A SMALL REVIEW OF ORAL FEATURES IN PAEDIATRIC AIDS

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Abstract:
Oral manifestations are often among the 1st symptoms of HIV/AIDS and thus can be useful in early detection of the disease. The distribution of some of the specific oral manifestations are reported to differ between adults and children HIV.As the oral manifestations are among the earliest and most important indicators of HIV infection, a better understanding of these manifestations in children is a must for all dental health care workers.

Keywords: HIV; AIDS; Children; Oral; Paediatric; Orofacial.
DOI Number: 10.48047/nq.2023.21.7.nq23102 **NeuroQuantology 2023;21(7):1138-1142**

Introduction

Oral manifestations have been linked to immunological suppression and are frequently among the first symptoms experienced by patients infected with the human immunodeficiency virus (HIV).[1,2] It has been noted that there are differences in the distribution of certain oral symptoms between adults and children.[3] The oral symptoms of oral hairy leukoplakia and oral candidiasis in particular are clinical indicators of the advancement of acquired immunodeficiency syndrome (AIDS) and are typically linked to high viral load levels and a CD4+ T-lymphocyte cell count of less than 200 cells/µl of blood in patients.[4,5] The prevalence of oral manifestations in HIV-infected adults tends to vary from country to country. Previous studies, at least in Africa, showed a wide range of prevalence rates from 1.5% up to 94%.[6] However, in HIV-infected children, the prevalence of oral manifestations in developed countries has been reported to be as high as 72%.[7,8] Comparable studies in children from developing countries, including Africa, indicated variations in the occurrence of oral manifestations, for example 61% in Brazil,[9] 55% in Romania,[10] 49% in Thailand[11] and 63% in South Africa.[12]

The mid-1990s saw the emergence of highly active antiretroviral medication (HAART), which had a significant impact on HIV infection clinical prospects and had therapeutic effects. Since the advent of HAART, the rate of oral symptoms due to HIV has decreased.[13] In a prospective study, Schmidt-West-hausen et al[13] reported a decrease in the prevalence of soft tissue oral lesions from 34.4% to 8.2% six months following HAART. Previous studies have not reported a specific trend in the association between HAART and dental caries. In a cohort of United States HIV-infected patients receiving HAART, the prevalence of dental caries was reported to be less compared with a group not taking HAART,[14] whereas other reports suggested a positive correlation between HAART and caries prevalence.[15] Furthermore, in a recent Brazilian study[16] on HIV-infected children, oral manifestations were reported to be clinical predictors of HAART failure. In Uganda, the assessment of oral manifestations in HIV-infected adults showed prevalence rates ranging from 42% to

72%. [16] The only study involving Ugandan HIV-infected children, the majority of whom were on various medications including HAART, reported a prevalence of 68.6% dental caries, 40% gingivitis and 8.6% pseudomembranous candidiasis.[1]

Classification of oral lesions

Consensus classification of orofacial lesions associated with paediatric HIV infection [6]:

GROUP 1: lesions commonly associated with paediatric HIV infection

- Candidiasis
  a) Erythematous
  b) Pseudo membranous
  c) Angular cheilitis
- Herpes simplex virus infection
- Linear gingival erythema
- Parotid enlargement
- Recurrent aphthous ulcers
  a) Minor
  b) Major
  c) Herpetiform

GROUP 2: lesions less commonly associated with paediatric HIV infection

- Bacterial infections of oral tissues
- Periodontal diseases
  a. Necrotizing (ulcerative) gingivitis
  b. Necrotizing (ulcerative) periodontitis
  c. Necrotizing (ulcerative) stomatitis
- Viral infections
  a. Cytomegalovirus
  b. Human papilloma virus
  c. Molluscum contagiosum
  d. Varicella zoster virus
  - Herpes zoster
  - Varicella
  - Xerostomia

GROUP 3: lesions strongly associated with HIV infection but rare in children

- Neoplasm
- Kaposi's sarcoma and non-Hodgkin's lymphoma

Diagnostic evaluation of HIV-exposed infant

All infants born to HIV-infected women should be evaluated regularly until definitive determination is made regarding their HIV status.4 Maternal antibodies to HIV is passively transferred to the neonate in virtually all children born to seropositive women; however, only about one-third of these infants are infected. The preferred method for diagnosing HIV infection in infants is by HIV culture or PCR testing.[4]

Viral culture is performed on peripheral blood mononuclear cells that are co-cultured with uninfected mononuclear cells to promote HIV growth and detect latent HIV-infected cells by stimulating viral replication. The HIV DNA polymerase chain reaction assay facilitates the detection of minute amounts of HIV proviral DNA that have become incorporated into the DNA of infected cells. In children and infants older than 18 months definitive diagnosis of

HIV infection is made using the EIA and confirmatory Western blot assays.[4]

