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Oral Manifestations Associated with Human Immunodeficiency Virus

Ivan Minic*
Department of Periodontology and Oral Medicine, Medical Faculty, University of Nis, Serbia

***Corresponding author:** Ivan Minic, Department of Periodontology and Oral Medicine, Medical Faculty, University of Nis, Serbia

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Abstract

Human immunodeficiency virus (HIV) infection is an ever-growing pandemic that can lead to Acquired Immunodeficiency Syndrome (AIDS) in later stages. Oral lesions are common in patients infected by the HIV virus and may indicate an impairment in the patient's general health status and consequently a poor prognosis. Many of these HIV-positive patients present manifestations involving the maxillofacial region in all stages of the disease, and, in some cases, the oral lesions are the first signs of infection. The various oral manifestations can be categorized into: Infections: bacterial, fungal, viral; neoplasms: Kaposi's sarcoma, non-Hodgkin's lymphoma; immune mediated: major aphthous, necrotizing stomatitis; others: parotid diseases, nutritional, xerostomia and oral manifestations as adverse effects of antiretroviral therapy. Oral candidiasis and oral hairy leukoplakia is "the most commonly occurring changes in the oral cavity in people suffering from side effects. Oral lesions that are associated with this disease are important, since they affect the quality of life of the patient and are useful markers of disease progression and immunosuppression.. Almost all patients with HIV infection will contract oral diseases. Guidelines for recognizing, diagnosing, and managing these conditions are presented. Most conditions can be treated or alleviated through the combined efforts of the physician and the dentist.

Keywords: HIV infection; Oral manifestation; Oral candidiasis; Hairy leukoplakia

Introduction

Human immunodeficiency virus (HIV) infection is an ever-growing pandemic that can lead to Acquired Immunodeficiency Syndrome (AIDS) in later stages [1]. Over 5 million people continue to be newly infected with HIV every year, despite advances in understanding the factors that drive the epidemics. HIV prevalence is increasing worldwide because people on antiretroviral therapy are living longer, although new infections decreased from 3·3 million in 2002, to 2·3 million in 2012 that 2018 would be even smaller [2].

Patients with Human Immunodeficiency Virus (HIV) infection often develop multiple complications and comorbidities. Opportunistic infections should always be considered in the evaluation of symptomatic patients with advanced HIV/AIDS, although the overall incidence of these infections has decreased [3]. The immune system is gradually disrupted. HIV kills cells in the lymph nodes (small glands filled with immune cells that trap foreign organisms) and in other sites. This throws the immune system out of balance. Virus levels in the blood and the lymph nodes increase because the immune system cannot keep up with the amount of virus constantly produced. HIV constantly changes itself, avoiding attack by the antibodies and immune cells that normally control infections [4].

Oral lesions are common in patients infected by the HIV virus and may indicate an impairment in the patient's general health status and consequently a poor prognosis. Many of these HIV-positive patients present manifestations involving the maxillofacial region in all stages of the disease, and, in some cases, the oral lesions are the first signs of infection [5-6].

Moreover, some authors state that oral manifestations are the earliest sign of HIV infection. The various oral manifestations can be categorized into: Infections: bacterial, fungal, viral; neoplasms: Kaposi's sarcoma, non-Hodgkin's lymphoma; immune mediated: major aphthous, necrotizing stomatitis; others: parotid diseases, nutritional, xerostomia and oral manifestations as adverse effects of antiretroviral therapy [7]. Oral candidiasis and oral hairy leukoplakia is "the most commonly occurring changes in the oral cavity in people suffering from side effects [8-10].

Oral candidiasis

Oral candidiasis can be considered as one of the first side signs. It can vary from an acute form that involves the entire oral cavity, and even other parts of the digestive system, to a chronic form with discrete changes, only on certain parts of the oral cavity [11,12]. The first step in the development of a candida linfection is colonization of the mucocutaneous surfaces [13]. HIV infection is not only associated with increased colonization rates but also with the development of overt disease. During the course of HIV infection, the rate of Candida infection is inversely related to the CD4 counts of the patient which in turn depends on the use of Anti-retroviral treatment [14].

Oral conditions are usually diagnosed by appearance and symptoms. It can be confirmed by scraping a sore and checking it under a microscope. Further lab tests are usually performed if the infection does not clear up after treatment. Although oral candidiasis

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can occur at any stage of HIV infection, it is most common in patients with low CD4 counts [15].

Numerous oral and systemic therapies are used to treat oral candidiasis, the most popular of which are nystatin (topical), clotrimazole (topical), ketoconazole (systemic), fluconazole (systemic), and itraconazole (systemic) [16].

Oral hairy leukoplakia

Oral hairy leukoplakia (OHL) is more common among HIV-infected adults than among HIV-infected children. The reported prevalence of OHL in adults is about 20%-25%, increasing as the CD4+ lymphocyte count decreases, whereas in children the prevalence is about 2%-3% [17]. The presence of OHL is a sign of severe immunosuppression. OHL is a significant predictor of HIV disease progression in adults. Although its etiology is not clear, OHL seems to be caused by Epstein-Barr virus infection. In people living with HIV, OHL can occur at any CD4 count, but it is most common among people with less than 200 CD4s. OHL can also occur in HIV-negative people. More than 1 out of 4 people with HIV will develop OHL at some point during the course of their infection. It is most common among men and smokers [18-19].

