

# Evaluation of Periodontal Status in HIV Infected Persons in Croatia

Ana Stojković<sup>1</sup>, Vanja Vučićević Boras<sup>2</sup>, Darije Plančak<sup>1</sup>, Miroslav Lisić<sup>3</sup> and Srećko Srdjak<sup>1</sup>

<sup>1</sup> University of Zagreb, School of Dental Medicine, Department of Periodontology, Zagreb, Croatia

<sup>2</sup> University of Zagreb, School of Dental Medicine, Department of Oral Medicine, Zagreb, Croatia

<sup>3</sup> University of Zagreb, »Dr. Fran Mihaljević« University Hospital for Infectious Diseases, Zagreb, Croatia

## ABSTRACT

*A number of periodontal changes have been associated with human immunodeficiency virus (HIV) infection, however our knowledge of the epidemiology, microbiology, host response and natural history of these conditions remains limited. Therefore, the aim of our study was the assessment of possible differences in periodontal status of HIV infected subjects when compared with healthy controls matched for age, gender and smoking habit in Croatian population. Assessment included measurement of plaque accumulation using approximal plaque index, measurement of gingival inflammation by use of sulcus bleeding index, pocket depth, gingival recession as well as the number of decayed, missing and filled teeth in 25 HIV infected subjects (age range 22–61, X=40.8 years) in comparison with 25 healthy controls (age range 20–62, X=40.9 years). Statistical analysis was performed by use of descriptive statistics and Mann-Whitney U test showed significantly increased level of inflammation of the marginal gingiva in HIV infected subjects when compared to the controls ( $p < 0.002$ ). Significantly increased mean values of periodontal pockets ( $p < 0.002$ ) and the deepest periodontal pocket ( $p < 0.003$ ) were also observed when HIV infected subjects were compared to the healthy controls. In HIV infected subjects there was significant increase in the number of decayed, missing and decrease in the number of filled teeth ( $p < 0.002$ ;  $p < 0.002$ ;  $p < 0.009$ , respectively). The results of this study once again highlight the need for more prevalent periodontal check-ups and treatments in HIV infected subjects.*

**Key words:** HIV, periodontal status, recession, gingiva, Croatia

## Introduction

It is well known that periodontal disease has an infectious component besides genetic factors and the host immune system<sup>1</sup>. In immunocompromised patients, such as HIV/AIDS patients, a complex cascade of periodontal damage is seen, mainly as a result of T-cell destruction<sup>2</sup>. The recognition of unusual forms of periodontal disease associated with HIV infection has been of special interest since their original description in 1987<sup>2</sup>. Periodontal diseases seen in HIV infected persons are the same ones seen in persons who are HIV negative, although in the HIV infected persons their progression might be different together with their modified appearance. Chronic periodontitis which appears during HIV infection may progress more aggressively with intensified attachment loss as a result of specific immunology status<sup>3</sup>. Microbiological investigations of periodontal disease in HIV infected persons showed prevalence of classical periodontal

disease pathogens otherwise seen in periodontal pockets of persons not infected with HIV<sup>4</sup>. Klimiuk<sup>5</sup> reported connection between HIV infection and worsening of periodontal status which was measured by Gingival Index (GI) and Papilla Bleeding Index (PBI). Another Polish study<sup>6</sup>, published in 2006, showed worsening of the periodontal status in HIV-infected patients as the duration of HIV infection extends, as well as the fact that the health status deterioration, measured by a decrease in the absolute number of CD4 lymphocytes, is accompanied by more intensive pathological periodontal changes. Ranganathan<sup>7</sup> conducted a study using Community Periodontal Index of Treatment Needs (CPITN) and reported that periodontal disease was strongly associated with CD4 cell immunosuppression and oral candidiasis. The same report<sup>7</sup> also suggested that the periodontal status could be a useful tool being a minimal invasive proce-

dure during screening of population regarding HIV disease in countries with high HIV prevalence and resource deficits. A few studies have reported that prevalence of HIV-associated periodontal disease was approximately 8.5%, although the reported range varies from less than 5% to almost 70%, thus probably reflecting unequal periodontal disease recording techniques<sup>8</sup>. On the other hand, a few reports from the USA<sup>9,10</sup> point out that the periodontal disease usually seen in homosexuals, minorities and drug users might be a result of the lifestyle, mode of HIV acquisition and limited access to dental care. The third reason for currently unclear connection between periodontal disease and HIV seropositivity might be the fact that periodontal disease is a rather infrequent finding in HIV diseased. Alves et al.<sup>11</sup> reported that CD4 count and viral load had no consistent effects on probing depth and attachment loss values in HIV infected subjects. The relationship between periodontal disease and immunosuppression in HIV infection is still not fully understood. A few literature reports<sup>12–14</sup> could not confirm a connection between periodontal disease and HIV infection, but more recent studies<sup>15,16</sup> reported increased risk for periodontal attachment loss in HIV positive patients as well as deeper probing pocket depths in HIV diseased patients.

