HIV/AIDS

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Purpose

The purpose of this course is to provide a thorough understanding of HIV/AIDS, including the infective process, indications of disease, diagnostic procedures, treatment options, complications, and preventive measures.

Goals

Upon completion of this course, the nurse should be able to:

- Describe key events in the history of HIV/AIDS.
- Describe two species of HIV.
- Describe the 3 primary mechanisms of HIV syndromes.
- List and describe the 3 stages of HIV progression.
- Discuss 7 diagnostic tests.
- List at least 10 AIDS-defining conditions.
- Describe the 7 stages of the HIV life cycle.
- Discuss 7 different types of antiretroviral therapy plus a pharmacokinetic enhancer.
- Discuss the two types of testing used to monitor therapy.
- Discuss at least 6 types of prophylaxis/suppressive therapy.
- Discuss both pre-exposure and post-exposure prophylaxis.
- Discuss 4 types of pulmonary complications and 3 types of cardiovascular complications.
- Discuss 8 types of CNS complications and 3 types of PNS complications
- Discuss 3 types of musculoskeletal complications and one common ophthalmic complication.
- Discuss 6 types of GI complications and 2 endocrinological complications.
- Discuss 4 types of dermatologic complications.
- Discuss 4 types of HIV-associated malignancies.
- Discuss 5 preventive measures for individuals.
- Discuss 6 preventive measures for healthcare.

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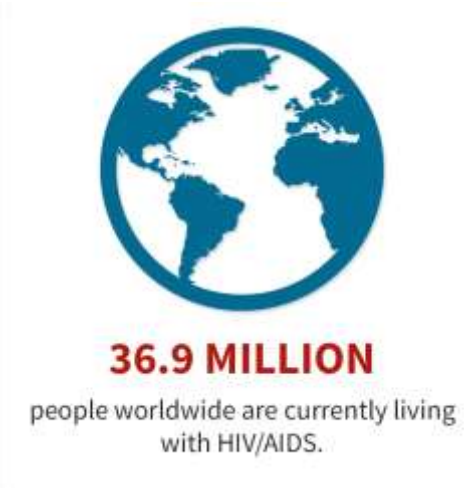
Introduction



The first inkling that a new epidemic was on the horizon occurred in June 1981 with a report from the CDC describing cases of a rare type of pneumonia, *Pneumocystic carinii*, in 5 young gay men in Los Angeles. By the end of that first year, 270 cases had been reported and 121 of those patients had died.

Because the disease was first identified in gay males, it was referred to as gay-related immune deficiency, but no one knew the cause. Since medical authorities believed the disease was limited to the gay population, they did not consider the danger to other groups, such as heterosexuals, IV drug-users, and people dependent on blood products, such as hemophiliacs.

By 1982, the disease had been renamed Acquired Immune Deficiency Syndrome (AIDS), and medical authorities recognized that it could be transmitted sexually. In 1982, the first cases of transmission by blood transfusions were reported. In 1983, researchers in France identified a retrovirus, called lymphadenopathy associated virus (LAV), that could cause AIDS. In the United States, researchers referred to the virus as human immunodeficiency virus (HIV). By 1984, there were 3064 diagnosed case of AIDS in the United States, and 1292 of those patients died.

In 1985, it was discovered that females could become infected through heterosexual sex. In March 1985, the FDA licensed the first commercial blood test, *ELISA,* to detect HIV; and blood banks began screening blood donations. By 1986, authorities recognized that HIV could also be transmitted in breast milk. AZT, the first anti-retroviral drug was approved in 1987. By 1990, between 8 and 10 million people were infected with HIV worldwide.

In 1995, about 50,000 Americans died of AIDS. In subsequent years, the number of deaths decreased as multidrug therapy became more widely available. The first protease inhibitor was approved in June 1995, beginning the era of treatment with highly active antiretroviral therapy (HAART), which became the standard of care by 1997.

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In 2002, the first rapid HIV diagnostic blood test was approved by the FDA for use in the United States, making diagnostic testing more readily available. In 2004, a rapid test using oral fluids rather than blood was approved, providing results within 20 minutes. In 2012, an at-home HIV test became available. In the same year, the FDA approved the use of Truvada® for pre-exposure prophylaxis.

Currently about 38,700 people receive a diagnosis of HIV each year in the United States, and about 1.1 million Americans are infected with HIV. About 15,000 to 17,000 people die with AIDS complications each year in the United States. There is not yet an FDA-approved vaccination, but preventive vaccines are available in clinical trials.

Scientists now believe that HIV developed in chimpanzees in West Africa during the 1930s and spread to humans through contact with infected blood from "bush meat trading." The disease spread slowly over decades in African, but began to spread at an alarming rate once it reached the United States and more populated areas.

Retroactive testing of blood samples has confirmed that the earliest case identified in the United States occurred in 1968 in 16-year-old Robert Rayford, who had never traveled out of the Midwest and never received a blood transfusion, suggesting the virus was present in the United States before 1966.

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HIV Infection

There are 2 species of HIV (HIV-1 and HIV-2) and multiple subtypes (referred to as clades). HIV-1 and HIV-2 are both retroviruses in the Retroviridae family, *Lentivirus* genus. They are single-stranded RNA virus. HIV-1 is almost exclusively found in the developed world while HIV-2 is more often found in third world countries, such as those in Africa. HIV-1 is easier to transmit than HIV-2 and progresses more quickly to AIDS because the viral load tends to be higher. HIV-1, clade B, is the most common in the developed world although all clades of HIV-1 have similar disease.

There are three primary mechanisms by which HIV syndromes occur:

- **Immunodeficiency:** This results from the direct impact HIV has on the immune cells, and resulting in increased susceptibility to infections and neoplasms.

- **Autoimmune reaction:** Dysfunction of B-lymphocytes occurs and impaired cellular immunity, decreasing the immune response to antigens.

- **Hypersensitivity reactions:** Increased allergic response to allergens and medications.

The average time period from infection with HIV to onset of AIDS is about 10 years. Many people remain essentially asymptomatic for years, increasing the risk that they can spread the infection to others. Early symptoms, however, may occur within one or two months of exposure.

When beginning signs and symptoms do occur, they are often very non-specific and flu-like, so the person and healthcare providers may not suspect HIV.

