e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(4); 1361-1369

Original Research Article

HIV Causes of Infection, Spread and Treatment Among Human Beings

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Received: 06-02-2026 / Revised: 16-03-2026 / Accepted: 10-04-2026
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Abstract

HIV was commonly known as human immunodeficiency virus. Nowadays it was a common disease among adults worldwide. The virus was grouped to the genus Lentivirus, family retroviridae and subfamily Orthoretrovirinae. HIV can be classified into two groups, HIV-1 and HIV-2. Genomic analysis reveals that HIV introduced among human in 19th century. It was spread from male to female and female to male by sexual transmission. Also, through injection and blood transfusion methods it spreads. Clinic was the best place where the treatment was done properly. DNA PCR and RT PCR were some methods used for the diagnosis of HIV. One more thing we can observe that prevention was more efficient than infection. We can discuss that how we can get cured from this disease.

Keywords: HIV, virus, HIV-1 and HIV-2, transmission, injections.

DOI: 10.25258/ijcpr.18.4.217

Introduction

In the last two decades, HIV has made remarkable progress in prevention and treatment of HIV. Incidence decreases considerably in most of the world and large improvements among the people on antiretroviral therapy (ART). We now understand the pathogenesis of HIV, which implicated both for cure and vaccine strategies. We understand more than ever before about the pathogenesis of HIV and nature of the latent reservoir, which have implications for both cure and vaccine strategies. To make HIV prevention and treatment simpler, conventional biomedical technologies becomes the new one. Long action of modern pills and injections give relief from the daily medication system. In the year of 2022, over 39 million were affected by HIV whereas in the year of 2010 persons affected were 8 million. The percentage of death was decreased by 69% on the due date[1]. HIV epidemic was still forgettable and challenges arises. If you take a vaccine, you will get cured and proved to be rescued from this disease. The risk and cost of cure against the landscape of treatment [2,3]. Attention was required for existing technologies to overcome the HIV problem than the modern problem [4]. A lot of resources were available in prevention, treatment and implementation made the United Nations General Assembly's 95-95-95 goals successful. By 2025, the goal was to ensure that 95% of patients with HIV were diagnosed, 95% of the diagnosed patients receive ART achieve viral load suppression [5]. HIV mortality show a wide decrease. The countries where political and other turmoil occurred, were highly stigmatized [6]. Number of patients increases with increasing methods of treatment.

37.7 million patients diagnosed in 2020 and 39 million patients diagnosed in 2022. Infection of HIV increases both the risk of cardiac arrest and neurological disorder. Due to ART treatment related to healthcare system old treatment should be adversed. Using local clinical guidelines for managing HIV improves patient's outcomes and prevents HIV transmission. Prolonged clinical treatments related to diagnosis decreases the risk of disease. Prevention is better than cure. HIV treatment can cure the disease the disease in the initial stage which doesn't prolonged the disease [7]. The HIV structure, genomics, pathogenesis, mode of transmission, clinical features all will be described in a regular mode. The HIV-1 was an opportunistic pathogen whose laboratory diagnosis were necessary. The strategy, RT Therapy and prevention were also necessary.

Structure: In electron microscope we can see that HIV Viron has an icosahedral structure. Two major envelope proteins involve in the external spike structure. The two envelope proteins were gp120 and gp41[5][Figure 1]. The viron buds from the surface of the infected cell and incorporates a variety of host cellular proteins into the lipid bilayers.

