



## HIV AND DENTISTRY: BRIEF REVIEW

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### ABSTRACT

In patients with HIV infection oral health status has a central role. After the discovery of acquired immune-deficiency syndrome (AIDS) in late 1980s, the spread of human immunodeficiency virus (HIV) has presented a global developmental and public health threat and is still spreading rapidly. Since oral lesions are quite common in HIV infected patients, dentists are the most ideal persons to identify, manage and

treat HIV-associated oral manifestations and have a responsibility to themselves and to their patients to be up-to-date with the evolving area of HIV and related issues. Thus this review contains the important aspects of the HIV and AIDS related to dentistry.

**KEYWORDS:-** HIV, health, dentistry.

### INTRODUCTION

Acquired immune-deficiency syndrome (AIDS) was first reported by *Gottlieb et al* in 1981, at University of California Medical Center. Barré-Sinoussi and Montagnier, isolated a new human T-lymphotropic retrovirus in 1983, later named human immunodeficiency virus type 1 (HIV-1); which turned out to be one of the causative agents of AIDS.<sup>[1]</sup> AIDS came into limelight in 1981. In India the detection of HIV infection for the first time was made in April

1986 in the state of Tamil Nadu. Since then, HIV infection has been spreading at an alarming rate.<sup>[2]</sup>

From the onset of the human immunodeficiency virus (HIV) epidemic over 20 years ago, more than 60 million people have become infected and more than 20 million people have died. An estimated 15,000 new infections occur each day, with more than 95% of these in developing countries.<sup>[3]</sup>

HIV/AIDS is one of the most important and preventable causes of mortality, morbidity, disability, medical care cost and associated productivity loss, especially in the world's poorest countries.<sup>[1]</sup> Human immunodeficiency virus (HIV) infection is a serious disorder of the immune system in which the body's normal defenses against infection break down, leaving it vulnerable to a host of life-threatening infections.<sup>[2]</sup>

The presence of certain oral diseases can be an important tool for identifying persons living with HIV and assessing the relative progression of their disease.<sup>[2]</sup> HIV related oral abnormalities are present in 30–80% of HIV-infected individuals. Individuals with unknown HIV status and oral manifestations may suggest possible HIV infection, although they are not diagnostic of infection.<sup>[2,4]</sup>

The presence of oral lesion may be an early diagnostic indicator of immunodeficiency and HIV infection, and is a predictor of the progression of HIV disease. Oral lesions of HIV infection are included in various classifications and staging of HIV diseases.<sup>[2]</sup> Dental expertise is necessary for proper management of oral complications in HIV infection or AIDS. Medical clinicians should be able to recognize HIV-associated oral disease and to provide appropriate care and referral.<sup>[5]</sup>

### **Molecular Organization of HIV**

Structurally, HIV is very peculiar. It is enveloped by a lipid bilayer of host origin in which viral glycoproteins are embedded as knob-like structures. Glycoproteins play a very important role in the attachment and fusion of virus with host cells and then lead to their cellular entry.<sup>[6]</sup>

As HIV is a retrovirus, its genome consists of RNA, which has various overlapping open-reading frame coding for several viral proteins. The retrovirus family is composed of three subfamilies: spumaviruses, oncoviruses, spumaviruses and lentiviruses. HIV is classified as

lentivirus based on the structure, biologic properties, and protein and nucleic acid sequence homology. A mature extracellular particle of HIV is characteristically 90–130 nm in diameter. HIV has a cylindrical eccentric nucleoid, or core. The nucleoid contains the HIV genome, which is diploid (i.e., composed of two identical single stranded RNAs). Encoded in the RNA genome are the entire complements of genes of the virus. These genes code for the structural proteins that are used to assemble the virus particles and the regulatory proteins involved in the regulation of viral gene expression.<sup>[3,6]</sup>

### **Pathogenesis**

HIV virus is a lymphotropic virus. Its primary target is T4 cell. When the virus enters the bloodstream, it integrates into DNA of some primary T4 lymphocytes. This viral DNA is then becomes integrated into the host chromosomes, which is prerequisite for replication of retroviruses and also for the latency of the virus. Once the viral gene is activated, virus particles convert T4 lymphocytes into AIDS virus factory. When the number of T4 lymphocytes is severely depleted, the immune system collapses and variety of infections occur and at this stage patient is said to have AIDS.<sup>[7,8]</sup>

### **Importance of oral lesions**

Oral lesions can indicate infection with HIV and also among the early clinical features of the infection, which can predict the progression of the disease to acquired immunodeficiency syndrome (AIDS). It can be used as entry or end-points in the therapy and vaccine trials and can also be used as determinants of the opportunistic infection and anti-HIV therapy. It can be used in the staging and the classification systems.<sup>[9]</sup>

