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A retrospective analysis of oral hairy leukoplakia in South Australia

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Abstract

Background: The features of oral hairy leukoplakia (OHL) have been widely reported in the literature. However, no studies have described this lesion in the Australian setting. This study retrospectively examines, with respect to specific clinical factors, the prevalence of OHL in a South Australian HIV-infected population.

Methods: Clinical data were collected from the records of 197 HIV-infected patients who had attended the Adelaide Dental Hospital between January 1986 and February 1995. Data were analysed using the chi-square test.

Results: The prevalence of OHL in South Australian HIV-infected patients was 45.2 per cent. The study found the presence of OHL was not related to $CD4^+$ T-lymphocyte count or AIDS-defining illness nor did the length of time a patient had been infected with HIV relate to the presence of OHL. An association was observed between a reduced prevalence of OHL in patients who were taking antiviral medication.

Conclusion: The prevalence of OHL in South Australia is comparable with results of other studies. This study supports the notion that OHL is not an indicator of immunosuppression in South Australian HIV-infected patients. Further longitudinal studies are required to ascertain the relationship of OHL to HIV disease progression.

Key words: Oral hairy leukoplakia, HIV infection, AIDS.

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Introduction

Oral hairy leukoplakia (OHL) is a white, non-removable lesion, usually confined to the lateral borders of the tongue. It was first reported in 1984 when it was detected in a group of HIV-infected patients in San Francisco.[1]

The importance of OHL as an indicator of immunosuppression and poor prognosis of HIV disease was recognised soon after it was first described.[1] The authors observed that a proportion of the patients developed AIDS-defining illnesses within a relatively short time following the diagnosis of OHL. Subsequently, OHL was included in the 1993 Revised Classification System for HIV Infection and Expanded Case Definition for AIDS among Adolescents and Adults.[2] Furthermore, the European Community (EC) Clearinghouse on oral problems related to HIV Infection and the World Health Organization (WHO) Collaborating Centre on the oral manifestations of the Human Immunodeficiency Virus[3] included OHL in its classification.

OHL has been reported to occur in all risk groups for HIV infection including homosexual men, patients with haemophilia, injecting drug users, partners of people with HIV infection and children. In rare cases, OHL has been identified in patient groups who are immunosuppressed as a result of immunosuppressive mechanisms other than HIV infection.[4-5]

Epstein-Barr virus (EBV) is accepted as the aetiologic agent for this lesion and demonstration of its presence is required for the definitive diagnosis of OHL.[3] This virus, a member of the herpes family, infects a large proportion of the world's population and is the aetiologic factor in a range of diseases including infectious mononucleosis, Burkitt's lymphoma and nasopharyngeal carcinoma. The exact role EBV plays in the pathogenesis of OHL is unclear and it is yet to be determined why OHL is predominantly found on the lateral border of the tongue.

Several studies have investigated OHL with respect to its clinical appearance,[1,6-8] histologic features[1,6] and relevance to HIV disease progression.[9-13] The role of OHL as an indicator of HIV progression in Australian populations has not been described. This analysis of OHL in a South Australian HIV-infected population was carried out in order to gain more information on the lesion in the Australian setting.

Materials and methods

Data were obtained from records of 197 HIV-positive patients attending the Medically Compromised Unit of the Adelaide Dental Hospital from January 1986 to February 1995. Patients were referred to this unit for dental management from a variety of sources,

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principally private medical practitioners, the Royal Adelaide Hospital and the Flinders Medical Centre.

The information recorded at each patient's visit included estimated time since HIV seroconversion, date of diagnosis, CD4+ T-lymphocyte counts, current medication and any AIDS-defining illnesses they may have experienced. All this information was provided through consultation with the patients' referring medical practitioners.

For the purpose of this study, data derived from records were analysed for the prevalence and site of OHL; presence of OHL and AIDS-defining illness; presence of OHL and concurrent medication; presence of OHL and length of time since infection with HIV; and presence of OHL and CD4+ T-lymphocyte count.

OHL was defined according to the EC-Clearing-house-WHO criteria.[3] According to these criteria, OHL is a white lesion that is present on the lateral border of the tongue. The lesions can extend on to the ventral and dorsal surfaces of the tongue and, in some cases, involve the buccal mucosa. Medical conditions were classed as AIDS-defining conditions according to the 1993 Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS among Adolescents and Adults.[2] Data were statistically analysed using chi-squared analysis.

