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HIV INDUCED ORAL HAIRY LEUKOPLAKIA - A CASE REPORT

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ABSTRACT

Oral Hairy leukoplakia was first described in 1984 and is strongly associated with human immunodeficiency virus disease, although some cases have been reported in human immunodeficiency virus negative individuals. After the introduction of Highly Active Antiretroviral Therapy, the prevalence of Oral Hairy leukoplakia in human immunodeficiency infected patients has declined virus significantly. Oral Hairy leukoplakia has been convincingly shown to be related to Epstein-This report describes a case Barr virus. wherein human immunodeficiency virus was diagnosed by a dentist due to the presence of white lesions seen on the tongue. The report emphasizes the need for thorough personal history and clinical examination from every general dentist.

KEYWORDS: Oral hairy leukoplakia; human immunodeficiency virus; highly active antiretroviral therapy; Epstein-Barr virus

INTRODUCTION

Oral hairy leukoplakia (OHL) is a viral infection caused by Epstein-Barr virus (EBV), which is a double-strand DNA virus belonging to the human herpes virus group.^[1] Snijders *et al.*,^[2] in 1990 proposed EBV as the etiologic agent of oral hairy leukoplakia. After the primary EBV infection has resolved, the virus stays latent in the B cells and causes lytic infection in the oropharynx, controlled by the immune system. Uncontrolled lytic infection in the oropharynx is manifested as oral hairy leukoplakia in immunocompromised hosts. The lesion is strongly related to Human immuno-deficiency virus (HIV) disease and was first described by Greenspan et al in 1984 as an oral manifestation in male homosexual HIVpositive patients.^[3] Accurate diagnosis is important because it may be an early indicator of an undiagnosed HIV infection. Moreover, a diagnosis of OHL may be of prognostic value.^[4] Here in, we describe a case of OHL in an HIV patient, whose HIV status was unknown at the time of OHL diagnosis.

CASE REPORT

A 31 year old male patient reported to the Department of Oral medicine and radiology, with the chief complaint of first episode of pain in the lower left back tooth region since 2-3 days, which aggravated on opening the mouth and relieved on medication (Diclofenac+ paracetamol). He suffered with an unknown fever 2 years back for which he was on an intravenous drip in a local chemist place. The personal history revealed his homosexual relationship with a partner for the last 3-4 years and reported that he had never used any sort of precautions before. The patient was unmarried and reported a sudden loss of weight and a habit of smoking 4-5 cigarette per day since 12 years. All the vital signs were within normal limits. Examination of the face (Fig. 1) showed asymmetry due to a diffuse swelling on the left lower third of the face, measured approximately 3x4 cm, extended from the infraorbital margin to 1 cm above the inferior border of the mandible superoinferiorly and anteroposteriorly from the nasolabial fold to 2 cm away from the ear lobule. The surface of the swelling appeared normal with no signs of active bleeding or draining sinus. On palpation, swelling was tender, soft in consistency and the soft tissue overlying the swelling was movable. A solitary left submandibular lymph node was palpable measuring approximately 1x1 cm, soft in consistency, movable and tender.



Fig. 1: Extra oral picture of the patient



Fig. 3: Vertical keratotic folds oriented as a palisade



Fig. 5: Partially healed lesions after 15 days

Examination of the oral cavity suggested a mouth opening of approximately 3.6 cm. Full complements of the teeth were present with generalized stains and dental caries w.r.t 36, 37, 47. There was partially erupted tooth w.r.t 38 with the pericoronal flap on the distal aspect which was severely inflamed. The soft tissues appeared to be normal except for a diffuse, elevated, vertical, white keratotic folds oriented as a palisade seen along the lateral borders of the tongue on both the sides, extended from the mesial aspect of the first premolar region to the distal aspect of the first molar region. The lesion had wrinkled or corrugated surface appearance with no signs of erythema, ulceration or any discharge. On palpation, the lesion was nontender, firm in consistency, non-movable and non-scrapable (Fig. 2 & Fig. 3). Based on the history and clinical examination, a provisional diagnosis of Pericoronitis w.r.t 38 and Hairy Leukoplakia on the tongue was considered. The

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Fig. 2: Vertical keratotic folds oriented as a palisade



Fig. 4: Partially healed lesions after 15 days patient was referred to HIV counselor for the pretest counseling and then testing where the patient was subjected to routine blood investigation, CD4 count, HIV tests such as COMB AIDS, Triline and Trispot. The routine blood investigation revealed normal values but the CD4 count was reduced to 223 cells/mm³ (normal: 500-1200 cells/mm³) and the HIV tests showed a positive result for HIV 1. On this basis, a final diagnosis of HIV induced Hairy Leukoplakia was established. The patient was then referred to the ART department of St.John's Hospital, Bangalore. A complete medical work up was done and highly active antiretroviral therapy (HAART) was initiated which included cotrimoxazole, zidovudine (300mg) + lamciclovir (150mg) + NVP (200mg) twice daily for an initial period of 15 days. The dosages of the medications were titred according to the routine blood investigations done at every appointment. On follow up after 15 days of HAART therapy, the lesion had partially resolved (Fig. 4 & Fig. 5). **DISCUSSION**