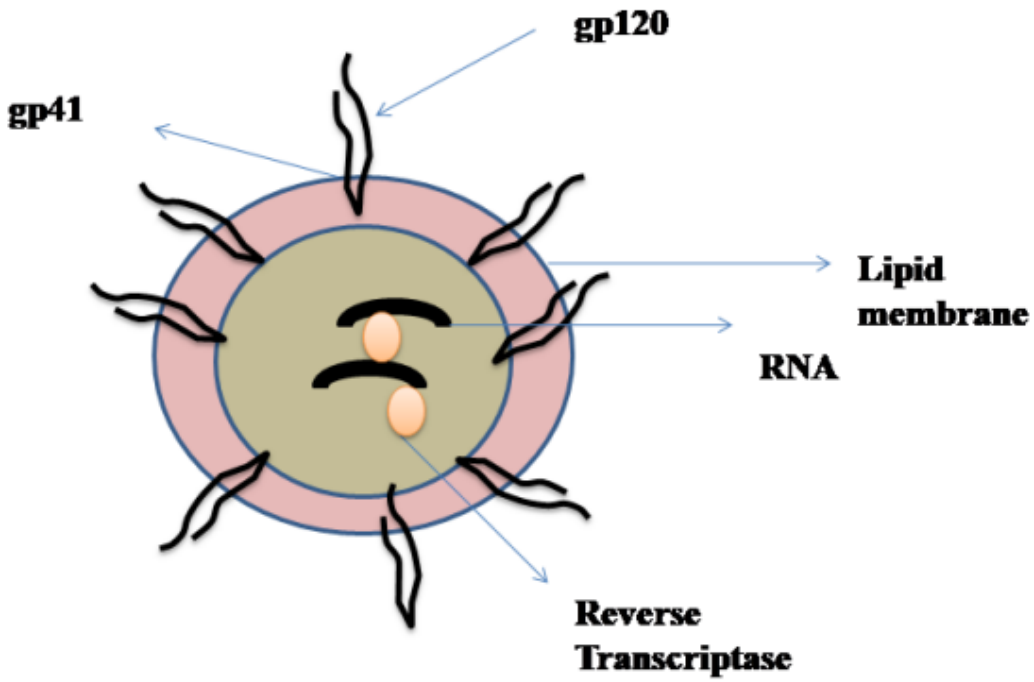

Figure 1: Representation of the structure of HIV (Human Immunodeficiency Virus)

Genomics: HIV-1 has genes that encode the structural proteins of the virus, gag encodes the proteins that form the core of the virion residing p24 antigens. Another protein pol enzymes that responsible for the protease processing viral proteins. Reverse transcription and integration encodes envelope proteins env. Six other regulatory genes (tat, rev, nef, vif, vpr and vpu) present on the nonproximate group of HIV1 retrovirus [15,16,17]. It involves in the modification of structure of host cell which enhance viral growth and regulate viral gene expression. Several proteins play crucial role in the pathogenesis of HIV disease. The major difference between HIV-1 and HIV-2 is that HIV-2 lacks the vpu gene and has a vpx gene not contained in HIV-1.a

Pathogenesis: The hallmark of HIV disease is a profound immunodeficiency resulting primarily from a progressive quantitative and qualitative deficiency of the subset of T lymphocytes referred to as helper T cells occurring in a setting of aberrant immune activation creating a state of immunosuppression. The helper subset of T cells define presence of CD4 molecule on the surface of Tcells [10,11,12,13]. It acts as a receptor for HIV. The helper subset of CD4 cells represent how it presents on the surface of Tcells. A co-receptor present with CD4 defined its binding, fusion and entry in HIV-1 target cells. HIV-1 uses two major co-receptors CCR5 and CXCR4. These two were selected for fusion and entry inside the cell.

These co-receptors were also the primary receptors for certain chemo attractant cytokines termed chemokines and belong to the seven-transmembrane domain G protein-coupled family of receptors. In vitro the dysfunction of CD4+ cells to T cells multiple mechanism developed. HIV destroyed all the cells. Cell death causes after cellular depletion. This dysfunction of CD4+ T cells undergo in vitro activity. Direct effects of cells by HIV causes by attack by HIV. Indirect effect causes by immune clearance of infected cells. Cell death occurs by caspase-1 mediated death by tissue CD4+ T cells. It undergoes abortive/nonproductive HIV infection.

Immune system breakdown result in the cellular deactivation and cellular dysfunction.

A variety of opportunistic diseases developed when the patients suffered low level of CD4+ T cells in human. Immunodeficiency caused by HIV infection causes Kaposi's sarcoma and neurologic abnormalities. Other immunologic complications may develop due to severe immunologic impairment. The course of HIV infection may be the combination of viral pathogenic and immunopathogenic events. The mode of infection varied from primary stage to advanced stage complex infection. It is important to appreciate that the mechanism of HIV disease was multifunctional and are different at different stages of the diseases.

