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Review Article

AIDS and its Impact on Oral Health: A Review

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Abstract

Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus (HIV) and is characterized by suppression of the immune system of the body. This immunosuppression leads to a scale of other diseases that include opportunistic infections, and secondary neoplasms. The major impact of HIV infections is on the immune system. A characteristic feature of AIDS is the great immunosuppression of cell mediated immunity. Seven cardinal signs of HIV infection have been internationally identified are oral candidiasis, hairy leukoplakia, Kaposi sarcoma, necrotizing ulcerative gingivitis, necrotizing ulcerative periodontitis, linear gingival erythema, and non-Hodgkin lymphoma. Oral lesions in HIV can help in diagnosis as well as prognosis of HIV diseases to AIDS and different stages in therapy and vaccine trials. It is of utmost importance to maintain oral health of the patients with HIV/AIDS. Oral physician should always aim towards prevention, diagnosis, treatment, and control of these oral manifestations in HIV/AIDS.

Keywords: HIV; AIDS; Oral Manifestations; Immunodeficiency

Introduction

Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus (HIV) and is characterized by suppression of the immune system of the body. This immunosuppression leads to a scale of other diseases that include opportunistic infections, and secondary neoplasms [1]. Immunosuppression most importantly occurs due to progressive deterioration by and large of cell mediated immunity. HIV is successor of simian immunodeficiency virus (SIV), further classified as HIV-1 and HIV-2 based on genetics. HIV 1 is classified into four groups: M, N, O, and P [1]. HIV-1 group M forms collectively account for most of the (94%) of human infections. HIV-2 tends to follow a more indolent clinical course and rarely occurs outside Africa [2].

The human immunodeficiency virus is a virion having an regular pulyhedron structure that contains numerous external spikes formed by two major envelope proteins, the external gp120 and the transmembrane gp41. The matrix of the virion is formed by core protein p17 is found outside the nucleoid. The HIV glycoprotein antireceptors (GP-41 and GP-120) makes an attachment on a human cell membrane of their specific receptors. These receptors are CD4, which are typically found on T4 lymphocytes and other white blood cells. A coreceptor called CCR-5 (fusin) permits docking with the host cell and fusion with the cell membrane leading to the formation of syncytia [1].

HIV infection is an infection with the human immunodeficiency virus - an RNA retrovirus whereas acquired immune deficiency syndrome is a term given to a group of disorders characterized by a profound cell mediated immunodeficiency consequential to irreversible suppression of T lymphocytes by the HIV [2].

Epidemiology

The first case of HIV infection was registered in southern India in Chennai in 1986. It was found that the heterosexual route is the common mode of transmission, followed by intravenous drug use. India's prevalence estimates are based according to the National AIDS Control Organization (NACO). Nationwide, an annual HIV sentinel surveillance was first organized in 1994, and later in 2007. Coverage and outreach of the HIV sentinel sites has been improving each year.

In 2019 overall HIV estimation report- The estimated adult (15-49 years) HIV prevalence trend has been declining in India since the epidemic's peak in the year 2000 and has been stabilizing in recent years. The estimate for this indicator in 2019 was 0.22% (0.17-0.29%) [3]. HIV prevalence among adult males (15-49 years) was estimated at 0.24% (0.18-0.32%) and among adult females at 0.20% (0.15-0.26%) [3].

Transmission of HIV

- **Horizontal Transmission**: The virus is most commonly acquired by sexual route with an infected partner. The virus can enter the body through the lining of the vagina, vulva, penis, rectum or mouth during sex. The infection can also be transmitted by exchange of infected blood, or other body fluids, and is not transmitted by social or casual, non-sexual contact. Currently, the predominant mode of HIV transmission is heterosexual sex worldwide. Other notable transmission modes include sharing of needles, breast feeding, and transfusion of infected blood or blood products (factor VIII concentrate). Occasional cases of HIV infection resulting from needlestick injuries in health-care settings have also been reported.

- **Vertical Transmission:** From Mother to child during delivery.

Pathogenesis

The major impact of HIV infections are the immune system and the central nervous system. A characteristic feature of AIDS is the great immunosuppression of cell mediated immunity. The first step is binding of the virion to target cell surface after entering into the host. After entering the host cell virus un-coats itself and viral RNA then undergoes reverse transcription into linear double-stranded viral DNA in the cytoplasm of infected cells by the reverse transcriptase. It is then transported to the nucleus, where it is first disperses and then integrated into the host cell. The integrated viral DNA is then again transcribed into full length RNA by the host cell. These RNA transcripts can serve as mRNA for the synthesis of viral gene products. HIV first infects macrophages where it multiplies and is shed, then other cells like lymphocytes and cells that possess the CD4 receptor site are infected [1].

