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Human Immunodeficiency Virus in Periodontal Treatment - A Review Article

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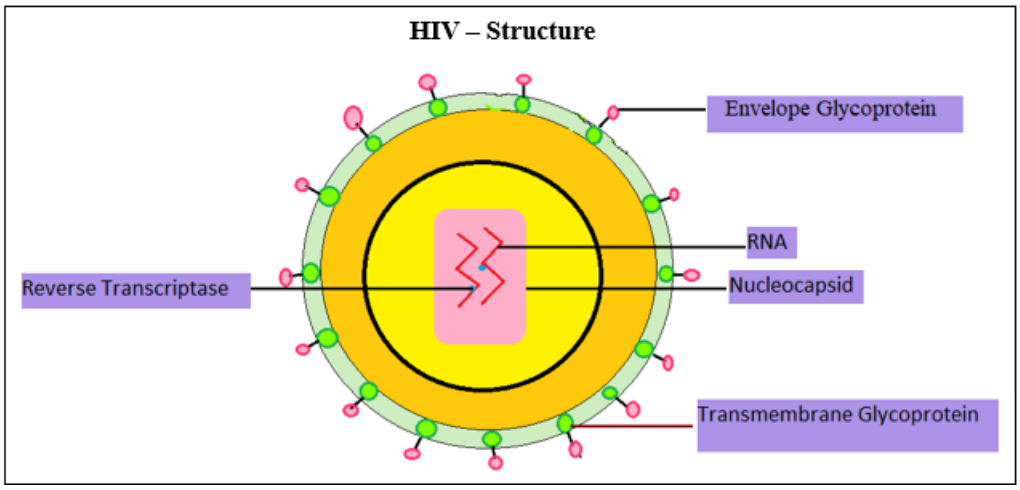
Abstract: ***The human immunodeficiency virus is a retrovirus belonging to the family lentiviruses which causes AIDS in humans. Since the identification of this extensive research has been done on HIV to understand its life cycle, routes of transmission, pathogenesis of AIDS and possible cure for the disease. The main reason because of which the successful treatment of HIV infection is so difficult is "its rapid rate of evolutionary change. The WHO and the Centers for Disease Control and Prevention have published guidelines regarding the definition and clinical stages of the disease, which classifies patients by the status of their symptoms and manifestations of immunodeficiency. An in - depth understanding of AIDS and its oral manifestations is important for appropriate management of oral lesions in patients infected with HIV.***

Keywords: HIV, AIDS, retrovirus, transmission, treatment

1. Introduction

HIV – Structure



- HIV consists of a cylindrical centre surrounded by a spherical shaped bilipid layer envelope. The virus particle has a diameter of about 1/10, 000nm.
- Structurally, the virus is composed of three components:
  a) Viral envelope
  b) HIV matrix protein
  c) Viral core

Viral Envelope: It consists of a lipid bilayer. There are two major glycoproteins **gp120 and gp41**. These glycoproteins of CD4+cells and chemokine receptors, thereby enabling the virus to attach to and invade CD4+ cells. HIV infects cell that carry the following receptor and co - receptors.

- **CD4**: Expressed on the surface of CD4 T - lymphocytes and macrophages.
- **CCR5**: Expressed on CD4+ T - lymphocytes and on macrophages.
- **CXCR4**: Expressed on CD4+ T - lymphocytes and T - cell lines.

HIV Matrix Proteins: It consists of **p17 protein**, which lies between the envelope and core.

Viral Core: It is formed of viral capsule **protein p24** which surrounds 2 single strands of RNA and the enzymes needed for HIV replication, such as reverse transcriptase and integrase. It also contains 3 out of 9 virus genes namely **gag, pol and env.**