Mode of Transmission: HIV was transmitted by sexual contact (by blood and blood products). It was spread from infected mothers to infant via breast milk. Different mode of transmission represented in Figure 2.

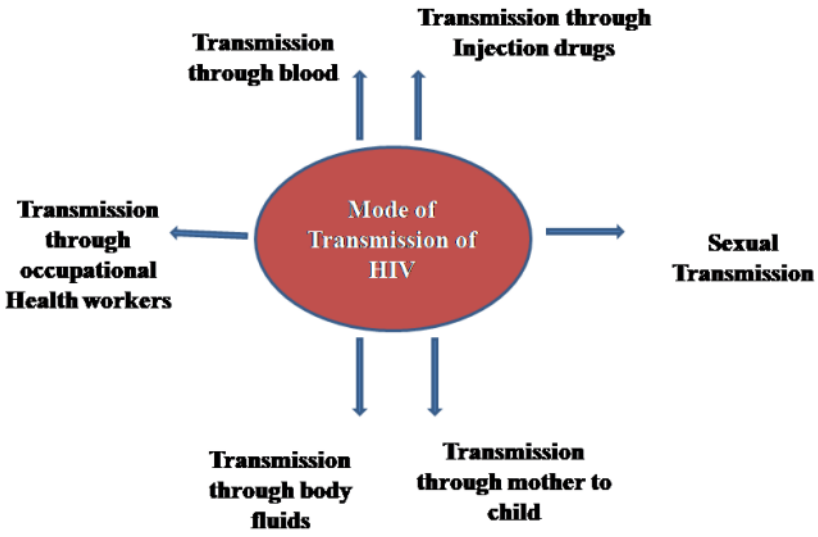

Figure 2: Different mode of transmission of HIV (Human Immunodeficiency Virus)

Table 1: listed different mode of exposure for HIV transmission.

Type of Exposure	RISK PER 10,000 Exposures
Parenteral	
Blood transfusion	9250
Needle-sharing during injection drug use	63
Precutaneous (needle-stick)	23
Sexual	
Receptive anal intercourse	138
Insertive anal intercourse	11
Receptive penile-vaginal intercourse	8
Insertive penile-vaginal intercourse	4
Receptive oral intercourse	Low
Insertive oral intercourse	Low
Other	Negligible
Biting	Negligible
Spitting	Negligible
Throwing body fluids (including semen or saliva)	Negligible
Sharing sex toys	Negligible

Sexual Transmission: In worldwide history, HIV was a predominantly sexually transmitted infection (STI). Male-to-male sexual transmission in western countries was a part of heterosexual transmission. This type of transmission [8,9] was most common in developing countries. It was a systemic review that found lower risk of heterosexual transmission in the absence of antiretroviruses. During vaginal intercourse in absence of protection or condoms 0.04% transmission occur from female to male and 0.08% occur from male to female (Table 1).

HIV occur in seminal fluid both in infected mononuclear cells and in cell-free material. When lymphocytes and monocytes were increased in number in fluids the virus concentrate in the seminal fluid.

Urethiritis and epididymitis were some diseases (STIs) arises for genital inflammation. The virus also located in the cervical smears and vaginal fluids. Anal intercourse (URAI) arises more infection of HIV than vaginal intercourse both in men and women. In both the cases unprotected intercourse was responsible for the infection. Although data were limited, the URAI (unprotected receptive vaginal intercourse) value has been estimated near about ~1.4% (Table 1).

The risk of HIV spreading associated with URAI was higher than penile-vaginal intercourse. It was because a thin, fragile mucosal membrane seperates the semens from mucosal membrane that associated with anal intercourse. There were two pathways in which anal intercourse provides infection: (1) direct inoculation into blood in cases of traumatic tears in the mucosa (2) infection of susceptible target cells in absence of trauma. HIV also infected among patient having vaginal intercourse. In Table 1 it was given that HIV was infected among male to female more than female to male. This difference arises due to exposure of vaginal and cervical mucosa to infected seminal fluid. Incomparison to the penis and urethral orifice of the uninfected male partnerare only exposed relatively briefly to infected vaginal fluid.