Oral hairy leukoplakia refers to a white patch that forms in the mouth. These patches usually appear along the sides of the tongue, or on the top and underside of the tongue or along the inside of the cheek. They may appear shaggy or contain a number of tiny folds or ridges. OHL can look like thrush, a similar oral condition that occurs in people with HIV [20]. However, thrush usually comes off when it is lightly scraped with a toothbrush, while OHL does not. These patches do not usually cause discomfort and generally do not affect the taste of foods or liquids. In some cases, however, OHL can cause mild pain and may alter a person's taste buds and sensitivity to food temperatures [21].

The most reliable diagnosis is taking samples to prove presence Epstein-Barr virus. OHL usually does not require treatment or cause serious symptoms. However, treatment is an option for people who are unhappy with the white patches on their tongues or for those with many lesions who are experiencing discomfort or altered taste because of the patches [22].

Antiviral medications, taken by mouth, are used to treat OHL. These are usually taken for 1-2 weeks or until the OHL patches have disappeared: Acyclovir (Zovirax): Acyclovir has been used for many years for OHL. It rarely causes side effects. The usual dose is 800 mg taken five times a day for at least a week. Taking lower doses over time can help prevent OHL from coming back for those with a history of frequent recurrences [23-24].

Kaposi's sarcoma

Kaposi's sarcoma (KS) is a type of cancer in which patches of abnormal tissue grow under the skin or mucous membranes in the mouth, nose, and anus [25].The cancer can also involve the lungs, GI tract, and other organs. Kaposi sarcoma tumors usually manifest as bluish-red or purple bumps. It is common for the lesions to first appear on the feet, ankles, thighs, arms, hands, face, or other parts of the body, but they can also occur on sites inside the body. Other symptoms of the disease may include bloody sputum and shortness of breath [26].

Oral KS (OKS) most often affects the hard and soft palate, gingiva, and dorsal tongue with plaques or tumors of coloration ranging from non-pigmented to brownish-red or violaceous. Its involvement ranges from an incidental finding to proliferative tumor formation that interferes with mastication [27].

OKS needs to be distinguished clinically from other entities, including pyogenic granuloma, hemangioma, bacillary angiomatosis, and gingival enlargement caused by cyclosporine, a drug frequently used in recipients of organ transplantation [28-29].

Before the AIDS epidemic, Kaposi sarcoma was rare, progressed slowly, and was mainly seen in older men, organ transplant patients, or African men. In patients with AIDS, the cancer moves quickly and can be deadly; in these individuals, the disease is caused by an interaction between HIV, a weakened immune system, and the human herpesvirus-8 (HHV-8). People who have organ or kidney transplants also have an increased risk of Kaposi sarcoma [30].

Treatment of Kaposi sarcoma is dependent on how much the immune system is weakened, the number and location of tumors, and symptoms. Options for treatment include antiviral therapy if AIDS is present, combination chemotherapy, cryotherapy, or radiation therapy. Many patients experience tumor recurrence even after being treated [31].

Conclusion

Oral lesions that are associated with HIV infection are important, since they affect the quality of life of the patient and are useful markers of disease progression and immunosuppression. Almost all patients with HIV infection will contract oral diseases. Guidelines for recognizing, diagnosing, and managing these conditions are presented. Most conditions can be treated or alleviated through the combined efforts of the physician and the dentist.

References

1. Sharp PM, Hahn BH. Origins of HIV and AIDS pandemic. Cold Spring Herb Perspect Med. 2011; 1: 006841.
2. Centers for Disease Control and Prevention (CDC) Voluntary medical male circumcision. MMWR Morb Mortal Wkly Rep. 2013; 62: 953-957.
3. Birrell PJ, Gill ON, Delpech VC. HIV incidence in men who have sex with men in England and Wales 2001-2010. A nationwide population study. Lancet Infect Dis. 2013; 13: 313-18.
4. Beyrer C, Sullivan P, Sanchez J, Baral SD, Collins C, Wirtz AL, Altman D, Trapence G , et al. The global HIV epidemics in MSM time to act. AIDS. 2013; 27: 2665-2678.
5. Bajpai S, Pazare AR. Oral manifestations of HIV. Contemp Clin Dent. 2010; 1: 1-5.
6. Nokta M. Oral manifestations associated with HIV infection. Curr HIV/AIDS Rep. 2008; 5: 5-12.
7. Gonçalves LS, Gonçalves BM, Fontes TV. Periodontal disease in HIV-infected adults in the HAART area. Clinical immunological and microbiological aspects. Arch Oral Biol. 2013; 58: 1385-1396.
8. Patton LL, Ramirez-Amador V, Anaya-Saavedra G, Nittayananta W, Carrozzo M, Ranganathan K. Ranganathan K. Urban legends series. Oral manifestations of HIV infection. Oral Dis. 2013; 19: 533-550.
9. Lortholary O, Petrikkos G, Akova M, Arendrup MC, Arikan-Akdagli S, Bassetti M, et al. ESCMID guideline for the diagnosis and management of Candida diseases. Patients with HIV infection and AIDS. 2010; 82: 68-77.
10. Nascimento FG1, Tanaka PY. Thrombocytopenia in HIV Infected Patients.