Results

The group of 197 patients was composed of 186 (94.4 per cent) males and 11 (5.6 per cent) females. The mean age of the group was 36.9 years (range 19-67 years). One hundred and eighty-one patients were male homosexuals, 13 patients (two male, 11 female) had a known history of injecting drug use and three patients (all male) had haemophilia. Thirty-nine patients died during the period January 1986 to February 1995.

Prevalence and site of oral hairy leukoplakia

Clinical evidence of OHL was seen on the lateral border of the tongue in 89 patients (45.2 per cent). In 40 (44.9 per cent) of these cases, OHL was bilateral.

In three patients, OHL also occurred on the buccal mucosa. These three patients also had bilateral OHL lesions on their tongues.

Table 1. AIDS-defining illnesses that occurred within the group and the number of study subjects with and without OHL according to each illness

AIDS-defining illnesses	OHL present	OHL absent
Pneumocystis carinii pneumonia	4	8
Kaposi's sarcoma	4	4
Mycobacterium avium complex	3	4
Cytomegalovirus infection	1	3
Cryptococcal infection	3	1
Aspergillus infection	3	1
Herpes simplex infection	3	2
Tuberculosis	0	1
Lymphoma	0	2

Table 2. The types of medications taken by study subjects and the numbers of patients with OHL present according to each type of medication

Medication	OHL present	OHL absent
Antiretroviral medication		
Zidovudine (AZT)	27	64
Didanosine (ddI)	16	21
Zalcitabine (ddC)	3	12
Stavudine (d4T)	1	0
Other antiviral medication		
Aciclovir	14	35
Ganciclovir	2	1
Antifungal medication		
Fluconazole	18	17
Ketoconazole	2	10
Itraconazole	4	2
Antibacterial medication		
Ethambutol	2	5
Rifampicin	1	3
Clofazamine	1	2
Clarithromycin	1	2
Trimethoprim/sulfamethoxazole combinations	19	37
Pentamidine	4	3

Presence of oral hairy leukoplakia and AIDS-defining illness

One hundred and sixty-one patients had no AIDS-defining medical condition recorded in their dental records. Of these, 70 (43.5 per cent) had OHL lesions. Thirty-six patients showed an AIDS-defining illness recorded in their case notes (Table 1). Seventeen (51.5 per cent) patients in this group had OHL [X^2=0.074, $p>0.5$ (χ^2 analysis)].

Presence of oral hairy leukoplakia and concurrent medication

One hundred and fifty-eight patients were taking medication (Table 2). Of these, 77 (48.7 per cent) had OHL [X^2=4.076, $0.02<p<0.05$ (χ^2 analysis)]. Thirty-nine patients did not take any medication and, of these, 30.8 per cent had OHL.

One hundred and thirty patients were taking anti-viral medication. Ninety-one of these were taking AZT and, of this group, 27 (29.7 per cent) had OHL compared with 62 (58.5 per cent) patients who were not taking AZT and showed OHL [X^2=16.420, $0.001<p<0.01$ (χ^2 analysis)].

Forty-nine patients were taking aciclovir and, of these, 14 (28.6 per cent) showed OHL compared with 75 (50.7 per cent) patients who were not taking aciclovir and had OHL [X^2=7.262, $0.001<p<0.01$ (χ^2 analysis)].

Table 3. Presence of OHL and length of time since infection with HIV

Time since infection	OHL present	OHL absent
<5 years	36	40
5-10 years	33	52
>10 years	20	16
Total	89	108

Table 4. The relationship between CD4+ T-lymphocyte count and the presence of OHL

CD4+ T-lymphocyte count	OHL present	OHL absent
<200 cells/μL	27	26
200-500 cells/μL	30	30
>500 cells/μL	15	26
Total	68	82

Presence of oral hairy leukoplakia and length of time since infection with HIV

The relationship between the presence of OHL and the length of time each patient had been infected with HIV is shown in Table 3 [X^2=3.116, 0.10<p<0.05 (χ^2 analysis)].