Some microorganisms that were ulceratives responsible for HIV infection, like *T. pallidum., H. ducreyi*, HSV. Non-ulceratives microorganisms responsible for HIV infection were *C. trachomatis., N. gonorrhoeae* and *T. vaginalis.*Bacterial vaginosis, an infection related to sexual behaviuor, but not strictly an STI, also may be linked to an increased risk of transmission of HIV infection. HIV transmission decreases on treating properly STIs and genital tract syndromes.

HIV transmitted through injection paraphernalia: HIV an also be transmitted through injection drug users (IDUs). Needles, syringes, water mixed with drugs, cotton used during fitration of drugs were exposed to environment after collection of blood. Intramuscular injection can also causes HIV. IDUs were commonly use the frequency of needle sharing, duration of injection drug use, number of partners among which the injections commodity will be used. In Table 1 the per act risk of transmission from injection drug use with a contaminated needle as been estimated to be approximately 0.6%.

Transmission of HIV through blood and blood products: HIV can be transmitted through individuals by blood transfusions, blood products and by transplanted tissue. In major cases HIV can be occurred by blood components, transplanted tissue and contaminated blood. It was tested prior to the spring of 1985, when mandatory test of donated blood for HIV-1 was required. More than 90% of the patients HIV contaminated blood products were spreaded presented in Table1. Transfusions of whole blood, packed red blood cells, platelets, leukocytes, and plasma were all capable of transmitting HIV infection. In contrast, hyperimmune gamma globulin, plasma-derived hepatitis B vaccine, and Rh immune globulin have not been associated with transmission of HIV infection. The procedures involved in processing the products either inactive or remove the virus.

Epidemiology: AIDS/ HIV infection was a pandemic disease. In the year of 2023, in UNAIDS a programme was there. 95% people affected in the low- and middle-income countries. Among them ~53% were female and children less than 15 years old were 1.4 million. The global prevalence arises as people affected ~0.7%, among 15-49 years old. From the start of the pandemic ~88.4 million people [2] affected worldwide. Globally some factors such as marginalization, discrimination, criminalization affect HIV disproportionately to the population. Sex workers, people who inject drugs, transgender people, prisoners, gay men, men those who have sex with men clients of sex workers and sexual partners all around 55% have new HIV infections (In the year of 2022). Although the AIDS epidemic was first recognized in the United States then it was spread in the Western Europe and to Sub-Saharan Africa. In eastern and southern Africa were near about 7% of the world population; i.e near about 20.8 million people in the year of 2023. HIV observed among adults in the age of 15-49 years was 5.7%. Among 21 countries 17 were generalized epidemics (>1%). In 6 country it was >10% (15-49years old). South Africa has highest HIV (7.7 million). Eswatini had a

highest HIV among adults (25b.1%). In 2022 in the survey, it was reported that sex workers were accounted for about 23% of new infections.

In 25 countries of Western and Central Africa near about 5.1 million people had HIV. 3.1 millions of women and 380000 were children. In Nigeria the HIV rate was near about 1.3%. In eastern and southern Africa the sexual transmission accounts for about one-fifth of new infections. In West and Central Africa new infections occur among sex workers. In the Middle East and North Africa, the lowest HIV prevalence rate was near about< 0.1%. The new infection doubled between 2010 and 2023 (from 11000 to 23000). In Asia and the Pacific, at the end of 2023, about 6.7 million people were living with HIV. In Caribbean the infection was near about 15000.

Death occurs in the region of Eastern Europe and entral Asia, Latin America, Middle east and north Africa this were near about 140000,120000 and 23000 respectively. 56000, 450000 and 190000 deaths respectively occur in Western and central Europe and North America, Eastern and southern Africa, Western and central Africa.

Clinical Features: In clinical features HIV infection starts from primary infection. Primary infection associated with plasma viremia (wide dissemination of virus) and trafficking of lymphocytes. This three (after 3-6 weeks of infections) leads to a disease known as acute syndrome [18]. After 1 week-3 months of acute syndrome immune response of HIV arises. Curtailment of plasma viremia also associated with above reason [Table 2]. Chronic, persistent infection in lymphoid tissue also established with acute response of HIV. After 1-2 weeks of infection clinical latency arises. The length of time from initial infection to the development of clinical disease varies greatly, the median time for untreated patients is ~10 years. In asymptomatic period, it was very difficult to emphasize HIV disease with active virus replication. Rate of disease progression was directly proportional to HIV RNA levels. Patients with high level of HIV RNA had faster symptomatic progress than lower HIV RNA levels. Clinical findings in the acute HIV syndrome were represented here.

