International Journal of Applied Pharmaceutics

ISSN- 0975-7058 Vol 11, Special Issue 4, 2019

Full Proceeding Paper

ORAL HAIRY LEUKOPLAKIA MANIFESTATIONS RELATED TO CD4 COUNT IN HIV/AIDS PATIENTS AT DR WAHIDIN SUDIROHUSODO HOSPITAL

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Received: 21 Jan 2019, Revised and Accepted: 02 Jun 2019

ABSTRACT

Objective: The aim of this study to determine the oral hairy leukoplakia manifestations related to CD4 counts in HIV/AIDS patients.

Methods: A cross-sectional study was conducted among 30 patients with HIV/AIDS, who have oral hairy leukoplakia manifestations. Diagnosis of oral hairy leukoplakia (OHL) based on clinical features. CD4 cell counts were obtained from medical records of patients.

Results: The result show that oral hairy leukoplakia related to CD4 cell counts in HIV/AIDS patients had CD4 count<100 cells/mm^3 (13.3%) and CD4 counts>100 cells/mm^3 (3.3 %).

Conclusion: Oral hairy leukoplakia manifestations in HIV/AIDS patients correlated with low levels CD4 cell counts.

Keywords: Oral hairy leukoplakia, CD4 cell, HIV/AIDS

DOI: http://dx.doi.org/10.22159/ijap.2019.v11s4.35292

INTRODUCTION

Oral hairy leukoplakia (OHL) is an oral mucosal lesion caused by Epstein Barr virus (EBV). This lesion is a hyperplasia disorder of mucocutaneous epithelial cell. Clinical manifestations of lesions usually asymptomatic, white, extended, unilateral or bilateral on the lateral of the tongue border and often hairy or corrugated appearance and can also be plaque-like lesion [1-3]. EBV is a human herpes virus associated with important diseases in humans, including mononucleosis syndrome, infectious, lymphoma malignant and nasopharyngeal carcinoma. The prevalence of serologic EBV is estimated about 95% in adult humans worldwide. Circulation of the latently infected B-lymphocyte is believed to be a persistent area of lifelong infection of EBV [4].

The prevalence of OHL in adults is 20%-25% and increases with decreasing CD4 cell count, whereas in children the prevalence is about 2%-3%, OHL is more common of adults with HIV infection than in HIV-infected children, the presence of OHL is a sign of severe immunosuppression [5]. In people with AIDS, decline in the ability of the immune system is closely related with the incidence of opportunistic infections [6]. Greenspan *et al.* first found OHL in a group of homosexual patients in San Francisco. OHL is found usually in patients with human immunodeficiency virus (HIV), although it may be reported in OHL patients with other immunosuppressive conditions, such as patients undergoing organ transplantation, patients with haematological malignancies, and in patients requiring systemic steroid therapy [7-9].

The results of observational studies indicate that OHL in HIV-infected patients with CD4 cell counts below 200 cells/mm^3 have rapid progression to AIDS. OHL is a disease with small morbidity and does not always include intervention. Therefore, the OHL lesions are benign, asymptomatic and potentially self-limiting. OHL when associated with HIV infection, often with highly active anti-retroviral therapy (HAART) can trigger healing of OHL with decreased viral load and an increase in CD4 cell count. In general, OHL clinical findings are sufficient to diagnose patients with HIV infection, which is a typical manifestation [10].

This study aims to determine the oral manifestation of hairy leukoplakia associated with CD4 cell count in HIV/AIDS patients.

MATERIALS AND METHODS

Material

The analytical observational study with cross-sectional study was conducted at Infection Center of DR Wahidin Sudirohusodo Hospital, Makassar, South Sulawesi (Ethical clearance from Medical Research Ethics Committee of Hasanuddin University NO UH16050421) study sample of 30 HIV/AIDS patients. The inclusion criteria was HIV/AIDS patients with CD4 cell counts<350 cells/mm2 at DR Wahidin Sudirohusodo Hospital while exclusion criteria were HIV/AIDS patients who were unwilling to participate this research. The diagnosis of oral hairy leukoplakia is determined based on clinical features in the oral cavity. The research procedure included data collection on HIV/AIDS patients at DR Wahidin Sudirohusodo Hospital. The type of data is secondary data obtained from the medical record and primary data obtained directly from the sample.

Data was obtained by looking at the medical records of CD4 cell counts of HIV/AIDS patients then examining the patient's oral cavity to see the manifestation of OHL in the form of unusual white lesions on the lateral border of the tongue.

RESULTS AND DISCUSSION

In this study, OHL was examined by the presence of oral hairy leukoplakia manifestations associated with CD4 cell counts of HIV/AIDS patients who complied the inclusion and exclusion criteria of 30 patients. The decrease in CD4 cell count with the development of OHL manifestation in HIV/AIDS patients in terms of age group, CD4 cells, which can be seen in the table below.