Consequently the objective of this study was to assess possible differences in periodontal status of HIV infected subjects when compared to healthy controls matched for age, gender and smoking habit in Croatian population.

## Materials and Methods

Prior to this investigation an informed consent, in accordance with the Declaration of Helsinki II was obtained from participants. The study was approved by the Ethics committee of the School of Dentistry, University of Zagreb, Croatia.

The HIV infected group was recruited from the »Dr. Fran Mihaljević« University Hospital for Infectious Diseases in Zagreb, where HIV infected subjects routinely seek and receive proper medical care. All HIV infected subjects had serum antibodies to HIV determined by an ELISA (Biotest AG, Germany) and confirmed by Western blot test (INNO-Lia, INNO Genetics). HIV-1 RNA was determined with Cobas Amplicor HIV-1 MONITOR 1.5 test version with detection limit of 20–50 copies/mm<sup>3</sup> of plasma. The HIV infected group consisted of 6 women and 19 men, age range 22–61, X=40.8 years.

In the HIV infected group there were 15 smokers and 10 non-smokers. A single dentist examiner conducted periodontal assessments and measures. Each tooth was measured at four sites in order to assess pocket depth and at two sites for recession (vestibular and lingual/palatal). Periodontal probe with 3 mm increments, a mouth mirror, chair and room light were used. Presence of dental plaque was assessed with dental probe and presented with aproximal plaque index (API). API is ratio of number of plaque positive sites and total number of tested sites. Gingivitis examination was performed using sulcus

bleeding index (SBI), which records bleeding on probing as an early symptom of inflammation at an otherwise healthy gingival site<sup>17</sup>. The number of decayed, missing and filled teeth (DMFT) was recorded according to the World Health Organization (WHO)<sup>18</sup>. The control group consisted of 25 volunteers: 7 women and 18 men, age range 20–62, X=40.92 years, who were all healthy and did not take any medicines one month prior to this investigation. In the control group there were 10 smokers and 15 non-smokers.

Statistical analysis was performed by use of descriptive statistics, Spearman's correlation, Mann-Whitney U test and values lower than 0.05 were considered as statistically significant.

## Results

In the group of HIV infected persons CD4 counts ranged from 1–724 cells/mm<sup>3</sup>, X=164.88 cells/mm<sup>3</sup>. Viral load ranged from below 20 to 1,650,000 HIV copies/mm<sup>3</sup>. The mode of HIV transmission was homosexual route in 14 subjects, heterosexual route in 10 subjects, whereas 1 subject was an intravenous drug user. Four HIV infected subjects were in Center for disease control, USA (CDC) stage A2, 4 subjects in B2, 5 subjects in B3, 2 subjects in C2 and 10 subjects in C3. Additionally, an examination of the oral tissues was performed and the following observations were made: One subject had the CD4 count of 237 cell/mm<sup>3</sup> and non-Hodgkin lymphoma on the gingiva in the molar region, hairy leukoplakia and gingival hyperplasia. Oral candidiasis was recorded in 6 subjects with CD4 counts being 1, 4, 17, 38, 155, 161 cells/mm<sup>3</sup>, respectively. One subject, with the CD4 count of 13 cells/mm<sup>3</sup>, had a herpes virus infection on the lip, candidiasis and leukoplakia. One had candidiasis and angular cheilitis, with CD4 count being 31 cells/mm<sup>3</sup>. The other one had linear gingival erythema, with CD4 count of 150 cells/mm<sup>3</sup>. Another subject had a periodontal abscess, candidiasis and leukoplakia and the CD4 count of 46 cells/mm<sup>3</sup>.

Mann-Whitney test shows significantly higher API in HIV infected subjects when compared to the control group ( $p < 0.001$ ). Dental plaque expressed as API was 94.2% in the male HIV infected subjects and 86.7% in the HIV infected females. In the male and female subjects in the control group dental plaque was present on 45.7% and 42.6% of the tooth arch, respectively, a finding which was significantly decreased when compared with HIV infected subjects of both genders.