Table 2: Clinical Findings of the Acute HIV Syndrome

General	**Neurologic**
Fever	Meningitis
Pharyngitis	Encephalitis
Lymphadenopathy	Peripheral neuropathy
Headache/retroorbital pain	Myelopathy
Arthralgias/myalgias	Dermatologic
Lethargy/malaise	Erythematous maculopapular rash
Anorexia/weight loss	Mucocutaneous ulceration
Nausea/vomiting/diarrhea	Not mentioned

Opportunistic pathogen

Table 3: Opportunistic illness in HIV Infection

Opportunistic illness in HIV Infection
Bacterial infections, multiple or recurrent
Candidates of bronchi, trachea or lungs
Cervical cancer, invasive
Coccidioidomycosis, disseminated or extrapulmonary
Cryptococcosis, extrapulmonary
Cryptosporidiosis, chronic intestinal(>1 month's duration)
Cytomegalovirus disease (other than liver,spleen,or nodes), onset at age>1month
Cytomegalovirus retinitis (with loss of vision)
Encephalopathy attributed to HIV
Herpes simplex: chronic ulcers (>1 month's duration) or bronchitis, pneumonitis, or esophagitis (onset at age>1month)
Histoplasmosis, disseminated or extrapulmonary
Isosporiasis,chronic intestinal (>1 month's duration)
Kaposi's sarcoma
Lymphoma,Burkitt's
Lymphoma,immunoblastic

Lymphoma,primary,of brain
Mycobacterium avium complex or Mycobacterium kansasii, disseminated or extrapulmonary
Mycobacterium tuberculosis of any site, pulmonary, disseminated or extrapulmonary
Mycobacterium , other species or unidentified species, disseminated or extrapulmonary
Pneumocystis jirovecii pneumonia
Pneumonia, recurrent
Progressive multifocal leukoencephalopathy
Salmonella septicemia, recurrent
Toxoplasmosis of brain, onset at age>1 month
Wasting Syndrome attributed to HIV

HIV breakdown our immune system. Our body readily attacked by pathogen. These pathogens were known as opportunistic pathogen[18]. In Table 3 all pathogens were opportunistic pathogen. They attack our body and causes different disease syndrome.

Laboratory diagnosis: In 1984, syndromes of AIDS were established as HIV virus. 1n 1985, blood donors in USA screened and sized for antibodies of HIV. In 1996, blood banks in the USA added p24 antigen to capture the screening process to identify rare, infected individuals. The person who was donating blood capture between donation and development of antibodies. In 2002, early detection of HIV was enhanced by the nucleic acid testing (NAT) test. Antibody testing and NAT testing developed day by day. Detection of plasma viremia was also a test to monitor the progress of HIV disease. These tests coupled with the measurement of CD4+ T lymphocytes was an important component that present in HIV infected patient. The CDC has recommended that screening for HIV infection be performed as a matter of routine health care. Antibodies bind to HIV and it detected as one of its components. In addition to laboratory-based tests several home tests were also available.

The standard blood screening tests depend on the antibodies and p24 antigen of HIV. A common laboratory-based platform is the ELISA, also referred to as an enzyme immunoassay (EIA)[19,20].

Antigens from HIV-1 and HIV-2 present in the diagnostic laboratories commercial kits. These natural and recombinant antigens continuously discovered species such as group O viruses. EIA tests were generally scored as positive, negative or indeterminate. EIA result should have the result confirmed with a more specific assay such as an HIV-1 or HIV-2 specific antibody immunoassay or a plasma HIV RNA level. EIA test score positive result for all infected individuals. Negative infection occurs for recent infection. This signifies level of infection to virus; it was insufficient to maintain a measurable antibody response.

CDC recommendation occurs for positive-fourth generation assay. HIV-1 and HIV-2 specific immunoassay or plasma HIV-RNA specific assay used for diagnosis. Western blot was not used for this purpose.