Presence of oral hairy leukoplakia and CD4+ T-lymphocyte count

CD4+ T-lymphocyte counts were recorded in the case notes of 150 of the 197 patients. The numbers of CD4+ T-lymphocytes in patients with OHL ranged from 0-1200 cells/μL (mean 294 cells/μL). Patients without OHL had CD4+ T-lymphocyte counts ranging from 0-2200 cells/μL (mean 377 cells/μL).

The patients were divided into three groups according to CD4+ T-lymphocyte count, namely <200 cells/μL, 200-500 cells/μL and >500 cells/μL. Table 4 shows the relationship between CD4+ T-lymphocyte count and the presence of OHL [X^2=1.966, 0.1<p<0.5 (χ^2 analysis)].

In patients infected with HIV for less than five years, those who had OHL had a mean CD4+ T-lymphocyte count of 303.14 cells/μL compared with 400.18 cells/μL in patients without OHL. Table 5 shows the relationship between CD4+ T-lymphocyte count and the presence of OHL in patients infected with HIV for less than five years [X^2=0.425, p>0.5 (χ^2 analysis)].

In patients who had had HIV infection for between five and 10 years, those who had OHL had a mean CD4+ T-lymphocyte count of 404.03 cells/μL compared with 385.21 cells/μL in patients without OHL. Table 6 shows the relationship between CD4+ T-lymphocyte count and the presence of OHL in patients infected with HIV for between five and 10 years [X^2=1.122, p>0.5 (χ^2 analysis)].

In patients infected with HIV for more than 10 years, those who had OHL had a mean CD4+ T-lymphocyte count of 174.85 cells/μL compared with 345.60 cells/μL in patients without OHL. Table 7 shows the relationship between CD4+ T-lymphocyte count and the presence of OHL in patients infected with HIV for more than 10 years [X^2=3.608, 0.1<p<0.5 (χ^2 analysis)].

Table 5. The relationship between CD4+ T-lymphocyte count and the presence of OHL in patients where HIV seroconversion occurred <5 years previously

CD4+ T-lymphocyte count	OHL present	OHL absent
<200 cells/μL	11	7
200-500 cells/μL	9	7
>500 cells/μL	8	8
Total	28	22

Table 6. The relationship between CD4+ T-lymphocyte count and the presence of OHL in patients where HIV seroconversion occurred between five and 10 years previously

CD4+ T-lymphocyte count	OHL present	OHL absent
<200 cells/μL	7	16
200-500 cells/μL	10	16
>500 cells/μL	6	15
Total	23	47

Discussion

There are limited Australian data describing the oral manifestations of HIV infection. Coates et al[14] reported a study of the social impact of oral conditions among HIV-infected dental patients in South Australia. This study concentrated on clinical dental indices such as the number of decayed, missing and filled teeth and the community periodontal index of treatment needs. The present study provides additional, more specific, information on OHL in relation to its association with systemic features of HIV disease.

OHL attracted attention early in the global HIV epidemic because of its potential significance as a prognostic factor for HIV infection. Its presence, as suggested by Greenspan et al,[1] was thought to herald the progression to AIDS or even death. Other international studies have since found OHL to be a marker of immunosuppression and progression of HIV disease.[9-12,15]

The diagnosis of OHL in this study was based on presumptive criteria as outlined by the 1993 EC-Clearinghouse-WHO criteria.[3] These diagnostic criteria require the lesions are white and present on the lateral border of the tongue. The lesions can extend on to the ventral and dorsal surfaces of the tongue and, in some cases, involve the buccal mucosa. Definitive diagnosis of OHL requires the demonstration of EBV within the lesions, or, where this is not possible, a lack of response to antifungal treatment can add weight to a presumptive diagnosis. It was not always evident, by examination of the case note entries, whether antifungal treatment was employed to differentiate lesions.

In the present study, the clinical findings in relation to the predominant site of occurrence of OHL were in accord with those of investigators from other countries.

Table 7. The relationship between CD4+ T-lymphocyte count and the presence of OHL in patients where HIV seroconversion occurred >10 years previously

CD4+ T-lymphocyte count	OHL present	OHL absent
<200 cells/μL	9	3
200-500 cells/μL	9	5
>500 cells/μL	1	3
Total	19	11

Schiødt et al[6] found, in a sample of 50 patients, OHL occurred exclusively on the lateral border of the tongue and 86 per cent of patients had OHL occurring bilaterally. In the present study, bilateral OHL was not observed as frequently as this and only 44.9 per cent of our patients showed OHL occurring bilaterally on their tongues.

