



AIDS: Where the Pendulum Swings



Indian Institute of Sexology
Bhubaneswar

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Editorial

One of the super medical challenges that has boggled the human mind over the last few decades ever since it was first identified, Acquired Immune Deficiency Syndrome or AIDS has kept the global medical fraternity on its toes. Caused by a retrovirus called Human Immunodeficiency Virus (HIV), AIDS has become a real global pandemic staring at our face. It can affect individuals irrespective of their age, gender and race. An individual infected with HIV, progress to AIDS over several years (10-15 years). As per a WHO report, globally, by November 2015, 34 million people died of HIV infection. By the end of year 2014, 36.9 million people were living with HIV infection, worldwide. It has been observed that HIV infection is most rampant in the Sub-Saharan Africa.



Cover Art : Nivedita Pramanik

The viral pathogen responsible for causing AIDS gets transmitted from one individual to other when the contaminated body fluid comes in contact with the denuded surface or blood of the healthy individual. Sexual transmission is the commonest modes of transmission of HIV, however, various non-sexual modes like – transplacental, blood transfusion, sharing of needle also serve as some of the other modes of transmission of HIV.

The morbidity and mortality associated with AIDS are the direct and indirect consequences of immune suppression. Severe life-threatening infections, malignancies involving multiple organ systems attribute to these serious outcomes. Mental health morbidities have also been found to common in patients suffering from AIDS, which further affects the quality of life of the individuals.

A lot of advancement has been recorded over the last four decades in various aspects of management and prevention of AIDS. However, till now no definite, curative treatment for AIDS has become possible. Anti Retroviral Treatment (ART) has been found to prolong the life of persons living with AIDS and improve their quality of life. In the year 2015, around 16 million people were receiving ART on record, worldwide. Initiatives by WHO has also been reinforced and expanded significantly. Considering these, it is anticipated that by 2030, 21 million AIDS related deaths can be prevented.

Although we don't have any specific curative modality for AIDS, we have a strategy that is better than cure, and that is 'Prevention'. This has led a majority of the current management goals to be centred on various modalities of prevention. Prevention of AIDS ranges from building awareness about AIDS to the rehabilitation of AIDS patients and improvement of their quality of life.

Despite all these, a lot of stigmas, social and otherwise, are still associated with AIDS. The patients suffering from AIDS are not only becoming subjects to these stigmas, but also their caregivers. Stigma again results in significant psychological distress for the victims. Lack of awareness is one of the major causes of these stigmas. Awareness decreases stigma and make people alert about healthy sexual practices as well as generates empathy for the victims of AIDS.

This edition of Indian Institute of Sexology Bhubaneswar, focuses on different aspects of HIV infection and AIDS.



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HIV Infection and AIDS: Basic Understandings



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Abstract

Acquired immunodeficiency syndrome (AIDS) is a retroviral infection in the humans and other primates. Lot of development in understanding the virus, its life cycle, pathogenesis, opportunistic infections, laboratory investigations for diagnosis and management of AIDS with antiretroviral drugs have taken place over the last few decades. This article attempts to highlight a basic conceptual preview of HIV infection and AIDS.

Introduction

Acquired immunodeficiency syndrome (AIDS) is the single most important modern day pandemic in our times. The first case of AIDS was described in 1981 [1, 2]. In 1983, the causative virus of AIDS was isolated by Luc Montagnier and his colleagues from a West African patient suffering from generalised lymphadenopathy [3]. They named it Lymphadenopathy Associated Virus (LAV). In 1984, it was established as the etiological agent of AIDS by Robert Gallo and his co-workers when it was named Human T cell Lymphotropic Virus III [3]. Thereafter, on various occasions, this virus was isolated from AIDS patients and was

given various names from time to time. In 1986, International Committee on Virus Nomenclature decided the generic name of the virus as Human Immunodeficiency Virus (HIV) [3, 4].