Guideline for a use of serologic tests was attempting in this Figure 3.

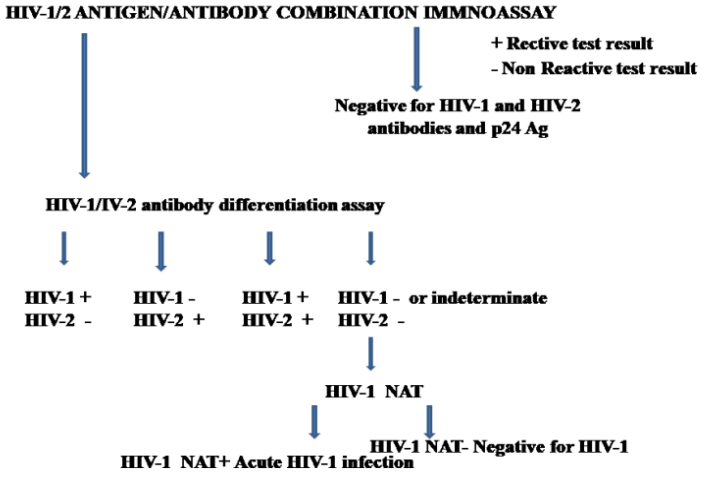

Figure 3: Different mode of infection (different positive and negative mode of infection)

Table 4:

Test	Technique	Sensitivity	Cost/Test
Immune complex-dissociated p24 antigen capture assay	Measurement of levels of HIV-1 core (p24) protein in an EIA-based format following dissociation of antigen-antibody complexes by weak acid treatment	Positive in 50% of patients; detects down to 15 pg/ml of p24 protein	Dollar 1-2
HIV RNA by PCR	Target amplification of HIV-1 RNA via reverse transcription followed by PCR	Reliable to 20 copies/ml of HIV RNA	Dollar 75-150
HIV RNA by bDNA	Measurement of levels of particle-associated HIV RNA in a nucleic acid capture assay employing signal amplification	Reliable to 50 copies/ml of HIV RNA	Dollar 75-150
HIV RNA by TMA	Target amplification of HIV-1 RNA via reverse transcription followed by T7 RNA polymerase	Reliable to 100 copies/ml of HIV RNA	Dolllar 225
HIV RNA by NASBA	Isothermal nucleic acid amplification with internal controls	Reliable to 80 copies/ml of HIV RNA	Dollar 75-150

A variety of laboratory tests were available for the direct detection of HIV or its components in Table 4. Antibody were indeterminate but HIV infection diagnosis can be done easily by the above test. The level of HIV RNA was used for antiretroviral therapies. The easiest, least expensive test was p24 antigen capture assay. It was detected the viral protein p24 in the blood of people with HIV where it exists either as free antigen or complexed to anti-p24 antibodies [Table 4]. For fourth generation test recommended for initial screening. Untreated HIV present in 30% cases have free p24 antigen. p24 antigen and p24 antibodies had equilibrium throughout the course of HIV infection. In the first week of infection level of p24 antigen levels rises. After anti- p24 antibodies development the level of antigen decreases. When virus levels were high, the p24 antigen levels also increases. The p24 antigen capture assay has its greatest use as a screening test for HIV infection in patients suspected of having the acute HIV syndrome, as high level of p24 antigen were present prior to the development of antibodies. It was used as an alteration of 'fourth generation' assays. This assay use for the combination of antigen and antibody testing or NAT.

The ability to measure and monitor levels of HIV RNA in the plasma of patients with HIV infection was observed. The extraordinary value of the plasma can be understood by the pathogenesis of HIV infection. In response to ART, the diagnostic tool used for the measurements of anti-HIV antibodies. This misleads to acute and neonatal infection. In addition to HIV RNA detection test DNA PCR assays were also used by the laboratories. It is used to amplify HIV proviral DNA from peripheral blood mononuclear cells. HIV RNA assay can be considered as a gold standard for a diagnosis of HIV infection.

RT-PCR technique also used in the treatment of HIV. It was used to treat DNA in presence of DNAase and primary primer tRNALys3. This was the same primer use for all viral life cycle. As HIV was a RNA virus uses in the production of DNA copies proportional to the amount of HIV RNA present in plasma. cDNA amplified using PCR techniques.