Indian J Hematol Blood Transfus. 2012; 28: 109-111.

11. Ling Y, Cypowyj S, Aytekin C, Galicchio M, Camcioglu Y, Nepesov S . et al. Inherited IL-17RC deficiency in patients with chronic mucocutaneous candidiasis. J Exp Med. 2015; 212: 619-631.

12. Whibley N, Gaffen SL. Brothers in arms. Th17 and Treg responses in Candida albicans immunity. PLoS Pathog. 2014; 10: e1004456.

13. Manches O, Frleta D, Bhardwaj N. Dendritic cells in progression and pathology of HIV infection. Trends Immunol. 2014; 35: 114-122.

14. Liu Y, Yang B, Zhou M, Li L, Zhou H, Zhang J, et al. Memory IL-22-producing CD4+ T cells specific for Candida albicans are present in humans. Eur JImmunol. 2009; 39: 1472-1479.

15. Gosselin A, Monteiro P, Chomont N, Diaz-Griffero F, Said EA, Fonseca S. et al. Peripheral blood CCR4+CCR6+ and CXCR3+CCR6+CD4+ T cells are highly permissive to HIV-1 infection. JImmunol. 2010; 184: 1604-1616.

16. Kim CJ, McKinnon LR, Kovacs C, Kandel G, Huibner S, Chege D. et al. Mucosal Th17 cell function is altered during HIV infection and is an independent predictor of systemic immune activation. JImmunol. 2013; 191: 2164-2173.

17. Bravo IM, Correnti M, Escalona L, Perrone M, Brito A, Tovar V, Rivera H. Prevalence of oral lesions in HIV patients related to CD4 cell c ount and viral load in a Venezuelan population. Med Oral Patol Oral Cir Bucal. 2006; 11: E33-E39.

18. Piperi E, Omlie J, Koutlas IG, et al. Oral hairy leukoplakia in HIV-negative patients report of 10 cases. Int J Surg Pathol. 2010; 18: 177-183.

19. Nokta M. Oral manifestations associated with HIV infection. Curr HIV/AIDS Rep. 2008; 5: 5-12.

20. Greenspan JSD, Greenspan J. Webster-Cyriaque Hairy leukoplakia lessons learned 30 plus years. Oral Diseases. 2016; 22: 120-127.

21. Moura MDG, Grossmann SDMC, Fonseca LMDS, Senna MIB, Mesquita RA. Risk factors for oral hairy leukoplakia in HIV-infected adults . Journal of Oral Pathology and Medicine. 2006; 35: 321-326.

22. Khammissa RAG, Fourie J, Chandran R, Lemmer J, Feller L. Epstein-Barr virus and its association with oral hairy leukoplakia. a short review. International Journal of Dentistry. 2016; 2016: 6.

23. Slots J, Saygun I, Sabeti M, et al. Epstein-Barr virus in oral diseases. J Periodontal Res. 2006; 41: 235-244.

24. Chambers AE, Conn B, Pemberton. Twenty-first-century oral hairy leukoplakia a non-HIV-associated entity. Oral Surg Oral Med Oral Pathol Oral Radiol. 2015; 119: 326-332.

25. Dal Maso L, Polesel J, Ascoli V, Zambon P, Budroni M, Ferretti S, et al. Classic Kaposi's sarcoma. Br J Cancer. 2005; 92: 188-193.

26. Dilnur P, Katano H, Wang ZH , Osakabe Y, Kudo M, Sata T, et al. Classic type of Kaposi's sarcoma and human herpesvirus 8 infection in Xinjiang China. Pathol Int. 2001; 51: 845-852.

27. Mohanna S, Ferrufino JC, Sanchez J, Bravo F, Gotuzzo E. Epidemiological and clinical characteristics of classic Kaposis sarcoma in Peru. J Am Acad Dermatol. 2005; 53: 435-441.

28. Mohanna S, Maco V, Gown A, Morales D, Bravo F, Gotuzzo E. Is Classic Kaposi's Sarcoma Endemic in Peru. Report of a case in an indigenous patient. Am J Trop Med Hyg . 2006; 75: 324-326.

29. Mohanna S , Sánchez J , Ferrufino JC, Bravo F, Gotuzzo E. Lymphangioma-like Kaposi's sarcoma report of four cases and review. J Eur Acad Dermatol Venereol . 2006; 20: 1010-1011.

30. Shetty K. Management of oral Kaposi's sarcoma lesions on HIV-positive patient using highly active antiretroviral therapy. Case report and a review of the literature. Oral Oncology. 2005; 41: 226-229.

31. Opariuc O, Napoli S, Zegarelli DJ, Yoon AJ. Recurrent and Multicentric non-HIV related Kaposi's Sarcoma. Columbia Dental Review. 2008; 12: 15-18.