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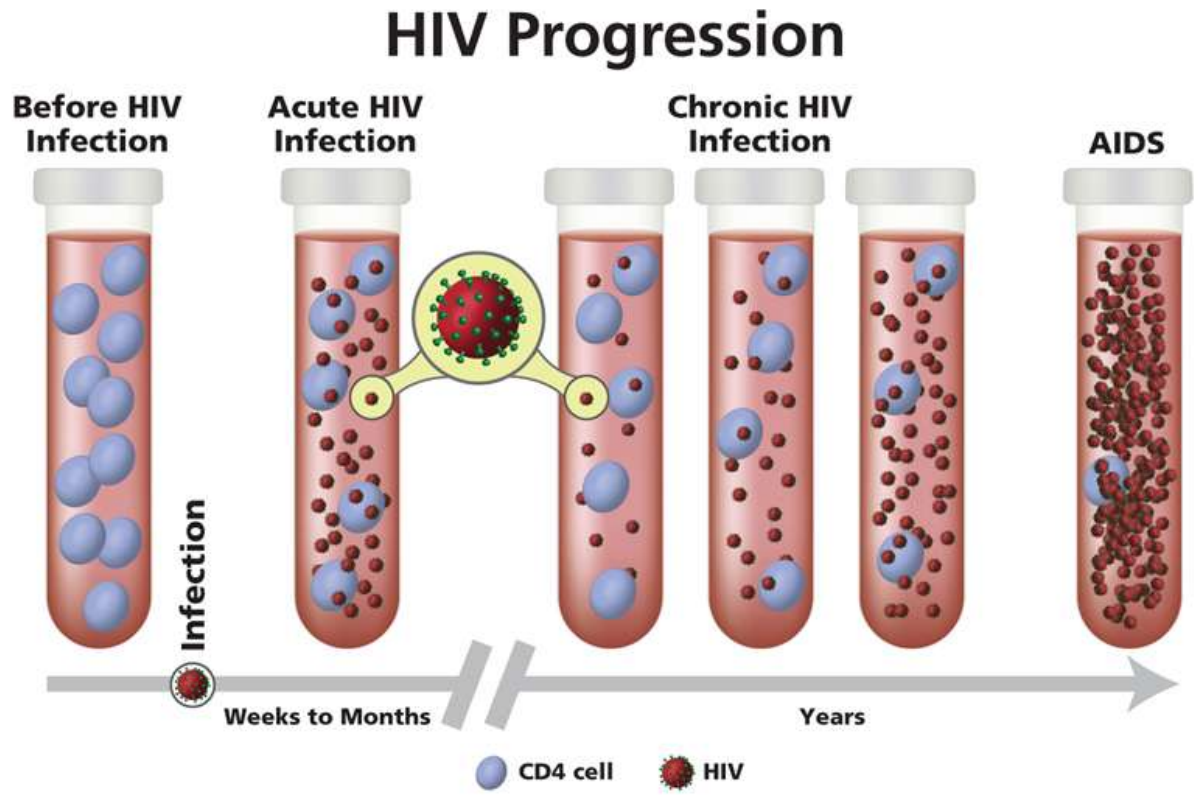
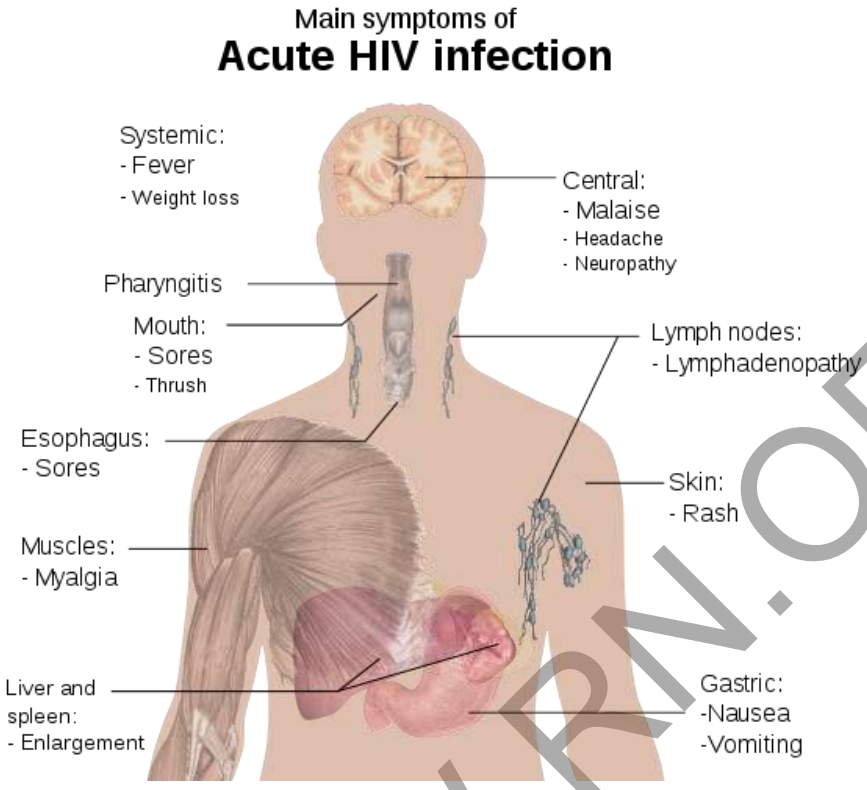


HIV stages

1. Acute HIV infection (Seroconversion)



Those infected may experience flu-like symptoms 2 to 4 weeks after exposure. During this stage, the virus is multiplying rapidly and spreading throughout the body, attacking CD4 cells of the immune system. A provirus reservoir of persistently infected cells is created, and the size of this reservoir correlates with the viral load.

The level of HIV in the blood is typically quite high at this stage, making the individual at high risk of transmitting the disease. The CD-4 count often drops precipitously.

As the body mounts a defense, the CD-4 count returns to normal range (but usually lower than before infection). Beginning treatment during this stage provides significant health benefits.

2. Chronic HIV infection

This stage is generally asymptomatic (clinical latency) but the HIV continues to multiply although at lower levels. Treatment at this stage can slow the progression of the disease for several decades; but untreated, the disease will progress to AIDS within 10 years in most people (although much faster