Prognosis In Paediatric Patients

There are two general patterns of presentation of HIV infection in children. The first pattern

representing about one-third of all perinatally acquired infections is a fulminant **c**ourse of illness represented by early onset of severe disease with rapid progression and poor prognosis. Rapid progressors are infants with early onset of disease manifestations who die by the age of 4 years. Infants in this group usually have severe opportunistic infections, encephalopathy, or both within the first 2 years of life. Many of these children become identified as being infected with HIV because of severe illness that arises abruptly.[4]

The second pattern of paediatric HIV infection involves a later onset of disease symptoms and is associated with a better prognosis. These children generally are seen after the first year of life with a more indolent disease course, consisting of a variety of the more general clinical manifestations. These children more often have a diagnosis of lymphoid interstitial pneumonitis and manifest other signs of lymph proliferation such as generalized lymphadenopathy and parotitis.[4]

Two major clinical factors that appear to

affect the prognosis in children with HIV infection are the specific HIV-related disease that they develop and their age at presentation of the disease.

Oral Features

Several oral lesions have also been associated with HIV seropositivity in children but the incidence progression and prognostic implications are not yet well understood.4 Oral lesions strongly associated with HIV infection in adults but less common in children include Kaposi's sarcoma, non-Hodgkin lymphoma, and oral hairy leukoplakia.[7]

Bacterial infections

Bacterial infections have been noted at many sites in HIV-infected children and are often recurrent.5 Several children have HIV-associated gingivitis associated with both the primary and permanent dentition. The lesion is characterized by linear erythema of the facial and interproximal gingival margins and is unresponsive to improved oral hygiene. Punctate diffuse erythema may involve gingival and mucosa. In children particularly in the primary dentition, it may be localized or generalized. In adolescents the more generalized form seems to occur, comparable to the lesion seen in adult patients.[3] Other common infections include cellulitis and abscesses caused by Staphylococcus and Streptococcus most frequently identified in children with HIV disease and is often implicated in pneumonia and bacteremia.4 In developed countries acute necrotizing ulcerative gingivitis has seldom if ever been reported in children under 10 years of age.[3]

Fungal infections

Candidiasis the most common HIV-related oral lesion is candidiasis, predominantly due to Candida albicans. While Candida can be isolated from 30–50% of the oral cavities of healthy adults, making it a constituent of the normal oral flora, clinical oral candidiasis rarely occurs in healthy patients. Based on clinical appearance, oral candidiasis can appear as one of four distinct clinical entities: erythematous or atrophic candidiasis, pseudomembranous candidiasis, hyperplastic or chronic candidiasis, and angular cheilitis.[9,17] In all cases, the infection is superficial. While in most instances the clinical appearance is adequate to arrive at a diagnosis, simple exfoliative cytology will identify the characteristic budding yeast and hyphae when the clinical diagnosis is uncertain.[17,18]

The clinical type, distribution, and severity of the infection all affect how oral candidiasis is treated. Topical therapy works well for small, easily accessible lesions. Nystatin oral suspension, nystatin pastilles, and clotrimazole troches are useful for treating mild-to-moderate erythematous and pseudomembranous candidiasis. However, because of the fermentable carbohydrate substrates, prolongation can cause serious tooth caries. Antifungal drugs classified as triazole (fluconazole and itraconazole) and imidazole (ketoconazole) are used in systemic treatment for oral candidiasis. [19]

Ketoconazole is hepatotoxic and requires gastric acid for absorption, thereby limiting its usefulness in patients with HIV infection who may also have developed gastric achlorhydria. Fluconazole is an excellent systemic antifungal medication with a favorable therapeutic index, making it the preferred systemic antifungal medication. Itraconazole (100 mg tablet,1–2 tabs/day) is another excellent systemic antifungal for use alone, or in combination with fluconazole (100 mg tablet, 1–2 tabs/day), for resistant candidiasis. Therapy should be continued for 2 weeks until clinical infection is eliminated.[19]

Viral infections

Children living in lower socio-economic situations are at higher risk of getting HSV.[1] Recurrent episodes of severe ulcerative herpetic lesions develop in 5% to 10%.4 This causes both oral lesions and systemic manifestations. The illness is acute with varying degrees of fever and malaise, cervical lymphadenopathy, and perioral and intraoral lesions. The lesions appear as crater-like

ulcers with well-defined raised white borders and have a gray-white pseudomembrane.3HSV-1 can also cause esophagitis with pain on swallowing, encephalitis, and widely-disseminated disease in the liver, spleen, adrenal glands, lungs, kidney and brain.[4]

Herpes zoster is another herpes virus and can produce oral ulcerations usually accompanied by characteristic skin lesions.[3,19]

Conclusion

Clinically relevant and often occurring oral disorders are linked to HIV illness and are part of this complex of diseases. Most common lesions can be easily detected with a thorough examination of the oral cavity, and an experienced medical or dental professional with a good understanding and knowledge of these conditions can herald the treatment in such patients if they are undiagnosed or may require a change in treatment (be more aggressive) of a known patient living with HIV/AIDS. Comprehending the identification, importance, and management of these lesions is crucial for the long-term assessment and welfare of individuals living with HIV/AIDS.

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