Our results also show significant difference between HIV infected subjects and participants in the control group ( $p < 0.002$ ) with regard to the inflammation of the marginal gingiva, being significantly more inflamed in HIV infected persons. Significantly increased mean values of periodontal pockets ( $p < 0.002$ ) as well as the depth of the deepest periodontal pocket ( $p < 0.003$ ) were found in HIV infected subjects when compared to the healthy controls. No significant differences could be found between the two tested groups when gingival recession

(mean; maximal on one tooth) was measured ( $p < 0.077$ ;  $p < 0.541$ ).

Clinical attachment loss (CAL) was calculated as a sum of recession and periodontal pocket depth measured always on the same tooth position. Mean values of CAL calculated for HIV infected subjects were 2.6 mm and were significantly higher when compared to the 2.1 mm mean values of CAL calculated for the control group.

Significantly more decayed and missing teeth as well as significantly less filled teeth were found in HIV positive subjects in comparison to the controls ( $p < 0.002$ ;  $p < 0.009$ ) (Table 1).

Smoking habit also correlated significantly with gingival inflammation, however smokers had lower mean values of gingival inflammation when compared to the non-smokers (Figure 1). Correlation between smoking habit and pocket depth could not be established (Figure 2). No significant difference regarding any of the tested variables between genders could be noticed. The presence of oral manifestations and mode of HIV transmission did not correlate with any of the tested variables.

### Discussion and Conclusion

There is a growing agreement that periodontal diseases commonly associated with HIV disease are similar or modified presentations of periodontal disease seen in non-infected population<sup>19</sup>. Robinson<sup>15</sup> found that periodontal attachment destruction was connected with progressive HIV infection, but that the pocketing was not.

Swango et al.<sup>20</sup> reported that periodontal inflammation and destruction were prevalent among HIV infected patients, but the severe form of HIV-related periodontal destruction reported by others was rarely seen in his patients. The same author stated that gingival banding occurred in 50% of the HIV infected participants independently of the stage of immune deficiency and that gingival

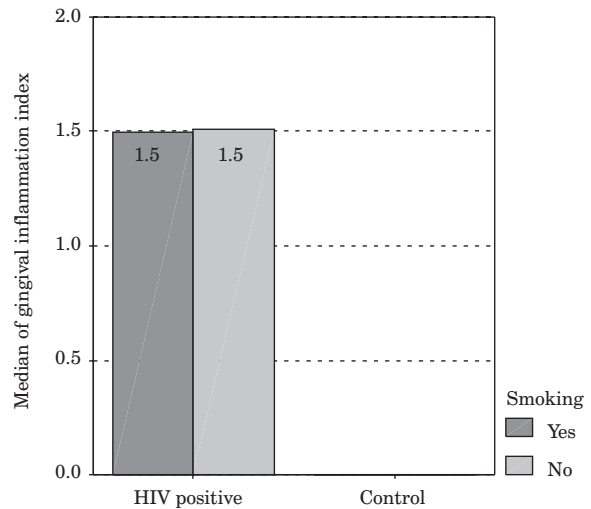


Fig. 1. Median gingival inflammation values between HIV infected subjects and controls with regard to the smoking habit.

bleeding and papillary destruction were less extensive in subjects with more advanced stages of HIV infection. Twenty five percent of these subjects had evidence of ulceration or cratering of one or more papillae, but cases of rapid and severe periodontal destruction were rare. Also, gingival bleeding and papillary destruction occurred in all stages of infection, but somewhat less extensive in subjects with lower CD4 and later stages of HIV infection. Swango et al.<sup>20</sup> found that more bleeding sites and higher gingival indices were significantly associated with CD4 count  $\geq 400$ , compared to those with CD4 count  $< 400$  in HIV infected persons. They also stated that the relation between periodontal health and the degree of immune dysfunction, as measured by T4 lymphocyte counts, remained unclear. Glick et al.<sup>21</sup> reported prevalence of necrotizing ulcerative periodontitis in 6.3% of the total number of 454 HIV infected subjects, as well as

TABLE 1  
COMPARISON OF APROXIMAL PLAQUE INDEX, GINGIVAL INFLAMMATION, MEAN AND MAXIMAL POCKET DEPTH, MEAN AND MAXIMAL GINGIVAL RESSION, DECAYED, MISSING AND FILLED TEETH BETWEEN HIV INFECTED SUBJECTS AND CONTROLS