There are two explanations for this difference. First, there may have been inaccuracies in the diagnosis of OHL in both the present study and in the study by Schiødt et al. Second, the presence of OHL in patient records could have been under-reported. Apart from the possibility of a real difference in the clinical presentation of OHL between the two studies, the differences may be a reflection of the difference in sample sizes. The present study may give a more accurate picture of the clinical presentation of OHL because of the greater number of subjects in the study.

Other studies have observed more extensive involvement of the oral cavity by OHL,[7-8] and have described OHL occurring on the buccal mucosa, floor of the mouth, palate and dorsal surface of the tongue. OHL was found on the buccal mucosa in three patients in the present study. Although an interesting clinical feature from a diagnostic point of view, it has been shown the severity of OHL, as opposed to its presence, does not seem to be predictive for immunosuppression or for the development of AIDS in patients infected with HIV.[6]

The most common oral manifestations of HIV infection, OHL and oral candidiasis, have been found to occur sooner after HIV seroconversion than the development of AIDS. Lifson et al[13] found, within five years after seroconversion, 42 per cent of the patients in their study developed OHL. Although not statistically significant, the present study found a slight increase in the prevalence of OHL after a period of infection with HIV that was greater than or equal to 10 years; in this instance, OHL was found in 55.6 per cent of patients.

Extensive OHL has been reported in two studies where it occurred concurrently with an AIDS-defining illness. Kabani et al[7] described extensive OHL in a patient four months after the patient developed KS and PCP. Southam et al[16] reported a case where OHL occurred in the epithelium overlying KS lesions in the oral cavity.

The present study examined current or previous AIDS-defining illnesses relative to the prevalence of OHL. Only 36 out of the 197 patients were identified as having had an illness that was AIDS defining. In this group of patients, the prevalence of OHL was 52.8 per cent. The prevalence of OHL in the 161 patients without any reported AIDS-defining condition was 43.5 per cent. There was no statistical significance in the prevalence of OHL between these two groups.

In relation to a single AIDS-defining illness, because of the low patient numbers, it was not possible to draw any conclusions regarding the relationship to OHL.

One hundred and fifty-eight patients in this study were taking medication of some kind, either for the treatment of HIV infection, the treatment of opportunistic infections, or for the prophylaxis of opportunistic infections. The prevalence of OHL in patients who were taking medication was 48.7 per cent. Although this figure is only slightly higher than the overall prevalence of 45.2 per cent for the sample as a whole, it may be a reflection of more advanced immune deficiency in some of these patients.

Some studies have reported a resolution of OHL following the commencement of antiviral medication.[17-19] Forty-nine patients in the present study were taking aciclovir and the prevalence of OHL in this group was 28.1 per cent, statistically significant compared with the prevalence of OHL in patients who were not taking aciclovir. Given the postulated role of EBV in the pathogenesis of OHL, it follows that aciclovir should have some impact on the prevalence of the lesion. This drug has been shown to stop EBV production by inhibiting the EBV DNA polymerase enzyme.[21]

Resnick et al[18] described the regression of OHL following the use of aciclovir in five out of six patients who had clinical and histological evidence of OHL. The use of intravenous aciclovir as a potential treatment for OHL has also been reported, resulting in resolution of OHL until cessation of the treatment.[20] Although the response of OHL to various drugs is interesting as far as the behaviour of the lesion is concerned, treatment of OHL is not generally required because of the asymptomatic nature of the lesion.

Antiretroviral drugs have also been reported to result in resolution of OHL. Phelan and Klein[19] presented a case report of a patient who had a 10-month history of continuous OHL. Within six weeks of treatment with AZT for HIV infection, the OHL had completely resolved. Ninety-one patients in the present study were taking AZT and again the prevalence of OHL was significantly reduced compared with the overall prevalence in the total sample population.