Gag gene: contains around 1500 nucleotides. It gives rise to a 55 KD gag precursor protein p55. This protein later is cleaved into 4 smaller proteins which form the building blocks for the viral core,

a) CA - capsid
b) MA - matrix
c) NC - nucleocapsid
d) p6

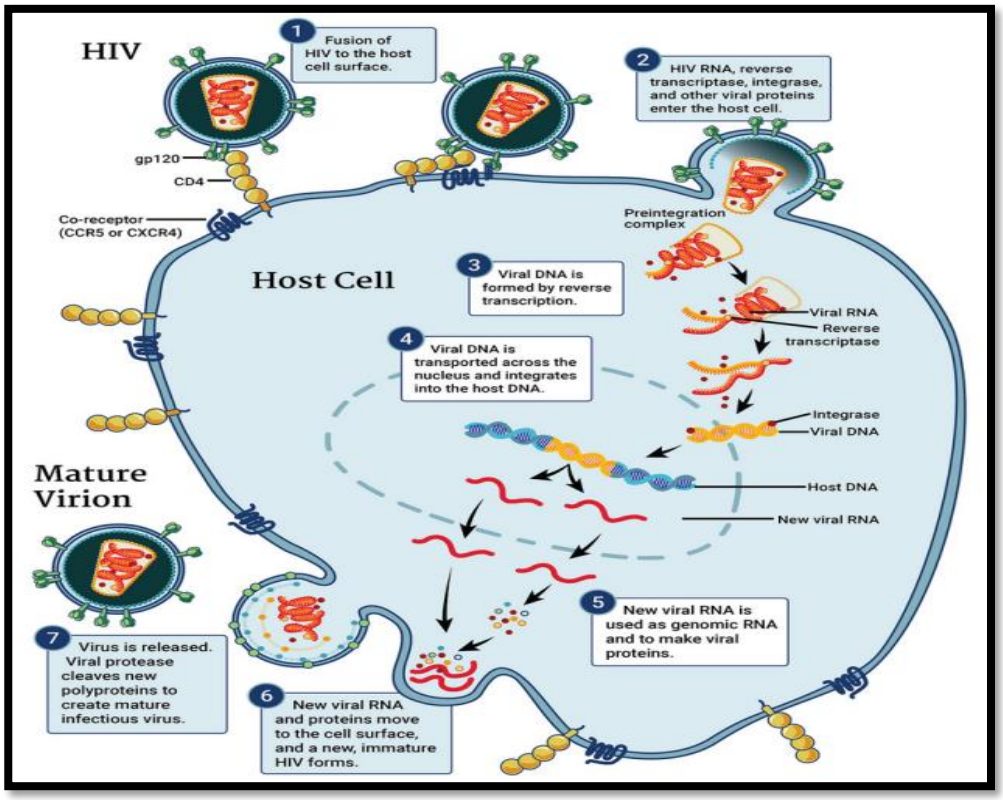
Pol gene: It encodes for enzymes required for various cellular processes in HIV which include,

a) protease

b) RNAse H
c) Integrase

Env gene: It encodes for a single protein, gp160. After synthesis, this protein travels to the cell surface where it splits into 2 parts gp120 and gp41 by enzymatic action.

HIV Life Cycle



Testing for HIV Infection

- The enzyme - linked immunosorbent assay (**ELISA**) and Western blot (**WB**) tests were the original methods for determining the presence of HIV - 1 and HIV - 2 antibodies in serum or plasma, and they are still considered the gold standard of confirmatory testing.
- The ELISA test is performed first, and it is repeated if the first test is positive. If it is positive a second time, WB is performed, and a positive finding is considered diagnostic for the infection.
- **WB TESTS** for the proteins **p24** (capsid protein), **gp41**, and **gp120** through **gp160**. In many laboratories, a positive band to two of these three proteins is considered positive, whereas anything less is considered indeterminate or negative if no antigenic proteins are detected.
- Both ELISA and WB require the presence of circulating HIV antibodies, which may take several weeks or even months to become detectible.
- Today, several other test methods are based on the presence of an HIV antigen. Although these tests are expensive, they may be of benefit, especially for early diagnosis after exposure.
- The window of time between exposure and the development of antibodies can lead to an increased risk for disease transmission, and some evidence suggests that early treatment during that window can significantly minimize the intensity of the infection and positively influence treatment outcomes.
- It must be recognized that **rapid tests** may have a relatively **high number of false - negative** results as well as some false - positive results, so repeat testing and follow - up testing with ELISA and WB are often indicated.
- Rapid tests may be of special benefit in undeveloped countries, where medical laboratory testing may not be readily available.
- Infected individuals treated with **HAART** may experience a marked **rise in CD4 cell levels** and a decreased plasma viral load. CD4 counts may reach normal levels, and viral bio load may decrease to a point below the level of detection. Despite this improvement, these individuals are still considered to have AIDS, because the virus is apparently sequestered somewhere in the body.
- This **acute phase** may last for up to **2 weeks**, with **seroconversion** occurring **3 to 8 weeks later**. However, antigenic viremia may sometimes be present for an extended time before seroconversion occurs. Some individuals experience asymptomatic HIV infection, whereas others may become asymptomatic after the initial acute infection.