Following the infection, the virus often enters a dormant stage lasting for 2-15 years. When HIV is transmitted, CD4 cells are rapidly infected within a few days followed migration to regional lymph nodes, and then enters the circulation. This leads to widespread dissemination of virus to the brain and the lymphatic system, causing the 'primary HIV infection' or the 'seroconversion syndrome'. The most common signs and symptoms of acute infection include fever, fatigue, headache, lymphadenopathy, pharyngitis, maculopapular rash, weight loss, depression, gastrointestinal distress, night sweats, myalgia, arthralgia, aseptic meningitis, retrorbital pain, and oral or genital ulcers. The acute illness may last from a few days to more than 10 weeks, but usually lasts for less than 13 days. After the initial rise in plasma viremia, there is a marked reduction to a steady-state level of viral replication. The decrease in the viral load during acute HIV-1 infection is probably due to virus specific immune responses that limit viral replication [4].

Antibodies against HIV are produced within 6-12 weeks, these antibodies can be detectable in the blood, and enzyme linked immunosorbent assay (ELISA), and Western blot testing can report seroconversion. HIV can be tested positive at this time and can also be transmitted in body fluids [1].

Clinical stages

Stage 1: Acute primary infection

The initial symptoms of HIV can include: Fever, rash, sore throat, swollen glands, headache, upset stomach, joint aches and pain, muscle pain. These symptoms eventually go by one or two weeks, but mat last for four weeks.

Stage 2: The asymptomatic stage

After the primary stage, seroconversion process occurs and patient starts feeling better. However, the virus is still active in this stage even if this stage is symptomless and these active viruses further infects new cells making copies of itself.

Stage 3: Symptomatic HIV infection

Immune system is severely damaged by this stage. At this stage patients are more prevalent of getting opportunistic infections. Symptoms can include: Weight loss, chronic diarrhoea, fever, persistent cough, night sweats, mouth and skin problems, regular infections, serious illness or disease. If serious opportunistic infections or diseases are developed by the patient- as a result of damage to their immune system from advanced stage 3 HIV infection - they are said to have AIDS [2].

Oral manifestations

The internationally identified and accepted seven cardinal signs of HIV infection are oral candidiasis, hairy leukoplakia, Kaposi sarcoma, necrotizing ulcerative gingivitis, necrotizing ulcerative periodontitis, linear gingival erythema, and non-Hodgkin lymphoma [5]. Oral lesions in HIV can indicate and show early clinical features of HIV infection. Oral lesions can predict progression of HIV diseases to AIDS and entry or end-points in therapy and vaccine trials [6]. Oral Sentinel Lesions are oral mucosal lesions found to indicate underlying immunosuppression (oral candidiasis, hairy leukoplakia, Kaposi sarcoma). Oral hairy leukoplakia is more of indication of progression of HIV.

Fungal infections

Candidiasis

Candidiasis is the most prevalent opportunistic infection affecting the oral mucosa. Species of oral Candida are: *C. albicans, C. glabrata, C. krusei, C. parapsilosis, C. pseudotropicalis, C. tropicalis*. Nearly all of the lesions are caused by Candida albicans. C. albicans is a normal commensal flora which can act as pathogenic organism due to number of predisposing factors. *C. albicans* is a weak pathogen, and it affects the very young, the very old, and the immunocompromised. Most candidal infections affect mucosal linings, but rare systemic manifestations may have a fatal course [6].

Pseudomembranous candidiasis (Thrush)

One of the most common form of candidiasis in HIV infection. The infection typically presents with loosely attached membranes comprising fungal organisms and cellular debris, which leaves an inflamed, sometimes bleeding area when the pseudomembrane is removed [5]. The white plaques grossly resembling milk curds, consists of tangled mass of fungal hyphae with intermingled desquamated epithelium, keratin fibrin, necrotic debris, leukocytes, and bacteria.

Erythematous candidiasis

Clinical appearance of this lesion has a diffuse border, this helps in differentiating between erythroplakia, which usually has a sharper demarcation and often appears as a slightly submerged lesion [6]. Erythematous candidiasis may be considered descendant of pseudomembranous candidiasis but may also emerge *de novo*.

Angular cheilitis

Clinical findings usually found are soreness, erythema, cracks, crusts, pain and fissuring at the corners of mouth. Angular Cheilitis can appear alone or in coexistence with another form of candidiasis [7].

Viral infections

Herpes simplex

Herpes Simplex infection in HIV patients can occur at any site intraorally. Ulcers appears atypical having several centimetres size may last several weeks or months if undiagnosed and untreated. Single ulcer appearing on a nonkeratinized site can be indistinguishable from recurrent aphthous ulcers, except that they may be larger and appear slightly depressed with raised borders [8].