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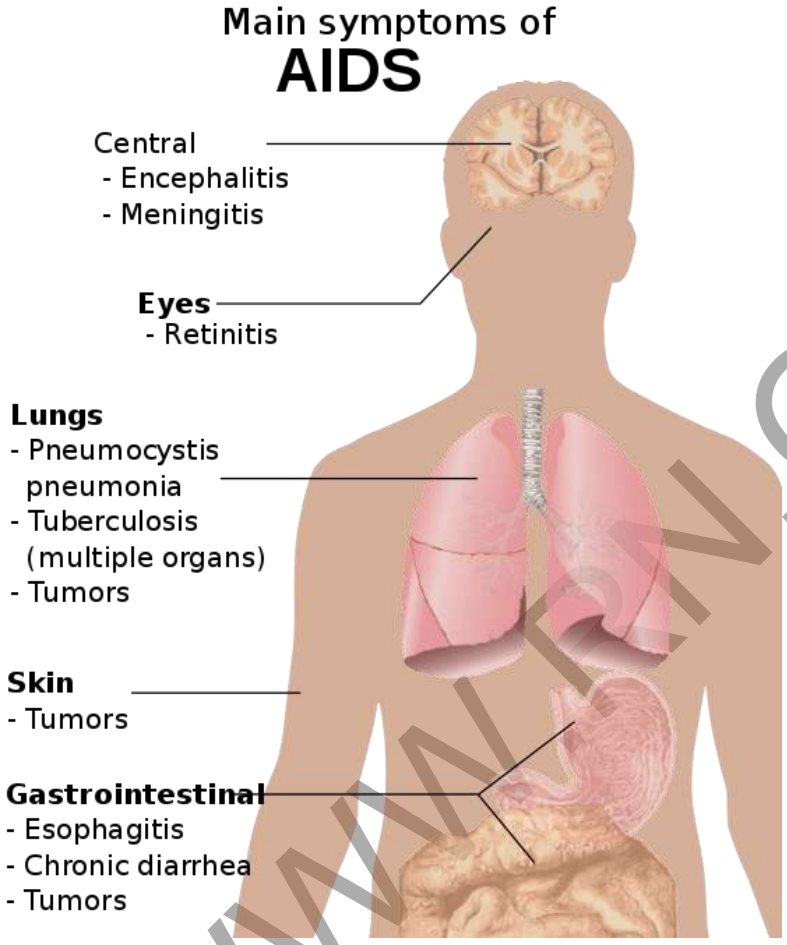
in some). It is possible to transmit the infection during this stage, but those receiving treatment and maintaining an undetectable viral load have low risk of transmitting HIV.

3. AIDS



This is the end stage in which the immune system has been severely impaired and opportunistic infections and diseases occur. Diagnostic criteria include a CD4 count of less than 200 cells/mm^3 or having an AIDS-defining condition (such as Kaposi's sarcoma).

Once a person is diagnosed with AIDS, the person can readily transmit HIV infection to others because of the high viral load. Untreated, the life expectancy after diagnosis with AIDS is about 3 years.

The CDC categorizes HIV infections according to symptoms:

- Category A: Asymptomatic HIV infection.
- Category B: Some symptoms present and related to HIV.
- Category C: AIDS-defining opportunistic infection(s) present.

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Diagnostic procedures

A series of tests are usually done to diagnose HIV/AIDS. Positive results should be verified by confirmatory testing. The US Preventive Services Task Force (USPSTF) recommends screening of all pregnant women and all adolescents and adults at increased risk. The CDC recommends screening for all patients in healthcare settings and annual screening for those at high risk.

HIV/AIDS diagnostic procedures	
HIV ELISA	Screening test. 95% test positive within 6 weeks of exposure.
Western blot	Confirmatory test. (Greater than 99.9% specificity combined with ELISA.)
HIV rapid antibody	Screening test produces results in 10-20 minutes, but positive results should be confirmed with ELISA or Western blot. False positives can occur.
CBC	Identifies blood disorders common to HIV/AIDS, such as anemia, neutropenia, and thrombocytopenia
CD4 absolute	Predicts HIV progression.
CD4 %-age	If percentage <14%, risk of opportunistic infection is high without treatment. Treatment <25% usually indicates the need for treatment. (Note: May be more reliable than the absolute count, especially for children under 5 and adults with chronic hepatis C and low absolute counts).
Viral load	Predicts HIV progression. Low levels (<500 copies/mL) may be false positives. Levels greater than 30,000 indicate high risk for death from AIDS.

AIDS-defining conditions are many and varied, and are important considerations when determining when a person has progressed from HIV to AIDS. Opportunistic infections tend to vary by geographical area and reflect the pathogens common to the area. While more common when the CD4 count has dropped below 200 cells/mm^3, AIDS-defining conditions may occur at higher levels as well and are considered diagnostic for AIDS.

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Table. The CDC's AIDS-defining conditions

- Bacterial infections, multiple or recurrent*
- Candidiasis of bronchi, trachea, or lungs
- Candidiasis of esophagus†
- 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 >1 month
- Cytomegalovirus retinitis (with loss of vision)†
- Encephalopathy, HIV related
- Herpes simplex: chronic ulcers (>1 month's duration) or bronchitis, pneumonitis, or esophagitis (onset at age >1 month)
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (>1 month's duration)
- Kaposi sarcoma†
- Lymphoid interstitial pneumonia or pulmonary lymphoid hyperplasia complex*†
- Lymphoma, Burkitt (or equivalent term)
- Lymphoma, immunoblastic (or equivalent term)
- 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 jiroveci pneumonia†
- Pneumonia, recurrent†
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia, recurrent
- Toxoplasmosis of brain, onset at age >1 month†
- Wasting syndrome attributed to HIV

* Only among children aged <13 years.

† Condition that might be diagnosed presumptively.

§ Only among adults and adolescents aged >13 years

HIV infection cycle

HIV can be transmitted through sexual intercourse, shared IV drug paraphernalia, mother-to-child transmission during birth or during breastfeeding, blood transfusions (infected blood), organ transplantation, and contact with body fluids.

There are 7 stages to the HIV life cycle:

1. **Binding/Attachment:** HIV attaches to receptors on CD4 cells.
2. **Fusion:** The envelope of HIV fuses with the cell membrane of CD4, allowing the HIV to enter the cell.
3. **Reverse transcription:** HIV release reverse transcriptase (an enzyme) to convert its genetic material from HIV RNA to HIV DNA, which allows the HIV DNA to enter the cell's nucleus and combine with the CD4's DNA.
4. **Integration:** Within the nucleus, HIV releases integrase (an enzyme) to insert its DNA into the CD4's DNA
5. **Replication:** After integration into the CD4's DNA, HIV begins to use the CD4 cells processes to produce long chains of HIV proteins (building blocks for HIV).