Table 1: Distribution of respondents by age group on development research of hairy Leukoplakia 2016

Age (Years)	N	%
30-35	3	10.0 %
36-40	1	3.3 %
>50	1	3.3 %
Total	30	16,6 %

Source: Primary Data, table 1 shows that from 30 respondents, the highest percentage of respondents is in the 30-35 y age group, which is 10%. While the lowest percentage of respondents is in the age group 36-40 y 3.3% and>50 y of 3.3%

Table 2: Distribution of respondents by CD4 category in development research of hairy Leukoplakia year 2016

CD4 category	N	%
<=100	4	13.3 %
>100	1	3.3 %
Total	30	16.6 %

Source: Primary Data, table 2 shows that of 30 respondents who had CD4 count<100 cells/mm^3 l counted 4 peoples (13.3%) while those with CD4 count>100 cells/mm^{3}were 1 people (3.3%).

Clinically, OHL is a white plaque with corrugated surfaces, painless, and not removable by scraping. Mortin *et al.* explained that it is called hairy leukoplakia because of its white colour, and corrugated or shaggy appearance of the lateral tongue seen in many cases. The histopathological characteristics are hyperkeratosis, epithelial hyperplasia [3].

Oral hairy leukoplakia (OHL) is the result of infection associated with the presence of Epstein Barr virus (EBV) and almost all are found in HIV-infected individuals. Therefore a study of oral hairy leukoplakia manifestations was associated with CD4 cell count in HIV/AIDS patients. In this study obtained a total of 30 samples. Primary and secondary data taken by examining the oral cavity and also by looking at the medical records of people with HIV/AIDS.

Characteristics of samples were seen from the age, known that the OHL manifestation in people with HIV/AIDS occurred at the age ranged from 30-35 y 10.0%, and in the age group 36-40 y and over 50 y 3.3%. This is consistent with the results of several studies suggesting that OHL is more common in HIV-infected adults than in HIV-infected children. The result of research by Innes (2014) conducted at Dr Kariadi Hospital Semarang on 42 HIV/AIDS patients in the group age 30-39 y. This result is also accordance with previous research showing that the highest percentage of HIV/AIDS patients recorded in CVT clinic RSUD Arifin Achmad is age group 30-39 y amounted to 37 people (42.04%) followed by age 20-29 y amounted to 26 people (55%). From the results of these studies, it can be seen that cases of HIV/AIDS occur in the productive age 20-49 y. In the productive age group, a person is more likely to engage in sexual activities and drug abuse that is a high risk of transmitting the virus.

Based on CD4 cell count with hairy leukoplakia progression, this study found<100 cells/mm^3 count in 4 people (13.3%) while CD4 count>100 cells/mm^3 counted in 1 person (3.3%). Ghate *et al.* showed that patients with CD4 cell count<200 cells/mm^3 had a six-fold susceptibility in the development of opportunistic infections compared with CD4 cell count>350 cells/mm^3. The Center for Disease Control and Prevention classified HIV patients into 3 categories based on CD4 cell counts: CD4>500 cells/mm^3, 200-499 cells/mm^3 and<200 cells/mm^3. The lower CD4 cell count is associated with more severe immunosuppression [11].

According to Greenspan *et al.* hairy leukoplakia is an unusual white lesion on the lateral border of tongue. Recent study indicating that this hairy leukoplakia is the result of opportunistic infections associated with the presence of Epstein Barr virus (EBV) and almost all found in individuals with HIV infection. OHL is caused by the autoinoculation of Epstein Barr virus (EBV) through saliva and is associated with immunosuppression caused by HIV infection. EBV that has infected the epithelium will persist latently and periodically. Previous Morris research supports that in hairy leukoplakia is found 100% Epstein Barr virus (EBV) particles. Research has shown that EBV replication in tongue cells is found only in patients with severe immunosuppression [12].

Frimpong *et al.* studied 6 to 9 types of lesions present in HIV patients: candidiasis, periodontitis, hyperpigmentation, melatonic, gingivitis, xerostomia and hairy leukoplakia. This condition is closely related to the number of CD4 cells in the patient [12, 13]. The results showed the lowest number of CD4 cells found in HIV patients with hairy leukoplakia, where the CD4 cell count was 138.8 cells/mm^3. CD4 count is used as a marker to identify the progression of HIV and stage of immune suppression. Decreasing of CD4 cell count means increasing stage of immunosuppression, allowing for an increasing Epstein Barr Virus (EBV) replication and resulting in hairy leukoplakia [12]. Observational studies of several authors suggest that OHL patients develop into AIDS in a short period of time. This emphasizes the importance of OHL as an early indicator of immunosuppression and the progression of AIDS.

HAART administration may trigger a decrease in viral load and increase CD4, thus helping to significantly reduce the prevalence of OHL patients. Systemic antiviral therapy of herpes provides rapid recovery although recurrence can sometimes occur when therapy is stopped. The combination of HAART and systemic antiviral therapy give satisfactory results of therapy and prevention of recurrence [10].

CONCLUSION

Based on results about oral hairy leukoplakia manifestations associated with CD4 cell counts in HIV/AIDS patients it can be concluded that low CD4 cell counts are associated with presence of oral hairy leukoplakia manifestations.

ACKNOWLEDGMENT

We thank our colleagues from Faculty of Dentistry Hasanuddin University who provided insight and expertise that greatly assisted the research, although they may not agree with all of the interpretations/conclusions of this paper.

AUTHORS CONTRIBUTIONS

All the authors have contributed equally

CONFLICT OF INTERESTS

There are no conflict of interest in this study

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