Human Immunodeficiency Virus (HIV), the causative agent of AIDS, belongs to the family Retroviridae and sub-family Lentivirinae [5]. Two genotypes of HIV have been recognised – HIV I and HIV II. HIV II shows around 40% homology with HIV I in the context of surface glycoproteins [2]. It presents more similarity to sooty mangabey simian immunodeficiency virus [2,3]. It is considered less virulent and less transmissible than HIV I. HIV II was earlier confined to western Africa, but presently found world-wide. Presently, in Asia pacific, especially, parts of India with previous ties to Portugal, such as Goa and Maharashtra, HIV II is being frequently detected [6].

By 2014, approximately 80 million people world-wide had become infected with HIV. Out of these, over 45 million people had already died of AIDS [7,8]. According to estimates by the Joint United Nations Program on HIV/AIDS, 35 million people were living with HIV by 2013 [9]. In 2013 alone, 2.1 million people were newly infected, half of whom were young individuals between the ages of 15 and 24 years [9]. A continuous rise has been recorded in the number of people living with HIV infection due to the combined effects of high rates of HIV infection and the beneficial impact of antiretroviral therapy (ART) resulting in declining deaths [10].

Four groups of HIV-I exist and represent three separate transmission events from chimpanzees (M-major, N-non M non O, and O-outlier), and one from gorillas (P-pending the identification of further human cases) [11, 12]. All groups are found in Western Africa suggesting it to be the place of origin of human infection. Group M is the cause

of global HIV pandemic, while N, O, and P are restricted to West Africa [12]. Group M is divided into nine subtypes: A–D, F–H, J, and K. Subtype C is predominant in Africa and India. Subtype B predominates Western Europe, the Americas, and Australia. It has been observed that circulating recombinant subtypes are becoming more common [13]. The most common recombinant subtype is AE. The marked genetic diversity of HIV-1 occurs due to the error-prone function of reverse transcriptase leading to high rate of mutation.

Structure of HIV

HIV is a spherical, enveloped virus having diameter of 90–120nm. Virus has a lipid envelope with several glycoproteins projecting out in the form of spikes. Under the lipid covering lies a matrix protein layer, which supports the transmembrane glycoproteins. Inner to the matrix protein is the capsid, with icosahedral symmetry, surrounding the two copies of positive sense ss-RNA. These two RNAs are linked together at the 5' end. Closely linked with the ss- RNA are various viral enzymes [2].

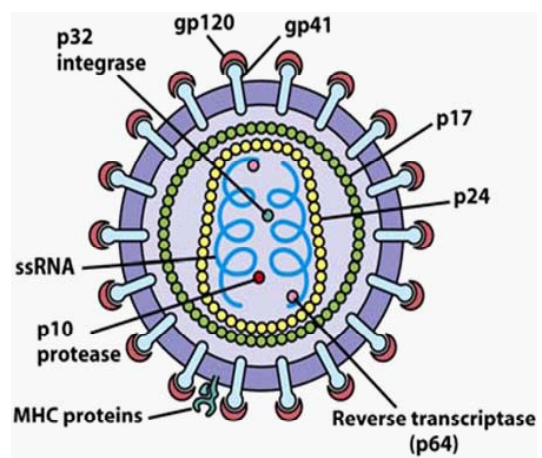


Figure 1. Structure of HIV virus [Source:<http://microbeonline.com/describe-structure-of-hiv-virus/>]

These include reverse transcriptase (RNA-dependent DNA polymerase), integrase and proteases. These enzymes are carried into the host cell by the virus along with itself.

Modes of Transmission

There are various modes of entry of the HIV virus into the body of which sexual route is the most common mode worldwide.

1. Sexual transmission: It accounts for 80% of the spread of HIV. Male-to-female HIV transmission is more efficient than female-to-male transmission. This is because the infected semen tends to stay longer in contact with the female genital tract. If an uninfected male, however, has intercourse with the infected female, the vaginal secretions are in contact only for a brief time period with the penis and urethral orifice.

Heterosexual transmission risk is lower than homosexual transmission [1]. In vaginal intercourse, there is less risk of trauma. In men having sex with men (MSM) due to anal intercourse, the receptive partner has more risk of acquiring the infection. This is due to fragility of rectal mucosa and abundance of Langerhans cells there.