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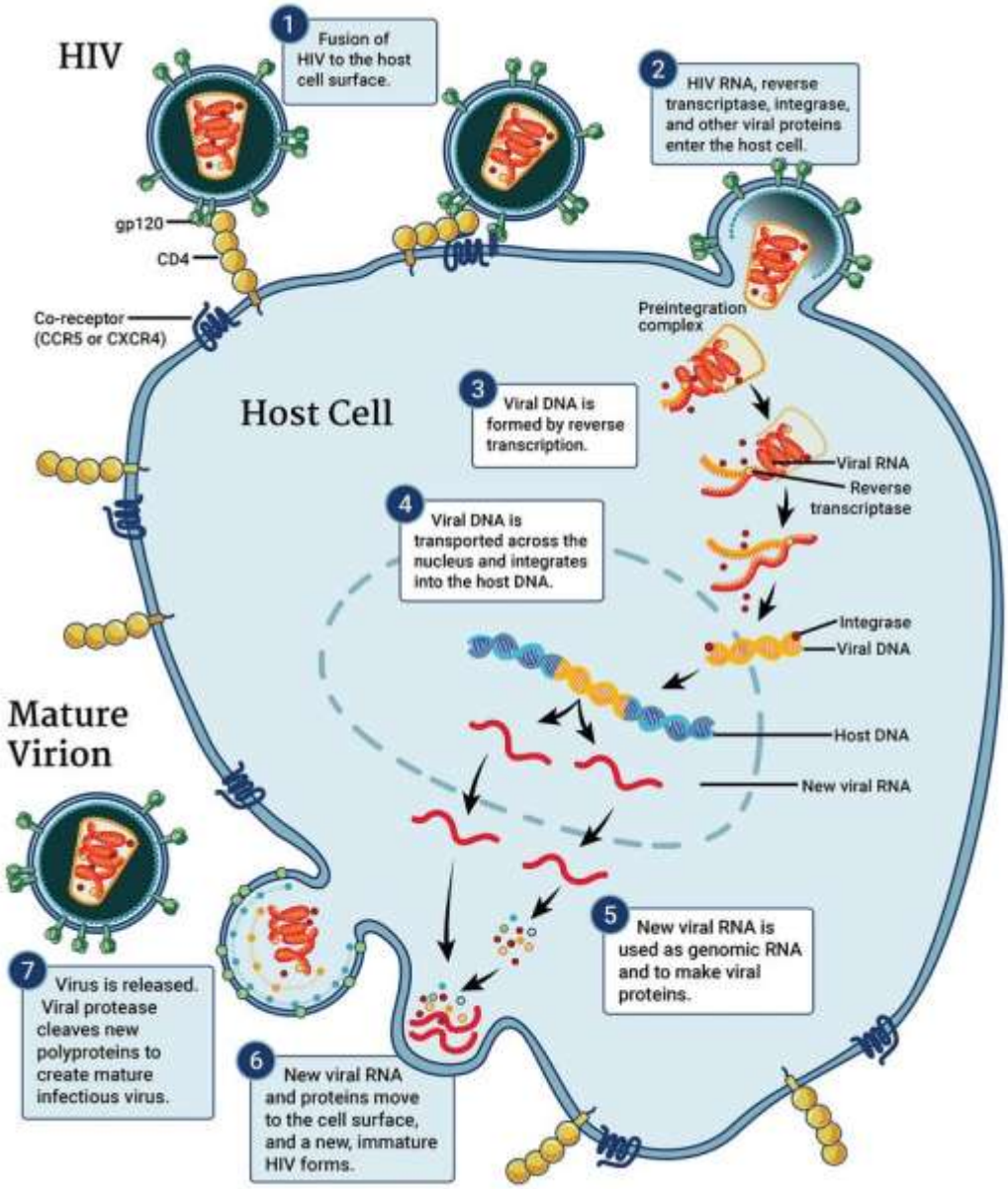
6. **Assembly:** New HIV proteins and HIV and RNA migrate to the cell surface and form immature (non-infectious) HIV.
7. **Budding:** The newly formed immature HIV push out of the CD4 cell and release protease (an enzyme), which breaks apart the long protein chains found in the immature HIV, allowing smaller HIV proteins to combine to make mature HIV.



NIAD/NIH

Scientists at Westmead Institute for Medical Research recently discovered that the epithelial layer of genital tissue in the surface of the vagina, inner

foreskin, and anus contains a type of previously undiscovered immune cell: CD11c+ dendritic cells. These cells appear to be the first line of defense and capture pathogens (such as HIV) and carry them to CD4 T cells. Unfortunately, the CD4 T cells are the target of HIV, and the CD11c+ dendritic cells are more susceptible to HIV infection than any other known dendritic cells. Thus, these cells are the key drivers of HIV infection. This discovery provides a potential target for drug manufacturers to find a drug or vaccine that blocks the transmission of HIV before it can reach the CD4 cells.

Treatment

Treatment for HIV/AIDS targets different stages of the HIV life cycle in order to stop the production of mature HIV:

1. **Binding/Attachment**: CCR5 antagonist, and post-attachment inhibitors.
2. **Fusion:** Fusion inhibitors.
3. **Reverse transcription:** Non-nucleoside reverse transcriptase inhibitors (NNRTIs) and nucleoside reverse transcriptase inhibitors (NRTIs),
4. **Integration:** Integrase inhibitors.
5. **Replication:** (No medication).
6. **Assembly:** (No medication).
7. **Budding:** Protease inhibitors (PIs).

A combination of treatments, generally now referred to as simply antiretroviral therapy (ART), is used in order to place barriers at multiple stages. There are 7 classes of drugs as well as pharmacokinetic enhancers.

Antiretroviral therapy (ART)		
Nucleoside reverse transcriptase inhibitors	Block reverse transcriptase, an enzyme needed by HIV to make copies of itself.	Abacavir (ABC), emtricitabine (FTC), lamivudine (3TC), tenofovir disoproxil fumarate (TDF), and zidovudine (AZT,ZDV)
Non-nucleoside reverse transcriptase inhibitors	Bind to and alter reverse transcriptase.	Doravirine (DOR), efavirenz (EFV), etravirine (ETR), nevirapine (extended release, NVP), and rilpivirine (RPV)

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Protease inhibitors	Block HIV protease, an enzyme needed by HIV to make copies of itself.	Atazanavir (ATV), darunavir (DRV) fosamprenavir (FOS-APV, FPV), ritonavir (although a PI, it is often used as a pharmacokinetic enhancer, RTV), saquinavir (SQV), and tipranavir (TPV)
Fusion inhibitor	Blocks HIV from entering CD4 cells of the immune system.	Enfuvirtide (T-20)
CCR5 Antagonist	Block CR5 coreceptors on the surface of certain immune cells. HJIV needs to attach to these receptors to enter the cells.	Maraviroc (MVC)
Integrase strand transfer inhibitors (INSTIs)	Block HIV integrase, an enzyme needed by HIV to make copies of itself.	Dolutegravir (DTG) and raltegravir (RAL)
Post-attachment inhibitor	Blocks CD4 receptors on the surface of certain immune cells. HIV needs to attach to these receptors to enter the cells.	Ibalizumab-uiyk (IBA, TMB-355, TNX-355, Hu5A8)
Pharmacokinetic enhancer	Increases the effectiveness of HIV medicine	Cobicistat (COBI, c)

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In addition to these single medications, there are many combinations of two to four drugs available. Examples include:

- Epzicom (abacavir and lamivudine).
- Trizivir (abacavir, lamivudine, and zidovudine).
- Symtuza (darunavir, cobicistat, emtricitabine, and tenofovir alafenamide).
- Dovato (Dolutegravir and lamivudine).
- Symfi (efavirenz, lamivudine, and tenofovir disoproxil fumarate).