The reduced prevalence of OHL in patients taking antiviral medication may be a result of direct action by the drug. This is especially likely in the case of aciclovir and ganciclovir, which are drugs directed at herpes viruses (EBV is a member of the herpes virus family). Alternatively, the patient's immune function may be improved by antiviral medication, enabling a more effective immune response to EBV, thus reducing viral replication. Although these drugs definitely have a beneficial effect on OHL lesions, the results of the present study indicate there must be other factors that lead to the development of OHL. Hence, it appears likely the pathogenesis of OHL is multifactorial and that viruses, namely EBV, are possibly co-factors in the development of OHL.

Thirty-nine patients were not taking any medication and the prevalence of OHL in this group was 30.8 per cent. The lower prevalence of OHL than that found in the group of patients taking medication may reflect the fact the patients not on medication were healthier in terms of their immune function.

CD4+T-lymphocyte counts have been used to monitor the health of patients with HIV infection to give an indication of HIV disease progression. CD4+ T-lymphocyte counts also help predict the development of opportunistic infections.[22-23] Reichart et al[24] found, in a study of 95 HIV-infected patients with clinical and histological evidence of OHL, the average CD4+ T-lymphocyte count was 149 cells/μL (range 10-470 cells/μL). Glick et al[25] found, in patients with OHL, the mean CD4+ T-lymphocyte count was 143.3 cells/μL. The same study reported the presence of OHL in HIV-positive patients had a positive predictive value of 70.1 per cent and the CD4+ T-lymphocyte count would be less than 200 cells/μL. In addition, Begg et al[11] found a significant association between a low level of CD4+ T-lymphocytes and OHL in injecting drug users.

A chronological model of HIV disease progression has been illustrated by Stewart[22] in which CD4+ T-lymphocyte counts and opportunistic infections associated with HIV are correlated. According to this model, oral manifestations of HIV infection are more likely to occur at an intermediate level of immunodeficiency where CD4+ T-lymphocytes are between 200-500 cells/μL.

In the present study, it was interesting to note the range of CD4+ T-lymphocyte counts was broad, both in the group of patients without OHL (0-2200 cells/μL) and in the group of patients with OHL (0-1200 cells/μL). This was also reported by Lifson et al.[13] The mean CD4+ T-lymphocyte count for patients with OHL in the present study was 294 cells/μL, which is in accordance with the model proposed by Stewart.[22] The mean CD4+ T-lymphocyte count in patients without OHL was 377 cells/μL.

Ravina et al[26] found patients with OHL demonstrated a greater decline in CD4+ T-lymphocytes over an eight-year period compared with HIV-infected patients without OHL. This is also inferred by the results of the present study by determining CD4+ T-lymphocyte counts and presence of OHL after placing the patients into groups according to the length of time since estimated seroconversion.

At less than five years since estimated seroconversion, the CD4+ T-lymphocyte count for patients with OHL was 303 cells/μL compared with 400 cells/μL in patients without OHL. When the time since estimated seroconversion was greater than 10 years, the CD4+ T-lymphocyte counts in patients with OHL was 175 cells/μL compared with 346 cells/μL in patients without OHL. An exception noted in the current study was a reversal of the CD4+ T-lymphocyte count in the group of patients who had seroconverted between five and 10 years previously. In this group of patients, CD4+ T-lymphocyte counts were higher in patients with OHL (404 cells/μL) than in patients without OHL (385cells/μL).

The results of this study do not demonstrate any statistically significant relationship between the presence of OHL and CD4+ T-lymphocyte count. This supports our original hypothesis that the presence of OHL was not associated with a lower CD4+ T-lymphocyte count in this Australian group of patients. Further detailed longitudinal studies would provide more accurate information and it would also be interesting to observe how the presence of OHL relates to HIV viral load.