Variable	HIV group	Control group	P
	Median (Interquartile range)	Median (Interquartile range)	
API (aproximal plaque index)	100 (100–100)	0 (18.8–100)	$< 0.001^*$
Gingival inflammation	1.5 (0.3–2.1)	0.0 (0.0–0.6)	0.002*
Pocket depth-mean	2.2 (1.8–2.5)	1.4 (1.7–2.2)	0.002*
Pocket depth-maximal	4.0 (3.0–4.0)	3.0 (3.0–3.0)	0.003*
Gingival recession-mean	0.6 (0.3–0.9)	0.1 (0.2–0.7)	0.077
Gingival recession-maximal	2.0 (2.0–3.0)	1.0 (3.0–4.0)	0.541
Decayed teeth	1.0 (0.0–2.5)	0.0 (0.0–0.0)	0.002*
Missing teeth	10.0 (4.0–14.0)	1.0 (3.0–7.0)	0.002*
Filled teeth	7.0 (3.0–10.0)	8.0 (10.0–11.0)	0.009*
DMF (decayed, missing and filled teeth)	18.0 (14.0–22.0)	11.0 (15.0–19.0)	0.094

\*Statistically significant

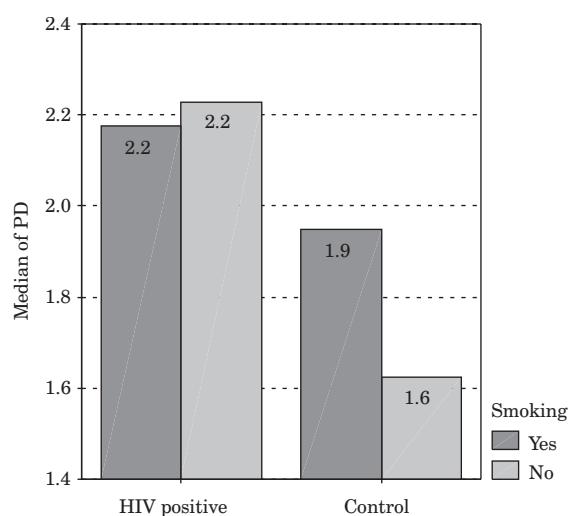


Fig. 2. Mean pocket depth values between HIV infected subjects and controls regarding smoking habit.

a strong connection between a low CD4 count and progressive periodontal disease. It is interesting to note that we have found significantly more gingival inflammation and pocketing in HIV infected subjects when compared to the healthy controls, but there was no correlation to the CD4 count. We have not seen progressive periodontal disease or necrotizing ulcerative disease of either gingival or periodontal tissues in any of our patients. Barr et al.<sup>22</sup> reported no association between the gingival index and the level of CD4 counts, but relative attachment loss of  $\geq 3$  mm was 4.8 times more prevalent when CD4 counts were  $< 400$ , and 6 times more likely when CD4 counts were  $< 200$ .

Matee et al.<sup>23</sup> did not find gingivitis and periodontal disease in 192 HIV infected subjects in Tanzania. Among 107 HIV subjects Hodgson<sup>24</sup> found only one case of necrotising ulcerative parodontitis, 2.8% of cases of necrotising ulcerative gingivitis and no cases of linear gingival erythema. On the other hand, it is interesting to point out that Laskaris et al.<sup>25</sup> found necrotizing gingivitis as the only and first sign of HIV infection in only four out of 178 HIV infected subjects.

Clinical signs of inflammation are less pronounced in smokers when compared to the non-smokers<sup>26,27</sup>. Decreased blood flow and decreased clinical signs of inflammation seems to be a consequence of smoking which induces gingival microvascular alteration<sup>28</sup>. These findings are in accordance with our results, showing inverse correlation of smoking habit with gingival inflammation.

Significant difference between HIV infected subjects and subjects in control group with regard to the decayed,

missing and filled teeth was also established. HIV infected subjects have more decayed and missing and less filled teeth than the control group subjects. Together with wider dental plaque range in HIV infected subjects showed by API, these results serve as a marker of inadequate dental hygiene in HIV infected subjects group. This also may implicate that HIV infected subjects seek dental help in emergency cases when extraction is the only solution or it may unfortunately be a sign of reluctant behavior of dentists towards HIV infected patients.

The results of this study are in concordance with previously published data, which show that a decreased prevalence of severe, necrotic periodontal lesions in HIV/AIDS patients is probably the result of HAART therapy, as suggested by Lamster et al.<sup>29</sup>.