While at one time treatment was delayed until the CD4 count had fallen, it is now recommended that all patients with a diagnosis of HIV immediately begin treatment with ART, ideally within the first 6 months of exposure.

Patients usually begin treatment with at least three medications from at least two different classes of drugs. A common first HIV regiment would include two NRTIs plus an INSTI, an NNRTI, or a PI boosted with cobicistat (Tybost®) or ritonavir (Norvir®). Cobicistat or ritonavir boost the effectiveness of the PI.

Many factors can affect the choice of drugs: pregnancy, other diseases, side effects, possible drug interactions, drug resistance, cost, and convenience (combined therapy is easier to manage than multiple individual drugs).

The goal of therapy is to reduce the viral load to undetectable, and this usually takes 3 to 6 months if the ART is effective. If a person takes combinations of three or more antiretroviral drugs, this is also referred to as highly active ART or HAART.

Monitoring of therapy to determine status initially after diagnosis and then to determine the effectiveness of therapy is carried out with two tests:

- **HIV viral load test**: This measures the number of HIV particles ("copies") in a milliliter of blood. Only about 1% of HIV is actually found in the blood, but it remains an effective marker.

  There are three types of viral load tests: PCR, bDNA, and nucleic acid sequence-based amplification (NASBA). Viral load tests have a 3-fold margin of error, so a result of 30,000 could mean the actual result is between 10,000 and 90,000.

  Additionally, each viral load test has a sensitivity cut-off. That is, the lowest level of HIV that the test can detect. This generally ranges from 20 to 50 copies/mL. A viral load of 10,000 is generally considered low and 100,000, high. Failure to respond to therapy is defined as a viral load greater than 200.

- **CD4 count:** A normal CD4 count ranges from 500 to 1600 cells/mm^3. A count lower than 200 cells/mm^3 is diagnostic of AIDS because the immune system is too impaired to effectively fight disease.

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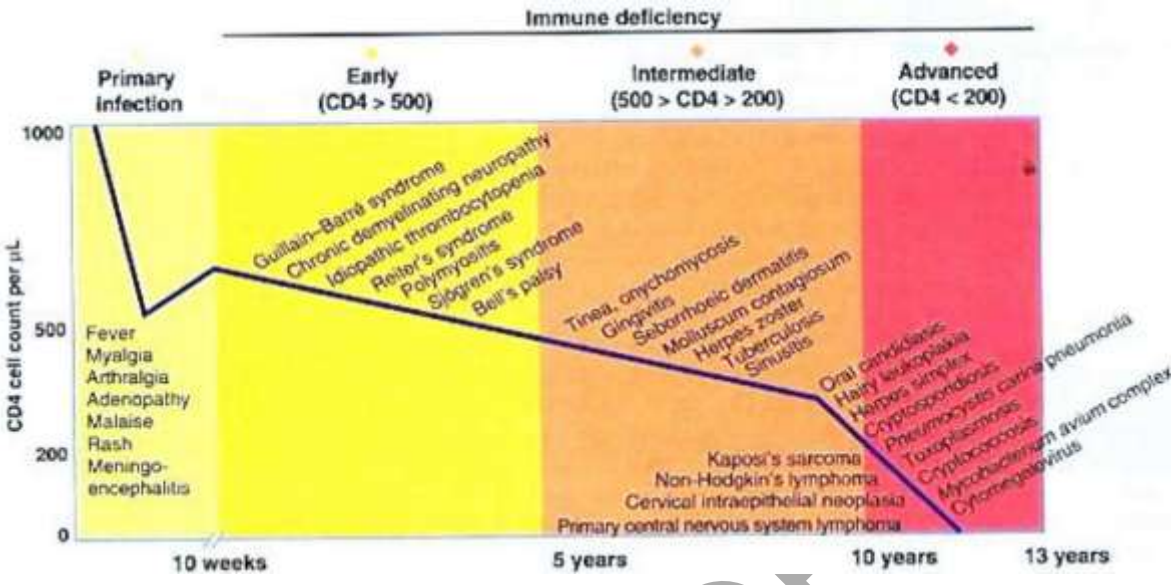

When treating HIV, the viral load test is more important than the CD4 test because the inability to get the viral load count below 50 indicates a poorer prognosis. As the viral load increases, the CD4 count falls. The goal of therapy is to decrease the viral load to undetectable. Even though a person's viral load becomes undetectable, this does not mean the person no longer has HIV, only that it is not detectable in the blood. Viral load may be higher in other tissue and fluids.

If the viral load remains detectable after three to six months of ART, then the patient is at increased risk of becoming resistant to current ART as well as other classes of drugs.

It's important to note that people sometimes experience a "blip," a sudden increase, in viral load before the level falls again. This is not usually cause for concern and may indicate an infection (cold, influenza) or variation in laboratory procedure. If, however, the elevation remains through two consecutive tests or tends to recur, a change in treatment may be indicated.

Prophylaxis/Suppressive therapy

Because patients whose viral load is high and CD4 count low are susceptible to many types of infections, prophylaxis or suppressive therapy is often administered:

Infection	Indications	Treatment
Pneumocystis jiroveci	CD4 count drops below 200 cells/mm^3, lymphocyte %-age less than 14%, or oral candidiasis	TMP-SMX DS, dapsone, atovaquone or aerosolized pentamidine

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M. avian	CD4 count drops below 75-100 cells/mm^3.	Clarithromycin or azithromycin (preferred)
M. tuberculosis	All with positive PPD (greater than 5 mm induration)	INH and pyridoxine
Herpes simplex virus 1 and 2	Those receiving ART (ineffective in those not receiving ART)	Acyclovir or other antiviral.
Toxoplasmosis	Positive IgG toxoplasma serology and CD4 count below 100 cells/mm^3	TMP-SMX DS or pyrimethamine plus dapsone and leucovorin
Cytomegalovirus	CD4 count below 50 cells/mm^3	Ganciclovir or letermovir

Pre-exposure prophylaxis (PrEP)

People who are uninfected with HIV but are at high risk, such as males having sex with males, partners of patients with HIV/AIDS, IV drug users, and sex workers, may be treated with pre-exposure prophylaxis. Currently, there is one drug marketed: Emtricitabine/tenofovir (Truvada®). Those taking Truvada® must be tested for HIV before beginning the treatment and then at least every 3 months. People taking the drug are urged to use safe sex practice, such as using latex or polyurethane condoms and lubrication, and to be tested for other STDs, as these may increase risk of HIV infection.

Post-exposure prophylaxis

Post-exposure prophylaxis must be initiated without 72 hours of possible exposure, and the sooner the better. PEP is taken twice daily for 28 days and is generally effective in preventing HIV infection (although not 100).