Oral hairy leukoplakia is considered as one of the most common oral disorder in a person with HIV, infected with HIV, accounting for approximately 15 to 20% of affected population. OHL is characterized by an asymptomatic flat and corrugated white plaque that is non- scrapable.^[3] The most common site of OHL is the lateral border of the tongue, but may also be observed on

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the dorsum, ventral surface and buccal mucosa. The relationship between OHL and the depletion of the immune system in HIV positive patients is well documented and accepted, suggesting a potential prognostic significance of OHL.^[5] Person with OHL generally have moderate to advanced immune suppression, with a median CD4 count of approximately 235 cells/mm³. The estimated adult HIV prevalence in India was 0.32% in 2008 and 0.31% in 2009. The total number of People Living with HIV (PLHIV) in India is estimated at 2.4 million with uncertainty bounds of 1.93 to 3.04 million in 2009. The percent distribution of HIV infection by age is estimated at 4.4% among children below the age of 15 years, 82.4% among adults aged 15 to 49 years and the remaining 13.2% among people over 50 years of age. South Indian states such as Andhra Pradesh, Maharashtra, Karnataka and Tamil Nadu mentioned in descending order account for 57% of all HIV infections in the country. At national level, HIV prevalence is highest amongst the injecting drug users (IDU) at 12.22% followed by men who have sex with men (MSM) at 6.82% and female sex workers (FSW) at 5.92%. In one study, patients with OHL had a 48% probability of developing AIDS within 16 months and 83% within 31 months.^[6] According to the WHO clinical staging of HIV disease, OHL is observed in clinical stage 3 with advanced symptoms such as unexplained moderate malnutrition, unexplained persistent diarrhoea (14 days or more), unexplained persistent fever (above 37.5°C intermittent or constant, for longer than one month), persistent oral candidiasis, acute necrotising ulcerative gingivitis or periodontitis, lymph node tuberculosis, pulmonary tuberculosis. According to CDC classification system for HIV infected individuals, OHL is listed as a B category condition.^[7] In general, the presence of OHL strongly suggests a diagnosis of HIV, but described rare reports have OHL in immunosuppressed persons with disorders other than HIV, including solid organ and hematopoietic cell transplantation, systemic lupus erythematosus, Behcet's disease, and immune suppression secondary to high-dose oral corticosteroids.^[4] OHL is most often confused with oral candidiasis, but OHL lesions are strongly adherent and cannot be removed by scraping with a tongue blade, whereas plaques

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associated with candidiasis are usually easily removable. In addition, candidiasis is typically seen on the buccal mucosa, the palate, and the gingiva rather than on the lateral aspect of the tongue. Hairy tongue (hyperplastic papillae) generally does not resemble OHL, but can be confused with OHL because of the similarity of the names of the two disorders. Hairy tongue is caused by hyperplasia of the filiform papillae concomitant with a decrease in the normal rate of desquamation, resulting in a thick, matted, hairy dorsal surface of the tongue.^[7] Fraga-Fernadez et al.,^[8] in 1990 described for the first time the histopathologic criteria for OHL diagnosis. In this report, three distinct nuclear changes such as Cowdry- A inclusions, ground glass nuclei and nuclear beading was described as the main features for OHL diagnosis. According to WHO, in most circumstances the diagnosis of OHL is made on clinical findings and a biopsy of the lesion is typically not performed. OHL usually does not generally require specific therapy. In addition, most patients will experience gradual of OHL after resolution taking potent antiretroviral therapy. The patient in the present case also showed a significant resolution in the lesion after 15 days of anti-retroviral therapy. Before the advent of highly active antiretroviral therapy (HAART), OHL was a frequently observed oral manifestation of HIV in 80% of the cases. After HAART, a significant decline in the prevalence of OHL has been observed and the current prevalence is estimated to be between 2.8% to 26.6%. Recurrence is often noted with discontinuation of therapy.^[9]

CONCLUSION

We are living in an age where every fourth patient we encounter may be affected with HIV. As dentists we are constantly in contact with the oral mucosa filled with fluids and blood. It becomes imperative that we follow universal precautions for every procedure. It is also of vital importance for the clinician to be aware of the more common oral manifestations associated with HIV infection. **CONFLICT OF INTEREST & SOURCE OF FUNDING**

The author declares that there is no source of funding and there is no conflict of interest among all authors.

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