References

1. Greenspan D, Greenspan JS, Conant M, et al. Oral hairy leukoplakia in male homosexuals: evidence of association with both papillomavirus and a herpes group virus. Lancet 1984;2:831-834.
2. Centers for Disease Control and Prevention. 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR 1992;41(RR-17):1-35.
3. EC-Clearinghouse on oral problems related to HIV infection and WHO collaborating centre on oral manifestations of the immunodeficiency virus: Classification and diagnostic criteria of oral lesions in HIV infection. J Oral Pathol Med 1993;22: 289-291.
4. Epstein JB, Sherlock CH, Wolber RA. Hairy leukoplakia after bone marrow transplantation. Oral Surg Oral Med Oral Pathol 1993;75:690-695.
5. Greenspan D, Greenspan JS, De Souza YG, et al. Oral hairy leukoplakia in an HIV-negative renal transplant recipient. J Oral Pathol Med 1989;18:32-34.
6. Schiødt M, Greenspan D, Daniels TE, et al. Clinical and histologic spectrum of oral hairy leukoplakia. Oral Surg Oral Med Oral Pathol 1987;64:716-720.
7. Kabani S, Greenspan D, DeSouza Y, et al. Oral hairy leukoplakia with extensive oral mucosal involvement – report of two cases. Oral Surg Oral Med Oral Pathol 1989;67:411-415.
8. Ficarra G, Romagnoli P, Piluso S, et al. Hairy leukoplakia with involvement of the buccal mucosa. J Am Acad Dermatol 1992;27:855-858.
9. Greenspan D, Greenspan JS, Hearst NG, et al. Relation of oral hairy leukoplakia to infection with the human immunodeficiency virus and the risk of developing AIDS. J Infect Dis 1987;155: 475-481.
10. Kolokotronis A, Kioses V, Antoniades D, et al. Immunologic status in patients infected with HIV with oral candidiasis and hairy leukoplakia. Oral Surg Oral Med Oral Pathol 1994;78: 41-46.
11. Begg MD, Panageas KS, Mitchell-Lewis D, et al. Oral lesions as markers of severe immunosuppression in HIV infected homosexual men. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82:276-283.
12. Ramírez-Amador VA, Esquivel-Pedraza L, Ponce de León S. Prognostic value of oral candidosis and hairy leukoplakia in 111 Mexican HIV-infected patients. J Oral Pathol Med 1996;25: 206-211.
13. Lifson AR, Hilton JF, Westenhouse JL, et al. Time from HIV seroconversion to oral candidiasis or hairy leukoplakia among homosexual and bisexual men enrolled in three prospective cohorts. AIDS 1994;8:73-79.
14. Coates E, Slade GD, Goss AN, et al. Oral conditions and their social impact among HIV dental patients. Aust Dent J 1996;41:33-36.
15. Ceballos-Salobreña A, Aguirre-Urizar JM, Baga-Sebastian JV. Oral manifestations associated with human immunodeficiency virus infection in a Spanish population. J Oral Pathol Med 1996;25:523-526.
16. Southam JC, Felix DH, Cubie HA, et al. Multifocal hairy leukoplakia associated with Kaposi's sarcoma. Br Dent J 1992;172:21-23.
17. Newman C, Polk BF. Resolution of oral hairy leukoplakia during therapy with 9-(1,3-dihidroxy-2-propoxymethyl) guanine (DHPG). Ann Intern Med 1987;107:348-350.
18. Resnick L, Herbst JS, Ablashi DV, et al. Regression of oral hairy leukoplakia after orally administered acyclovir therapy. JAMA 1988;259:384-388.

19. Phelan JA, Klein RS. Resolution of oral hairy leukoplakia during treatment with azidothymidine. Oral Surg Oral Med Oral Pathol 1988;65:717-720.
20. Schöfer H, Ochsendorf FR, Helm EB, et al. Treatment of oral hairy leukoplakia in AIDS patients with vitamin A acid (topically) or with acyclovir (systemically). (letter) Dermatologica 1987;174:150-151.
21. Elion GB. Mechanism of action and selectivity of acyclovir. Am J Med 1982;73:7-12.
22. Stewart GJ. The chronology of HIV-induced disease. Med J Aust 1993;158:3-5.
23. Dwyer DE, Adelstein S, Cunningham AL, et al. The laboratory monitoring of HIV infection. Med J Aust 1996;164:301-303.
24. Reichart PA, Langford A, Gelderblom HR, et al. Oral hairy leukoplakia: observations in 95 cases and review of the literature. J Oral Pathol Med 1989;18:410-415.
25. Glick M, Muzyka BC, Lurie D, et al. Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS. Oral Surg Oral Med Oral Pathol 1994;77:344-349.
26. Ravina A, Ficarra G, Chiodo M, et al. Relationship of circulating CD4+ T-lymphocytes and p24 antigenemia to the risk of developing AIDS in HIV-infected subjects with oral hairy leukoplakia. J Oral Pathol Med 1996;25:108-111.

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