conese M, et al. Oral manifestations in HIV-positive children: a systematic review. *Pathogens* 2020; 9: 88. DOI: 10.3390/pathogens9020088.
8. Eggleton JS, Nagalli S. Highly Active Antiretroviral Therapy (HAART). In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2022.
9. Coogan MM, Greenspan J, Challacombe SJ. Oral lesions in infection with human immunodeficiency virus. *Bull World Health Organ* 2005; 83: 700-706.
10. Brady LJ, Walker C, Oxford GE, Stewart C, Magnusson I, McArthur W. Oral diseases, mycology and periodontal microbiology of HIV-1-infected women. *Oral Microbiol Immunol* 1996; 11: 371-380.
11. Alpagot T, Duzgunes N, Wolff LF, Lee A. Risk factors for periodontitis in HIV patients. *J Periodontol Res* 2004; 39: 149-157.
12. Ranganathan K, Umadevi M, Saraswathi TR, Kumarasamy N, Solomon S, Johnson N. Oral lesions and conditions associated with human immunodeficiency virus infection in 1000 South Indian patients. *Ann Acad Med Singap* 2004; 33 (4 Suppl): 37-42.
13. Akmatov MK, Hu E, Rüsenberg R, Kollan C, Schmidt D, Kohring C, et al. Areas with high HIV prevalence: A spatial analysis of nationwide claims data in Germany. *HIV Med* 2024; 25: 498-503.
14. Bravo IM, Correnti M, Escalona L, Perrone M, Brito A, Tovar V, et al. Prevalence of oral lesions in HIV patients related to CD4 cell count and viral load in a Venezuelan population. *Med Oral Patol Oral Cir Bucal* 2006; 11: E33-E39.
15. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ; CDC Periodontal Disease Surveillance workgroup. Prevalence of periodontitis in adults in the United States: 2009 and 2010. *J Dent Res* 2012; 91: 914-920.
16. Bodhade AS, Ganvir SM, Hazarey VK. Oral manifestations of HIV infection and their correlation with CD4 count. *J Oral Sci* 2011; 53: 203-211.
17. Schätzle M, Löe H, Lang NP, Bürgin W, Anerud A, Boysen H. The clinical course of chronic periodontitis. *J Clin Periodontol* 2004; 31: 1122-1127.
18. Tukutuku K, Muyembe-Tamfum L, Kayembe K, Odio W, Kandi K, Ntumba M. Oral manifestations of AIDS in a heterosexual population in a Zaire hospital. *J Oral Pathol Med* 1990; 19: 232-234.
19. Masouredis CM, Katz MH, Greenspan D, Herrera C, Hollander H, Greenspan JS, et al. Prevalence of HIV-associated periodontitis and gingivitis in HIV-infected patients attending an AIDS clinic. *J Acquir Immune Defic Syndr* (1988) 1992; 5: 479-483.
20. Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions – introduction and key changes from the 1999 classification. *J Clin Periodontol* 2018; 45 Suppl 20: S1-S8. DOI: 10.1111/jcpe.12935.
21. Pólvara TLS, Nobre ÁVV, Tirapelli C, Taba M Jr, de Macedo LD, Santana RC et al. Relationship between human immunodeficiency

- virus (HIV-1) infection and chronic periodontitis. *Expert Rev Clin Immunol* 2018; 14: 315-327.
22. Highfield J. Diagnosis and classification of periodontal disease. *Aust Dent J* 2009; 54 Suppl 1: S11-S26. DOI: 10.1111/j.1834-7819.2009.01140.x.
 23. Fricke U, Geurtsen W, Staufienbiel I, Rahman A. Periodontal status of HIV-infected patients undergoing antiretroviral therapy compared to HIV-therapy naive patients: a case control study. *Eur J Med Res* 2012; 17: 2. DOI: 10.1186/2047-783X-17-2.
 24. Umezudike KA, Ayanbadejo PO, Savage KO, Nwhator SO, Akanmu AS, Ogunleye O. Comparative periodontal status of human immunodeficiency virus-positive patients and controls in a dedicated human immunodeficiency virus clinic in Nigeria. *Niger J Clin Pract* 2016; 19: 35-40.
 25. Vernon LT, Demko CA, Whalen CC, Lederman MM, Toossi Z, Wu M, et al. Characterizing traditionally defined periodontal disease in HIV+ adults. *Community Dent Oral Epidemiol* 2009; 37: 427-437. Erratum: *Community Dent Oral Epidemiol* 2010; 38: 96, 190.