Oral sex is a much less efficient mode of transmission of HIV, as shown by a number of studies [1]. However, it is not completely safe. Sexually Transmitted Diseases (STDs) have an associated risk of increased transmission of HIV. Any infection, whether ulcerative or non-ulcerative, always increases the risk of transmission.

In heterosexual males, circumcision has shown to be protective against transmission of the virus in large randomized control trials [1, 14]. Uncircumcised males have increased risk of ulcerative STDs. The highly vascularised

foreskin is rich in Langerhans cells along with other CD4 positive cells like macrophages which the virus can attach to.

Risk of transmission is greatly dependent on the viral load in plasma. Antiretroviral therapy dramatically reduces the viral load in most HIV-infected individuals, thus decreasing the transmission [1].

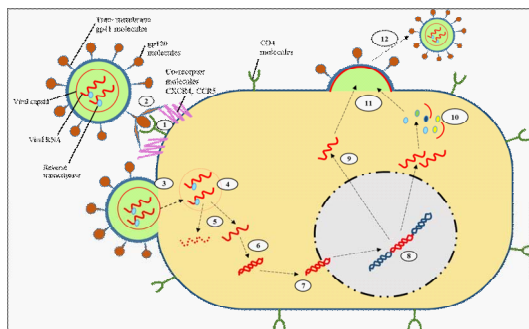
2. Blood and blood products: Blood has the maximum concentration of the virus among all body fluids. Whole blood, packed RBCs, platelets, leukocytes, plasma – all can transmit the virus. Transfusion associated transmission of HIV has decreased after initiation of mandatory HIV I testing for blood and blood donors. Frequent needle sharing puts Injectable Drug Users (IDUs) at increased risk [1].
3. Occupational transmission: Risk of HIV transmission after puncture with needle or sharp instrument contaminated with blood of a documented HIV infection is 0.3% whereas after mucous membrane exposure it is 0.09% and even less in non-intact skin exposure. Exposure to large quantity of blood, increased duration of exposure, inoculation into a blood vessel by a hollow bore needle, high viremia in patient, advance or acute stage of infection in patient are the factors that definitely increases risk [1].
4. Vertical transmission: Transmission from an infected mother to her child can occur during pregnancy (23–30%), during delivery (50–65%) or during breastfeeding (12–20%). High maternal viremia and low CD4 count increase the chances of transmission. Caesarean section delivery along with antenatal and perinatal ART can decrease the transmission risk dramatically. Exclusive breastfeeding

should be preferred over mixed type of feeding, especially, in developing countries where breast milk is the only source of adequate nutrition and immunity builder for the infant [1].

5. Transmission by other body fluids: Saliva has antiviral properties and has protective role in uninfected infants of infected mothers who breast feed their babies. Human bite can transmit HIV, but is rare. In sero-discordant couples where the infected male and uninfected female wish to conceive a child, artificial insemination after sperm-washing (to reduce risk of transmission) has been reported to be successful [1].

Pathogenesis

Infection by a single virion can lead to the disease. CD4+ cells that the virus can target are T helper cells, monocytes, macrophages, microglial cells of CNS, Langerhan's cells (dendritic cells in skin), follicular dendritic cells (in spleen, lymph nodes) and chromaffin cells (in intestines). Recent studies have shown the early locus of infection to be GALT (Gut Associated Lymphoid Tissue)[15].



[Figure 2. Life cycle of HIV : [a] gp120 attaches to CD4 molecule on the susceptible host cell, [b] gp120 undergoes conformational changes, attaches to co-receptors on the host cell membrane, [c] gp41 gets activated and causes fusion of the viral envelope and host cell membrane, [d] viral ss-RNAs of positive sense covered by the capsid enters the host cell

cytoplasm along with reverse transcriptase (RT) and other enzymes, capsid is digested by the host cell proteases, [e] RT transcribes a complementary DNA and degenerates the parent RNA, [f] RT forms ds-DNA, [g] ds-DNA enters the host cell nucleus, [h] integrase enzyme nicks host DNA and ligates viral ds-DNA to host DNA, leading to formation of provirus. This occurs in activated cells. [i] Genomic RNA is formed by the provirus, [j] provirus transcribes m-RNA that translates into viral proteins with the help of proteases, [k] assembly of the viral capsid proteins and genomic RNA and budding from the host cell carrying its lipid envelope, [l] new virus ready to infect another host cell]