PEP regimens include:

- Basic 2-drug PEP: Tenofovir plus emtricitabine (preferred); alternatives include zidovudine plus lamivudine, zidovudine plus emtricitabine, or tenofovir plus lamivudine
- Alternative: Lamivudine plus stavudine, lamivudine plus didanosine, emtricitabine plus stavudine, or emtricitabine plus didanosine
- Expanded: Basic PEP regimen plus raltegravir (preferred) or lopinavir-ritonavir or a number of other drugs.

Patterns of opportunistic infections vary according to geographical areas, reflecting the pathogens common to the area. Complications can involve any body system.

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Complications/Opportunistic conditions

Pulmonary complications

Chronic sinusitis:
Sinusitis is a common problem for patients with HIV infection even if they are adequately treated with ART. Patients often complain of congestion, discharge, headache, and fever. Some may be asymptomatic despite imaging evidence of infection. If the patient's CD4 count is low and viral load high, these symptoms may indicate *Pseudomonas* infection.

Neoplasms:
Patients may develop neoplasms affecting the pulmonary system, including lung cancer, Kaposi's sarcoma (not usually the initial site), and non-Hodgkin lymphoma (which may involve multiple organs as well as the lungs). Patients may develop pleural effusions and exhibit nodular or diffuse involvement of the lung parenchyma.

Pneumocystis jiroveci pneumonia:
The most common opportunistic pulmonary infection is *Pneumocystis jiroveci* (formerly *carinii*) pneumonia but rarely occurs with CD4 counts greater than 250 cells/mm^3. This infection may present with nonspecific symptoms (fever, cough, dyspnea) or severe hypoxemia (PO2 <60 mm Hg). Chest x-ray often shows infiltrates (diffuse or perihilar) but large pleural infusions are uncommon. LDH is almost always elevated but is nonspecific. Non-specific interstitial pneumonia may occur and can mimic *Pneumocystis* pneumonia.

Tuberculosis:
About 4% of those with AIDS in the United States develop tuberculosis. Because a low CD4 count can interfere with PPD testing, those with suspected TB should be tested with interferon gamma release assays, such as the QuantiFERON and T-SPOT tests. If the patient is receiving a protease inhibitor, the patient should not be given rifampin. Multi-drug resistant TB has become an increasing problem and requires the patient receive at least three medications to which the disease is sensitive. Patients may develop atypical mycobacterial infections that mimic TB, but they should be treated as for TB until a definitive diagnosis is made.

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Cardiovascular complications

HIV-associated cardiomyopathy:
Many of the ART drugs can directly or indirectly cause cardiovascular disease. Dilated cardiomyopathy is the most common cardiovascular problem and is sometimes associated with doxorubicin, a key drug in the treatment of Kaposi's sarcoma. Zidovudine (AZT) is associated with reversible damage to the cardiac muscle cells.

Patients may present with heart failure with left ventricular dysfunction and with or without ventricular dilation. Myocardial inflammation resulting from HIV is a primary cause of HIV-associated cardiomyopathy, and risk increases markedly with CD4 counts of less than 400 cells/mm^3. Viral infections may trigger cardiac autoimmune reactions that lead to cardiomyopathy, and micronutrient deficiency may also play a role.

Lipodystrophy syndrome:
This condition is characterized by fat redistribution (similar to that seen in Cushing's disease) in which subcutaneous fat decreases in the trunk but increases in the visceral area, resulting in a change in body fat. Other changes include a buffalo hump (dorsocervical fat), axillary fat pads, and loss of fat in the face, buttocks, arms, and legs.

Associated metabolic disorders include hypertriglyceridemia, and hypercholesteremia, hyperglycemia, and insulin resistance (similar to syndrome X). Lipodystrophy syndrome is caused by protease inhibitors, especially ritonavir. Some researchers also believe that NRTIs may be causative agents. Because of potential drug interactions, simvastatin and lovastatin, bile sequestrants, and niacin are usually avoided in HIV/AIDS patients taking multiple drugs.

Pericardial disease:
One of the leading causes of pericardial disease is *Mycobacterium tuberculosis*-associated pericarditis, and those with coinfection with TB myopericarditis have a much greater risk of cardiac death. Additionally, HIV-patients with pericarditis have a higher rate of relapse than non-HIV patients.

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Central Nervous System (CNS) complications

CNS lymphoma:
Primary non-Hodgkin lymphoma is a space-occupying lesion and produces symptoms similar to toxoplasmosis although lesions tend to be single rather

than multiple. Patient may exhibit focal neurologic deficits, headache, blurred vision, seizures, motor abnormalities, personality and/or cognitive changes, and confusion. Patients may benefit from radiation treatment.

Cognitive impairment/HIV-associated dementia:
Cognitive impairment is a common finding, especially in patients over 50 even if they respond well to ART. Patients may have problems with memory and attention as well as impaired motor function and emotional or behavioral problems. Changes in handwriting is often one of the first indicators.

Herpes zoster(shingles):
HIV/AIDS increases the risk of developing herpes zoster (shingles), which can cause inflammation of the brain and spinal cord. Herpes simplex meningoencephalitis causes behavioral, personality, and memory changes and is most common when the CD4 count is less than 200 cells/mm^3. Varicella zoster infection leads to vasculitis and stroke syndromes.

HIV myelopathy:
HIV myelopathy often occurs with encephalitis and is most common in late HIV infection. Patients usually exhibit spinal cord dysfunction with weakness of the legs, spastic paresis, and incontinence. Vacuolar myelopathy, which causes tiny holes to develop in nerve fibers of the spinal cord, results in difficulty walking. Vacuolar myelopathy is most common in children and untreated adults.

Neurosyphilis:
Patients with untreated syphilis can progress rapidly to neurosyphilis, leading to dementia, loss of hearing and vision, and difficulty walking.

Progressive multifocal leukoencephalopathy (PML):
This viral infection of the brain's white matter usually occurs late in HIV infection. Patients typically exhibit focal neurologic defects (hemiparesis, aphasia, cortical blindness).

Toxoplasmosis:
Toxoplasmosis is the most common cause of space-occupying lesions in patients with HIV/AIDS. Patients may experience headaches, seizures, focal deficits, and altered mental status. Patients usually develop multiple rather than single lesions.

Viral meningoencephalitis:
General symptoms include headache, fever, confusion, delirium, lethargy, focal deficits, and seizures. CMV infection tends to worsen focal deficits and is most common when CD4 count is less than 50 cells/mm^3. Patients with

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cryptococcal meningitis typically present with headache and fever. HIV meningitis is common in the early stages of HIV infection.

Peripheral nervous system (PNS) complications

Cytomegalovirus ascending polyradiculopathy:
This is characterized by weakness of the lower extremities. CMV can also cause transverse myelitis.