Results of this investigation reveal worse periodontal status of HIV infected subjects when compared to the control group participants, which can be consequence of inadequate oral hygiene. Therefore the influence of HIV virus itself on periodontal tissues could not be established in this investigation.

So far, this is the first report on the periodontal status in HIV infected subjects in Croatia. Croatia is considered to be a country with a low prevalence of HIV infection. Since 1984 when the first case was reported, 470 persons were registered as HIV infected, 218 of who developed AIDS and 122 died<sup>30</sup>. Survival following the first AIDS defining illness was significantly improved in period between 1997 and 2000 when combined antiretroviral therapy was introduced in Croatia compared to period between 1986–1996<sup>31</sup>. As HAART prolongs the life expectancy of HIV infected individuals significantly, periodontal health should also be considered as an important factor thus adding to the improvement of life quality as well.

More dental plaque in HIV infected subjects showed that there is a need for improvement in education regarding oral hygiene measures. Since there is no effective HIV vaccine we still have to rely on education, behavioral changes, antiretroviral therapy and other preventive measures to combat HIV/AIDS<sup>32</sup>. Our findings recommend daily personal oral hygiene procedures together with professional dental check-ups in order to prevent oral and periodontal diseases in HIV infected patients, regardless of the level of their immunosuppression.

## Acknowledgements

Special appreciation is extended to Dr. Lajos Szivovica for statistical analysis and to Prof. Stela Vazdar for proof reading.

## REFERENCES

1. PRESHAW PM, SEYMOUR RA, HEASMAN PA, *Dent Update*, 31 (2004) 570. — 2. WINKLER JR, MURRAY PA, *Calif Dent Assoc*, 15 (1987) 20. — 3. ROBINSON PG, ADEGBOYE A, ROWLAND RW, YEUNG S, JOHNSON NW, *Oral Dis*, 8 (2002) 144. — 4. GONCALVES LS, FERRE-

IRA SM, SILVA AJ, VILLORIA GE, COSTINHA LH, SOUTO R, UZEDA MD, COLOMBO AP, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*, 97 (2004) 196. — 5. KLIMIUK A, WASZKIEL D, CHOROMANSKA M, JANKOWSKA A, ZELAZOWSKA-RUTKOWSKA B, *Adv Med Sci*, 51

- (2006) 46. — 6. CHOROMANSKA M, WASZKIEL D, Adv Med Sci, 51 (2006) 110. — 7. RANGANATHAN K, MAGESH KT, KUMARASAMY N, SUNITI S, VISWANATHAN R, NEWELL WJ, Indian J Dent Res, 18 (2007) 55. — 8. ARENDORF TM, BREDEKAMP B, CLOETE CA, SAUER G, J Oral Pathol Med, 27 (1998) 176. — 9. CENTERS FOR DISEASE CONTROL, Morb Mortal Wkly Rep, 43 (1994) 644. — 10. CENTERS FOR DISEASE CONTROL, Morb Mortal Wkly Rep, 45 (1995) 392. — 11. ALVES M, MULLIGAN R, PASSARO D, GAWELL S, NAVAZESH M, PHELAN J, GREENSPAN D, GREENSPAN JS, J Periodontol, 77 (2006) 773. — 12. BARONE R, FICARRA G, GAGLIOTI D, ORSI A, MAZZOTTA F, Oral Surg Oral Med Oral Pathol, 69 (1990) 169. — 13. FRIEDMAN RB, GUNSOLLEY J, GENTRY A, DINIUS A, KAPLOWITZ L, SETTLE J, J Periodontol, 62 (1991) 623. — 14. WINKLER JR, HERRERA C, WESTENHOUSE J, ROBINSON P, HESSOL N, BUCHBINDER S, GREENSPAN JS, KATZ MH, AIDS, 6 (1992) 1041. — 15. ROBINSON P, SHEIHAM A, CHALLACOMBE S, ZAKRZEWSKA JM, Oral Dis, 2 (1996) 45. — 16. TOMAR SL, SWANGO PA, KLEINMAN DV, BURT BA, J Periodontol, 66 (1995) 421. — 17. MUEHLEMANN HR, SON S, Helvetic Odontologia Acta, 15 (1971) 107. — 18. WORLD HEALTH ORGANIZATION, Application of the International Classification of Diseases to Dentistry and Stomatology (World Health Organization, Geneva, 1995). — 19. ROBINSON PG, WINKLER JR, PALMER G, WESTENHOUSE J, HILTON JF, GREENSPAN JS, J Periodontol, 65 (1994) 236. — 20. SWANGO PA, KLEINMAN DV, KONZELMAN JL, J Am Dent Assoc, 122 (1991) 49. — 21. GLICK M, MUZYKA BC, LURIE D, SALKIN LM, Oral Surg Oral Med Oral Pathol, 77 (1994) 344. — 22. BARR C, LOPEZ MR, RUA-DOBLES A, J Clin Periodontol, 19 (1992) 794. — 23. MATTEE MI, SCHEUTZ F, MOSHY J, Oral Dis, 6 (2000) 106. — 24. HODGSON TA, Oral Dis, 3 (1997) 46. — 25. LASKARIS G, POTOURIDOU I, LASKARIS M, STRATIGOS J, Oral Surg Oral Med Oral Pathol, 74 (1992) 168. — 26. BERGSTROM J, PREBER H, J Periodont Res, 21 (1986) 668. — 27. DANIELSEN B, MANJI F, NAGELKERKE N, FEJERSKOV O, BAEUM V, J Clin Periodontol, 17 (1990) 159. — 28. NEWMAN MG, TAKEI H, CARRANZA FA, Carranza's Clinical Periodontology (WB Saunders Company, New York, 2002). — 29. LAMSTER IB, GRBIC JT, BUCKLAN RS, MITCHELL-LEWIS D, REYNOLDS HS, ZAMBON JJ, Oral Dis, 3 (1997) 141. — 30. GJENERO-MARGAN I, HIV/AIDS-Epidemiološka situacija u Hrvatskoj 2004, Služba za epidemiologiju zaraznih bolesti, Hrvatski zavod za javno zdravstvo, accessed 30.12.2005. Available from: URL: <http://www.hzjz.hr/epidemiologija/hiv.htm> — 31. BEGOVAC J, LISIĆ M, LUKAS D, MARETIĆ T, KNIEWALD T, NOVOTNY TE, Coll Antropol, 30 (2006) 175. — 32. BEGOVAC J, ŽIDOVEC LEPEJ S, KNIEWALD T, LISIĆ M, Coll Antropol, 25 (2001) 111.