HIV Infecting the Host Cell

It is known that gp120 (glycoprotein-120) is the primary docking protein that recognises and attaches itself to the CD4 molecule on the host cell. Once attached to the CD4 molecule the gp120 undergoes conformational changes such that it attaches itself to another co-receptor on the host cell. These co-receptors are chemokine receptors: CXCR4 or CCR5 surface receptor. CXCR4 receptor is specifically located on the surface of T helper cells and CCR5 receptor is present on the surface of macrophages. On initial infection, the viral gp120 can attach only to CCR5 co-receptors, and CXCR4 co-receptors turn susceptible only after the virus has undergone several cycles of replication inside the infected host. Thus, the T helper cells are affected only after some generations of virus have already been produced, i.e., quite late in the course of infection.

After the virus has successfully attached itself with the host cells by CD4 and co-receptor, the transmembrane gp41 (glycoprotein-41) molecule which till this time remained hidden behind gp120 undergoes a conformational change and attaches itself to the cell membrane of the host cell. This leads the viral envelope and the host cell plasma membrane to come closer and fusion of the two occurs.

The nucleo-capsid core of the virus enters the host cell cytoplasm. The proteases present in the host cell cytoplasm digest the capsid protein and cause the release of two copies of viral positive sense ss-RNA strands along with the viral enzymes, i.e., reverse transcriptase, integrase and protease.

Replication of the viral Genome

Each positive sense ss-RNA strand is acted upon by reverse transcriptase (RT) enzyme. Reverse transcriptase has polymerase as well as ribonuclease activity. By virtue of its RNA-dependent DNA polymerase activity, RT synthesises a complementary DNA strand taking the viral positive sense ss-RNA as template. Following the successful transcription of the DNA, the RT by virtue of its ribonuclease activity digests the parent RNA [2, 3, 4, 15].

Taking the newly formed ss-DNA as a template the same enzyme synthesises another complementary strand of DNA, thus now having a double stranded DNA derived from the parent viral genome. This ds-DNA derived from the virus then enters into the host nucleus and is integrated into the host DNA. At this stage, the virus derived DNA is called Provirus. Integrated provirus may remain latent for indefinite time. For the process of integration and provirus formation to be completed efficiently, the host cell should be an actively dividing one.

Assembly and Exit from Host Cell

The genomic RNA and proteins produced by the m-RNA transcribed by the provirus, i.e., the capsid protein, viral enzymes get assembled into virion and then the virion buds from the surface of the host cell. While budding, it takes up the lipid envelop from the host cell membrane. The complete viral particle along with the envelope and surface proteins buds

off from the host cell and is then ready to attack another CD4 positive cell.

Stages of HIV Infection

Acute HIV Infection

From the mucosal surface, the virus is carried by dendritic cells or macrophages to the draining lymphoid tissue where the virus can infect an activated CD4 positive T lymphocyte. Two to four weeks after infection, flu-like symptoms occur including fever, malaise, lymphadenopathy, rash, arthralgia, sore throat, diarrhoea, etc. The plasma virus level reaches as high as 10 million copies per ml. In the beginning of this phase, the p24 antigen can be detected. At the onset, however, the HIV antibodies are not detectable but become detectable during the course of the acute phase. This phase is, hence, known as the 'phase of seroconversion'. The symptoms undergo spontaneous resolution. The number of CD4 + T cells decreases to almost half [1, 2, 4].

'Window period' is the time elapsed between infection and the appearance of antibodies in the blood [2, 4].