The person's perception of social, economic and psychological consequences of oral conditions and of their treatment will play an important role in his/her oral health behaviors, including preventive and use of dental care.<sup>[10]</sup>

### **Mode of transmission of HIV**

Predominant mode of transmission is through sexual contact, which account for more than 70% of all cases of HIV transmission. It is also transmitted through blood and blood products, and breast milk. HIV has also been demonstrated in oral fluids, but their infectivity is low. So, saliva is not a significant route of transmission. Infection can also be transmitted vertically from mother to child.<sup>[8]</sup>

### **Dentists risk of transmission of HIV virus**

The most well-known case of HIV being transmitted to a patient within a dental practice was in Florida during 1992. A dentist was found to have been the source of infection of 5 of his patients.<sup>11</sup> The CDC states that the risk of HIV infection after a needlestick injury or a cut exposure to HIV-infected blood is 0.3%.<sup>[12,13]</sup>

It is highly unlikely that the patient will become inoculated with blood from the dentist during treatment and any procedures are stopped as soon as a needlestick injury occurs. It could be theoretically possible to acquire an infection through exposure to a percutaneous wound but environmental exposure is considered extremely low risk as HIV is only able to survive outside the body for short periods of time.<sup>[12,13]</sup>

However, there are also certain factors that determine if an infection will occur. The biggest factor in determining if a HIV infection will take place between an infected person and a recipient is the volume of contaminated blood during inoculation.<sup>[14]</sup> This is because the viral load in an infected person is directly proportional to the chance of the virus being transmitted. The amount of virus in a millimeter of blood can be as low as 200 units, and this is even lower if the person is receiving antiretroviral treatment.<sup>[15]</sup>

### **Oral pathophysiology of HIV infection**

HIV infection causes an immunodeficiency state by gradually impairing humoral and cell-mediated immunity. This allows new pathogens to more readily infect and cause disease, and this disease is usually more severe and widespread. Pre-existing or latent conditions held dormant by a functioning immune system can be re-activated. Also opportunistic infections may develop, a process whereby normally non-pathogenic organisms gain the ability to cause disease. Similar to systemic HIV-associated pathology, oral lesions presenting during HIV infection are more likely to occur with a high viral load or a reduced CD4 cell count.<sup>[16]</sup>

### **Oral manifestations of HIV**

**The various oral manifestations can be categorized into:**<sup>[4,17]</sup>

1. Infections: bacterial, fungal, viral
2. Neoplasms: Kaposi's sarcoma, non-Hodgkin's lymphoma
3. Immune mediated: major aphthous, necrotizing stomatitis
4. Others: parotid diseases, nutritional, xerostomia
5. Oral manifestations as adverse effects of antiretroviral therapy.

**Classification of oral lesions in HIV by Greenspan:** <sup>[18]</sup>

Group I	Group II	Group III
Hairy leukoplakia Kaposi sarcoma Linear gingival erythema Necrotizing ulcerative gingivitis Non-hodgkin lymphoma	Atypical ulcers Salivary gland diseases Viral infections like CMV, HSV,HPV,HZV	Diffuse osteomyelitis Squamous cell carcinoma Other rare lesios
HIV- Human Immunodeficiency Virus, CMV- Cytomegalovirus, HSV- Herpes Simplex Virus, HPV- Human Papilloma Virus, HZV- Herpes Zoster Virus.		

**Treatment of HIV infection**

The aim of treatment is to suppress HIV infection and allow immune recovery to optimize health. Ultimately, such treatment will prevent or lessen the likelihood of HIV-related complications and may prevent advanced HIV disease or acquired immune deficiency syndrome (AIDS). Current treatments are usually very successful with current life expectancy from diagnosis of a young person projected to be almost 40 year.<sup>[18]</sup>

Treatment of HIV infection involves the use of combinations of antiretroviral medications, currently referred to as combination antiretroviral therapy (cART). cART is instituted based on the monitoring of disease progression. Current guidelines recommend starting treatment when an individual's CD4 cell count declines to 350 cells/IL or there is a diagnosis of an AIDS-defining illness with treatment aiming to produce an undetectable viral load of less than 20–75 HIV copies/mL of plasma.<sup>[3,16]</sup>

In the recent years, the management of HIV-positive individuals has been based on highly active antiretroviral therapy (HAART), comprising a combination of nucleoside analogue RT inhibitors and at least one protease inhibitor and/or one non-nucleoside analogue RT inhibitor.<sup>[3,16]</sup>

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