Inflammatory demyelinating polyneuropathy:
Symptoms are similar to Guillain-Barré syndrome and occur before severe immunodeficiency. Because the condition often responds to plasmapheresis, it is believed to be an autoimmune response.

Peripheral neuropathy:
Numbness, pain, and tingling of the lower extremities is common in HIV patients. The neuropathy may result from HIV itself or from treatment with stavudine (not often used in the United States) or didanosine. If induced by medication, the symptoms may not be reversible. Gabapentin may provide some relief.

Musculoskeletal complications

HIV-associated arthritis:
Arthritis may affect one or multiple joints, but usually affects large joints. Joint effusions may occur, requiring aspiration. Patients often respond to NSAIDs.

Myopathy:
Myopathy, which was common with zidovudine (AZT), infrequently occurs with the more effective ART that is now available. However, some patients may still develop myopathy, usually characterized by proximal muscle weakness.

Osteoporosis/Osteopenia:
Osteoporosis is more common in those with chronic infection and long-term treatment with ART. Patients with HIV often have vitamin D deficiency which may interfere with absorption of calcium, resulting in bone loss. Vitamin D levels should be monitored and bone density scans done for patients over age 50.

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Opthalmic complications

Retinitis:
Retinitis is most commonly caused by CMV infection and is characterized by perivascular hemorrhage and white fluffy exudates. (These are distinct from the cotton wool spots that are common and benign.) This condition may progress rapidly, and patient should immediately be referred to an ophthalmologist.

Gastrointestinal complications

AIDS-related diarrhea/Enterocolitis:
Diarrhea is a common complication of AIDS and may persist for months and may be severe (gallons of liquid feces daily), increasing the risk of dehydration. Patients often have bloody stools, high fever, and severe cramping. Diarrhea is most often caused by infection with CMV, parasites (*Cryptosporidium*, microsporidia, *Giardia lamblia*), and bacteria, such as *Mycobacterium avium-intracellulare*.

Other causes may include antibiotics or drugs that interfere with normal intestinal flora, HIV enteropathy from disease processes (such as Kaposi's sarcoma), and lactose intolerance (a common finding). Treatment depends on identifying the underlying cause.

Cholecystitis:
Symptoms are typical but stones are usually not present; rather, sclerosing cholangitis and papillary stenosis (distal common bile duct) is common. Patients often have severe nausea, vomiting, and RUQ pain.

Hairy leukoplakia (oral):
This condition is triggered by the Epstein-Barr virus and is most common in those with HIV and immunocompromise. Hairy leukoplakia results in hairy-appearing white patches on the tongue and other parts of the mouth. The lesions are generally painless but cannot be removed with a toothbrush. The appearance of these lesions is often an indication of worsening of HIV. Smokers have increased risk of developing hairy leukoplakia.

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Hepatic disease:
Patients co-infected with hepatitis may have rapid progression of liver disease. The liver is also often the site of infections and neoplasms. Some medications (sulfonamides, anti-tuberculosis medications) may cause hepatitis. NRTIs all can cause lactic acidosis, which can lead to death, but the risk is greatest with the didanosine-stavudine combination (no longer recommended).

Malabsorption syndrome:
Patients with HIV/AIDS typically produce less gastric acid than normal, resulting in inadequate absorption of medications that are dependent on those acids, and the deficient acid may also increase susceptibility to infections from acid-sensitive organisms, such as *Campylobacter*, *Salmonella*, and *Shigella*. Malabsorption syndrome may also affect the small intestines, usually associated with infection with *M. avium* complex, *Cryptosporidium*, or microsporidia.

Oral/Esophageal candidiasis:
The impaired immune system allows the overgrowth of the candidal fungus ("thrush"), which can infect the oral and esophageal mucosa, resulting in pain, white patches, sore throat, and difficulty swallowing. Candidiasis can occur with patients who are receiving ART but is most common when the immune system is severely compromised, especially if the CD4 count is less than 200 cells/mm^3.

In some cases, candida infection can invade the skin and single or multiple organs. Candidiasis usually responds to antifungals, such as fluconazole, or echinocandins (IV drugs).

Endocrinological complications

Hypogonadism:
Hypogonadism (decreased sperm or testosterone production) affected about 50% of males prior to the use of ART, but the rate is still about 15 to 20% and the cause is not clear although it is associated with lower levels of testosterone, so the standard treatment is hormone replacement. Signs and symptoms may include erectile dysfunction, loss of libido, increased fatigue, and increased fat. Hypogonadism is often associated with decreased muscle and bone mass as well.

Pancreatic dysfunction:
Patients treated for pneumocystis pneumonia with pentamidine (a beta cell toxin) are at increased risk (15-28%) of developing pancreatic dysfunction

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and hypoglycemia. These patients may develop diabetes mellitus. Hypoglycemia may occur weeks or months after discontinuing pentamidine treatment because of its long half-life.

Dermatologic complications

Bacillary angiomatosis:

This condition may be caused by *Bartonella henselae* and *Bartonella quintana* and is typically transmitted from fleas of infected cats but can develop from traumatic cat exposure (bites, scratches). The lesions are often similar to those of Kaposi's sarcoma. The lesions may vary in color, size, and appearance and may be primarily superficial or involving subcutaneous tissue.

Additionally, the infection can invade bone and internal organs (liver, spleen, lungs, heart, intestines, and CNS). Those with skin lesions may develop bacteremia and cutaneous lesions may appear over areas of osseous infection.

Herpes zoster and herpes simplex infections:

Both types of herpes infections are common manifestations of HIV and they are often more severe than in non-HIV patients and more likely to disseminate systemically, so all patients should be treated with antiviral agents, such as acyclovir.

The herpes zoster vaccine appears to be safe for HIV patients over 50 and with CD4 counts of greater than 200 cells/mm^3.

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Molluscum contagiosum:

Molluscum contagiosum, caused by the pox virus (a double-stranded DNA virus), is common in HIV patients, affecting 10-20%. The pearly, dome-shaped paponodular lesions tend to spread over the face and neck as well as other parts of the body, and patients often have up to 100 lesions or more.

The infection can become chronic and progressive. In advanced disease, giant or verrucous forms may occur. MC is treated with liquid nitrogen, laser, surgical excision, cytodestructive methods, (iodine, silver nitrate, and tretinoin), chemotherapeutic agents, and antivirals.

***Staphylococcus* infection:**

Staph infections, including MRSA, are the most common bacterial skin disorder in HIV patients and may present as folliculitis, abscesses, or bullous impetigo and may progress to systemic infections, such as sepsis. Topical treatment (clindamycin, mupirocin) is typically applied but some may require oral antibiotics, nasal prophylaxis, and incision and drainage (abscesses).

HIV-associated malignancies

Many different types of cancers can develop in patients with HIV/AIDS infection, but a few types are most common.