A. Stojković

University of Zagreb, School of Dental Medicine, Department of Periodontology, Gundulićeva 5, 10000 Zagreb, Croatia  
e-mail: ana.a.stojkovic@gmail.com

## PROCJENA PARODONTOLOŠKOG STATUSA OBOLJELIH OD HIV-a U REPUBLICI HRVATSKOJ

### SAŽETAK

Brojne parodontalne promjene povezivane su s infekcijom izazvanom humanim imunodeficijencijskim virusom (HIV-om), ali naše znanje o epidemiologiji, mikrobiologiji, odgovoru domaćina i prirodnom tijeku ovakvih promjena i dalje ostaje ograničeno. Stoga je cilj ovog ispitivanja procjena mogućih razlika u parodontalnom statusu u oboljelih od HIV-a u odnosu na zdrave ispitanike kontrolne skupine podudarne po dobi, spolu i navici pušenja u hrvatskoj populaciji. Provedena je procjena nakupljanja plaka uporabom aproksimalnog plak indeksa, procjena upale gingive uporabom indeksa krvarećeg sulkusa, procjena dubine džepova, recesije gingive kao i broja karijesnih, ekstrahiranih i saniranih zuba u 25 osoba oboljelih od HIV-a (raspon dobi 22–61, srednja dob 40,8 godina) u odnosu na 25 ispitanika kontrolne skupine (raspon dobi 20–62, srednja dob 40,92 godina). Statistička analiza napravljena uz pomoć deskriptivne statistike i Mann-Whitneyevog U-testa pokazala je da je upala marginalne gingive značajno jače izražena u HIV oboljelih u odnosu na ispitanike kontrolne skupine ( $p < 0,002$ ). Srednje vrijednosti dubine parodontalnih džepova ( $p < 0,002$ ) i najdubljih parodontalnih džepova ( $p < 0,003$ ) izmjenjenih u oboljelih od HIV-a su bile značajno veće u odnosu na one izmjerene u kontrolnoj skupini. Značajno veći broj karijesnih ( $p < 0,002$ ) i ekstrahiranih ( $p < 0,002$ ) te manji broj saniranih zuba ( $p < 0,009$ ) nađen je u HIV pozitivnih osoba u odnosu na kontrolnu skupinu. Rezultati ovog ispitivanja još jednom naglašavaju potrebu za češćim parodontalnim pregledima i liječenjem HIV pozitivnih osoba.