Clinical Signs and Symptoms with HIV

- The population of viral particles in the blood may reach 10, 000, 000/ml.
- The infected person may experience flu - like symptoms for upto 2weeks.
- The patients usually complaints of their condition as '**worst flu ever** '.
- Other symptoms include fever, swollen glands, sore throat, rash, muscle and joint aches and pains, fatigue, and headache. This phase of HIV infection is referred to as

"**acute retroviral syndrome" or "primary HIV infection"**.

- The **seroconversion occurs after 3 - 8 weeks**. Because large numbers of viruses are produced during the initial period following infections, they use CD4 cells to replicate and destroy them in the process. Hence, there is a sharp downfall in CD4 cell count.
- After the initial Th cell decline, the body starts generating an antibody response against HIV. This reduces the HIV count in the blood.
- The helper T - cell population recovers and the immune response is generated against the virus which keeps it at low and steady level.
- This level of virus in the blood is referred to as '**viral set point'**. Over a period of time, the immune system fails to cope up with the viral load due to infection of helper T - cells and viral levels rise again.
- The potent starts experiencing opportunistic infections such as Candida albicans infections of the mouth or vagina, persistent diarrhea, fever, weight loss and reactivation of previous infections such as shingles and tuberculosis.
- Eventually, the body fails to recover from these infections and the patient dies.

Classification of oral lesions in HIV infection in adults:

The first classification of oral lesions associated with HIV infections was given by the European Economic Community in 1986. This classification was modified by **Pindborg** in 1989. This proposed classification proposed three groups of lesions:

Group 1 – lesions strongly associated with HIV infection.
Group 2 – lesions less commonly associated with HIV infection.
Group 3 – lesions only possibly associated with HIV infection.
Group 4 – contained lesions associated with the use of drugs.

- **Oral lesions less strongly associated with HIV infection** include melanotic hyperpigmentation, mycobacterial infections, necrotizing ulcerative stomatitis (NUS), miscellaneous oral ulcerations, and viral infections (e. g., herpes simplex virus [HSV], herpes zoster, and condyloma acuminatum).
- **Lesions that are seen in HIV - infected** individuals with undetermined frequency include less common viral infections (e. g CMV, molluscum contagiosum), recurrent aphthous stomatitis, and bacillary angiomatosis (epithelioid angiomatosis).

Antiretroviral Therapy:

- The goals of therapy are to reduce morbidity and mortality and improve and prolong life. However total eradication of HIV does not appear to be currently possible because reservoirs of latently infected CD4 cells are established early in the infection and persist even with treatment.
- Anatomic reservoirs of infection have been located in the gastro intestinal tract and reproductive tracts and in breast, lung, and brain tissue, and recently, the **oral cavity** has been proposed as **an anatomic reservoir** because as **small percentage of HIV - infected individuals were found to be salivary hyperexcretors of HIV**.
- **Nucleoside reverse transcriptase inhibitor (NRTI)** were the first class of antiretrovirals developed. They act by competing for incorporation into the proviral chain, eliminating chain elongation. They are effective against both HIV - 1 and HIV - 2. NRTIs are the principal drugs in use today in drug combinations.
- **Nonnucleoside reverse transcriptase inhibitors (NNRTI)** bind to the active site in the developing HIV - 1 viral chain and arrest its development. These drugs are quite effective but rapidly develop resistance by a single step - mutation.
- **Protease inhibitors** are active against both HIV - 1 and HIV - 2. They bind to the HIV protease enzyme and result in formation of noninfectious HIV virions.
- **Fusion inhibitors** bind to the gp41 glycoprotein membrane on HIV and prevent virus fusion into susceptible CD4 cells. The drugs are administered by subcutaneous injections.