Anal dysplasia/ Squamous cell carcinoma:

Invasive squamous cell cancers of the anus are associated with human papillomavirus infection (HPV 16 and HPV 18). In the HIV-infected patient, dysplasia caused by HPV is at increased risk of progressing to malignancy.

Hodgkin's lymphoma:

The incidence of Hodgkin's lymphoma is 5 to 10 times higher in HIV patients than the non-HIV population, and the incidence has increased since the initiation of combination ART. In almost all cases of Hodgkin's lymphoma in HIV patients, the Epstein-Barr virus is detected. Although patients may

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initially respond to treatment for HL, relapse rates are high in those with HIV/AIDS.

Kaposi's sarcoma:
KS, caused by the human herpes virus 8 (HHV-8), develops from cells that line lymph and blood vessels. The cells divide rapidly and spread into the surrounding tissues, resulting in dark (red, purple, brown, black) lesions on the body, typically the legs or face although they can appear anywhere, such as in the mouth or eye. About 40% of those with cutaneous lesions will develop visceral disease (lungs, GI tract). KS is an AIDS-defining condition. ART may prevent development of KS or slow the progression of the disease. KS was very common in HIV/AIDS patients before the use of ART but incidence has fallen.

Those with CD4 counts below 200 cells/mm^3 are at greatest risk although about 40% have higher CD4 counts, especially in older males who have taken ART for many years. At times, KS can flare up in the first months after a patient begins ART because of immune reconstitution inflammatory syndrome.

Non-Hodgkin lymphoma (AKA HIV-related lymphoma):
NHL is a diffuse large cell cancer of lymphocytes (typically B cells) and can occur in HIV patients even with a high CD4 count although those with low CD4 counts are most at risk. NHL can be very aggressive with higher mortality rates than the general public. HIV patients have 10 times the risk of developing NHL than in the general population. Symptoms may vary and often begin with a painless mass in the neck, axilla, or femoral area. If the lymphoma occurs in the GI system, patients may experience abdominal pain, indigestions, and weight loss; if in the chest, cough, dyspnea, and dysphagia. Patients may complain of fever and night sweats.

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Preventive measures

Preventive measures for individuals include:

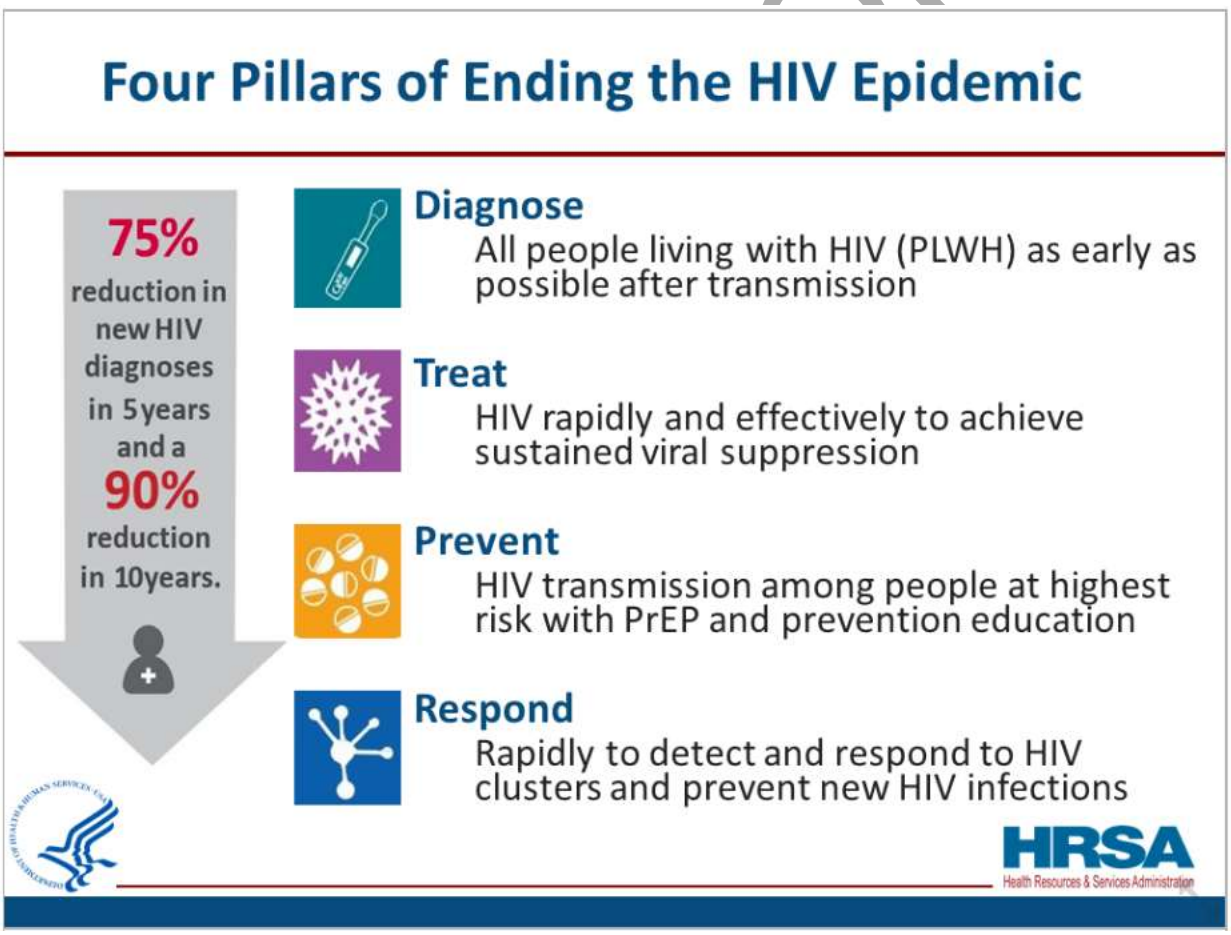
- Practicing safe sex: limiting partners, talking to partners about HIV status, using condoms and lubricants.
- Taking pre- or post-exposure prophylaxis (as above).
- Undergoing HIV testing and counseling.
- Avoiding contact with body fluids.
- Donating own blood prior to elective procedures.

Preventive measures for healthcare include:

- Utilizing universal standard precautions in healthcare settings.
- Avoiding contact with body fluids.
- Reducing needlestick injuries through use of safety needles and avoidance of recapping.
- Promptly treating and reporting needlestick injuries (wash with soap and water, HIV testing, prophylaxis for 4 weeks).
- Screening of all blood products and using the least amount necessary.
- Utilizing appropriate screening to identify patients with HIV or at risk.

Conclusion

The United Nations has established a goal of eliminating the public health threat of HIV by the year 2030. Education regarding HIV/AIDS, including both the public and healthcare providers, is essential in achieving this goal.



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