Herpes zoster

Herpes Zoster infection in HIV patients more severe, bilateral, affects multiple dermatone. On rare occasions, it not just affects dorsal root ganglion but also anterior horns leading to paralysis [8].

Human papillomavirus lesions

HPV lesions in the oral cavity may appear as solitary or multiple nodular lesions. They may appear as multiple, smooth-surfaced raised masses resembling focal epithelial hyperplasia or as multiple, small papillomatous or cauliflower-like projections [8].

Epstein barr virus

The most common lesion caused by EBV in HIV patients is Oral hairy leukoplakia (OHL). It appears as corrugated or “hairy” white lesion on the lateral borders of the tongue. It is less common in children than in adults. OHL can occur in about 20% of patients

with asymptomatic HIV infection and becomes more common as the disease progresses (CD4+ T-cell count falls). HIV infection and immunodeficiency both can be indicated by diagnosis of OHL.

Cytomegalovirus

Oral ulcers caused by cytomegalovirus (CMV) have been reported. These ulcers can appear on any mucosal surface and may be confused with aphthous ulcers, necrotizing ulcerative periodontitis (NUP), and lymphoma. CMV ulcers appear necrotic with a white halo unlike aphthous ulcers which usually have an erythematous margin.

Bacterial infections

Tuberculosis

It is most common opportunistic infection in AIDS. Primary lesions (tuberculous ulcer) in oral cavity may appear as single painless ulcer with regional lymph node enlargement. The secondary lesions are more common than primary and are often associated with pulmonary disease [9]. Lesions may appear as single, indurated, irregular, painful ulcer covered by inflammatory exudates.

Periodontal diseases

Periodontal diseases like linear gingival erythema and necrotizing ulcerative periodontitis (NUP) occur even with maintained oral hygiene (very little plaque or calculus to account for the gingivitis). In linear gingival erythema gingiva is red and oedematous with spontaneous bleeding on provocation. In acute-onset ulcerative gingivitis, ulcers starts at the tips of the interdental papilla and may also affect the gingival margins, with severe pain which is often present. Healing of these ulcers occur, leaving the gingival papillae with a characteristic cratered appearance. Necrotizing ulcerative periodontitis occurs as the result of rapid loss of supporting bone and soft tissue. Loss in supporting bone occurs with no formation of gingival pockets, sometimes involving only isolated areas of the mouth [10].

Neoplastic lesion-kaposi sarcoma

Kaposi's sarcoma (KS) can be present intraorally, with or without associated skin lesions. Multifocal vascular tumours can occur with the most common oral site- hard palate, but lesions may occur on any part of the oral mucosa, including the soft palate, gingiva, and buccal mucosa, and in the oropharynx. Maintenance of oral hygiene is important to avoid such complications.

Lymphoma

Soft tissue involvement with or with our involvement of underlying bone can occur any where in oral cavity. The lesion may have vivid presentation as firm, painless swelling that may be ulcerated. Some oral lesions may appear as shallow ulcerations. Oral NHL may appear as solitary lesions with no evidence of disseminated disease.

Other oral lesions

Aphthous stomatitis

Duration of ulcers is about 1-2 weeks, being healed without scarring. Minor aphthous ulcerations are also prevalent in HIV-infected and uninfected populations. Major aphthous ulcers can occur in any area of the oral mucosa and tend to persist for more than three weeks, being healed with scar formation. In HIV-infected patients, aphthous ulcerations have been associated with severe immunosuppression, with CD4 counts below 100 cells/mm^3, being markers of HIV disease progression.

Salivary gland diseases

Salivary gland disease associated with HIV infection can present as xerostomia with or without salivary gland enlargement. The enlarged salivary glands if present are soft but not fluctuant. In some cases, enlarged salivary glands may be due to lymphoepithelial cysts. Xerostomia is sometimes seen in individuals with HIV associated salivary gland disease. HIV-infected patients may also experience dry mouth in association with taking certain medications that can hinder salivary secretion, such as antidepressants, antihistamines, and antianxiety drugs [4].

HIV infection in children

Orofacial lesions commonly associated with HIV infection in children in Europe and North America include oral candidiasis, herpes simplex infection, linear gingival erythema, parotid enlargement and recurrent oral ulceration.

In contrast, oral lesions strongly associated with HIV infection in adults, but less common in children, include Kaposi sarcoma, non-Hodgkin lymphoma and oral hairy leukoplakia.

A high prevalence of concomitant dental diseases, especially caries and periodontal disease has been reported in HIV-positive children.

Conclusion

Recognition of Oral lesions with HIV is very important by a dental physician for diagnosis and progression of the disease and even for self-protection. It is imperative to integrate continuous and careful medical care of oral health as a part of the treatment for people with HIV/AIDS. The objectives of every dental health professional should be the prevention, diagnosis, treatment, and control of these oral manifestations.

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