Highly Active Antiretroviral Theraphy:

- With HAART therapy, combinations of three or more antiretroviral drugs are administered simultaneously for long periods of time in an effort to block HIV replication and plasma viral load and to restore immune function while minimizing antiretroviral drug resistance and adverse drug effects.
- Three commonly used combinations are **one NNRTI plus two NRTIs, one or two protease inhibitors plus two NRTIs, or three NRTIs**. One NRTI three - drug combination is available in a single tablet, but this antiretroviral therapy does not necessarily qualify as HAART.
- HAART has proved to be very successful at achieving a significant increase in the survival and quality of life of HIV - infected individuals, and life expectancy is markedly increased for many individuals using this therapy. However, not everyone benefits equally from HAART. Commonly described systemic conditions that may be found more often after HAART are lipodystrophy, increased insulin resistance, gynecomastia, blood dyscrasias, and dermatologic disorders
- **Survival time is shorter despite HAART -** who are injected drug abusers, those with AIDS before the initiation of therapy, those with a very high viral load before the initiation of therapy (i. e., >100, 000 copies/mL), those who are inconsistent in their adherence to the drug regimen, and those who discontinue the drugs.
- Other factors that affect life expectancy in the era of HAART include smoking; excessive alcohol consumption; older age at initiation of therapy; comorbidities such as hepatitis C or other viral, bacterial, or fungal infections; chronic liver, kidney, or cardiovascular conditions; and diabetes mellitus.
- Before the advent of antiretroviral therapy, opportunistic infections were the principal cause of morbidity and mortality among HIV - infected persons. Opportunistic infections continue to occur among those who are unaware of their HIV infection, those who do not take recommended medications, and those who are resistant or react adversely to recommended medications.
- In the oral cavity, candidal infections are markedly reduced among individuals who are responsive to antiretroviral therapy; hairy leukoplakia, Kaposi sarcoma,

deep mycoses, necrotizing ulcerative gingivitis (NUG), and necrotizing ulcerative periodontitis (NUP) are reduced in these patients as well.

Periodontal Treatment Protocol:

1) Health Status

- The patient's health status should be determined from the health history, the physical evaluation, and consultation with the patient's physician.
- Treatment decisions will vary, depending on the patient's state of health. Information should be obtained regarding 1. CD4 CELL LEVEL 2. H/O drug abuse 3. present medications

2) Infection Control Measures

- The clinical management of HIV - infected periodontal patients requires strict adherence to established methods of infection control, which should be based on guidance from the American Dental Association and the CDC.
- Immunocompromised patients are potentially at risk for acquiring as well as transmitting infections in the dental office and other health care facilities.

3) Goals of Therapy

- The **primary goal** of dental therapy should be the restoration and maintenance of oral health, comfort, and function.
- Acute periodontal and dental infections should be managed, and the patient should receive detailed instructions regarding the performance of effective oral hygiene procedures.
- Conservative nonsurgical periodontal therapy should be a treatment option for virtually all HIV - positive patients.
- Decisions regarding elective periodontal procedures should be made with the informed consent of the patient and after medical consultation, when possible.

4) Maintenance Therapy

- In addition, periodontal maintenance recall visits should be conducted at short intervals (i. e., every 2 to 3 months), and any progressive periodontal disease should be treated vigorously.
- As mentioned previously, systemic antibiotic therapy should be administered with caution.
- Blood and other medical laboratory tests may be required to monitor the patient's overall health status, and close consultation and coordination with the patient's physician are necessary.

5) Psychological Factors

- HIV infection of neuronal cells may affect brain function and lead to dementia. This may profoundly influence the responsiveness of affected patients to dental treatment.
- Coping with a life - threatening disease may elicit depression, anxiety, and anger in such patients, and this anger may be directed toward the dentist and the staff. (Asher etal 1993)
- Treatment should be provided in a calm, relaxed atmosphere, and stress to the patient must be minimized.

2. Conclusion

- The pathogenesis of periodontal disease in HIV subjects may be due to the microflora, the effects of HIV and other viral agents, and/or alterations in the host response. These factors should be taken into consideration in the treatment and prevention of periodontal diseases in the HIV patient.
- Early clinical picture thus act as a clinical indicator of HIV in otherwise healthy individual and also oral lesions are predictors of disease progression.

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