RT-PCR and DNA-PCR were some useful techniques used to amplify HIV genome. A sequence diversity will be done for microbial resistance and antiretroviral agents. In patients with a positive or indeterminate EIA test and an indeterminate Western Blot test can be done. Serological test also can be done. HIV RNA in plasma or DNA in peripheral blood mononuclear cels made a diagnosis of HIV infection. However, these all, should be used for diagnosis only when standard serologic testing has failed to provide a definitive result.

RT Therapy

The multiple antiretroviral drugs used for the treatment of HIV as antiretroviral agents. HIV resistance test can be measured by genotypic or phenotypic measurements. In genotypic assays sequence analysis can be done of HIV genomes obtained from different patients. Sequences of the patients should be compared with the known antiretroviral resistance profiles[21].

In the phenotypic assays, the in vivo growth of patient-derived viral isolates or genetically constructed pseudo viruses was compared with the growth of reference strains of the virus in the presence or absence of different antiretroviral drugs. In a patient these antiretroviral drugs can be easily

studied due to presence of past and future data. Resistance testing is recommended at the time of initial diagnosis. The initiation time of ART was indicated of virologic failure. Because the propensity for the pool of HIV quasispecies to rapidly revert to wild type in the absence of the selective pressures of ART. Viral load decreases ~0.5 log compared to the changing drugs. Resistance testing help in the selection of new drugs with patients of virologic failure. The patient needs to have an HIV-1 RNA level above 500-1000 copies/ml for an accurate resistance determination. Resistance assays lose their consistency at lower levels of plasma viremia.

Prevention

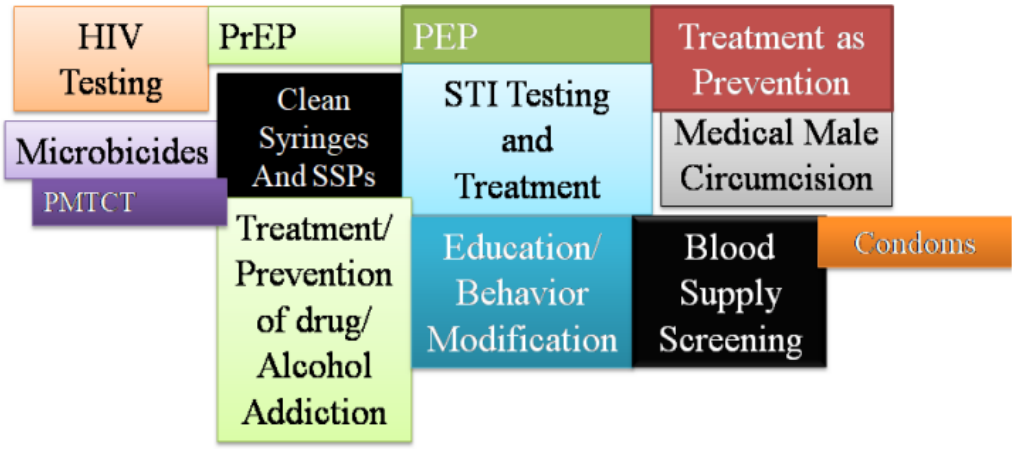

Figure 4: The HIV prevention 'toolkit'

Prevention of HIV represented in Figure 4. Different medicines were used in the treatment of HIV. It was get cured. But side effects were stay prolonged. So, its better to use prevention than disease. Sexual prevention[14,22] was best than other methods. If we see the above chart we can see how prevention was acts. So, its better to follow the path above for the prevention of HIV.

Conclusion

In this review paper we can conclude that the disease was known as HIV (Human immunodeficiency virus). It was subdivided into two group HIV-1 and HIV-2. It invades our immune system. Breakdown our immunity. Opportunistic pathogens attack our body and different diseases occur. Disease may be spread through epidemic mode. Sexual transmission, transmission through syringes, transmission through blood was also stopped by proper prevention methods. Prevention of sexual transmission done by condoms. People should be educated enough so that they can take proper protection during sex. Blood transfusion will be done in a proper way. If HIV get infected also proper medication was necessary.

Conflict of Interest: There is no conflict of interest.

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