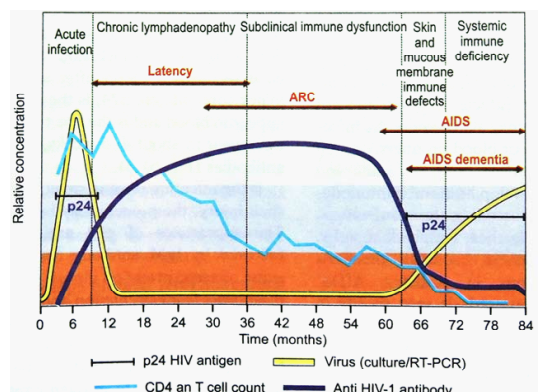


Figure 3. Illustration of usual time course of immune response, viremia and disease in untreated HIV I infection [Source: Ananthanarayan and Panikers, Text book of Microbiology, 9th edition, pg.578]

In the acute phase the virus triggers extensive activation of immune system leading to intense cytokine storm that helps more and more number of activated CD4 cells to be recruited by the virus.

Latent Infection

Variable duration of clinical latency is observed after the acute phase is over. In the absence of ART, after the initial acute phase of infection, the virus level peaks during the 3-4 weeks period, then it gradually drops and reaches a plateau. This occurs due to decreased availability of the activated CD4 positive cells and due to control of the host immune system. There occurs a rebound increase in CD4+ T cells at this stage [14]. Plasma viral load recedes after the acute phase and the viral set point can be the indicator of the disease progression [15].

Rapid progressors

In these group of patients increased viremia, CD4 depletion, and onset of opportunistic infection can occur as early as 6 months, i.e., duration of latent infection is less [15].

Long-term survivors/ Long-term non-progressors (LTNP)

In these group of patients, viral set point is low and they can remain asymptomatic for as long as 25 years with stable CD4 cell count. Active immune response keeps the viral levels in check during the long latent infection [15].

Persistent generalised lymphadenopathy (PGL)

This is another manifestation that may appear towards the end of latent infection. It is defined as persistence enlarged lymph node at 2 or more non-contiguous extra-inguinal site, for at least 3

months, in the absence of any current illness or medication that can cause lymphadenopathy [4]. Ultimately the clinically latent stage and PGL all progress to ARC or AIDS.

AIDS related complex (ARC)

There is considerable immunodeficiency with constitutional symptoms like fever, persistent diarrhoea and unexplained weight loss of more than 10% of body weight. Oral candidiasis, hairy cell leucoplakia, herpes zoster infection, tuberculosis, molluscum contagiosum etc., can occur as opportunistic infection in this stage. CD4+ T cell count falls below 400 per micro liter of blood.

Acquired Immunodeficiency Syndrome (AIDS)

It is the end stage disease. It represents irreversible break down of immune defence mechanism. It is marked by development of more severe opportunistic infections and malignancies. It occurs, when the destruction of peripheral lymphoid tissue is complete and the blood CD4+ T cell count drops below 200 cells/mm³. The CD4+ T cell count then, generally, begins to decline at an accelerated rate.

Opportunistic Infections

Opportunistic infections are those that take advantage of the weakened immune system and cause serious illnesses. According to CDC classification, HIV infected persons are categorised into Category A (Asymptomatic, Acute, PGL) Category B (Symptomatic) and Category C (AIDS indicator conditions) basing on CD4+ T cell counts and clinical conditions associated with HIV infection [1, 16,17]. Each of the above categories are further divided into 3 sub-categories. Sub

-categories A1, B1 and C1 are associated with CD4 + Tcell count >500/mm³. Sub-categories A2, B2 and C2 are associated with CD4 + Tcell count 200-499/mm³. Sub-categories A3, B3 and C3 are associated with CD4 + Tcell count <200/ mm³. Table 1. shows opportunistic infections associated with CD4+ T cell counts.

Table 1. CDC Classification with Associated Opportunistic Infections

CD4+T-lymphocyte counts/microliter of blood	Opportunistic infection associated with lowered CD4 count
>500 cells/mm ³	Hairy cell leucoplakia, candidal vaginitis, Kaposi's sarcoma
200-499 cells/mm ³	Oral thrush, <i>Herpes zoster</i> , <i>Herpes simplex</i>
< 200 cells/mm ³	<i>Pneumocystis jirovecii</i> pneumonia (PCP), Histoplasmosis, Disseminated Mycobacterium avium complex, Cerebral toxoplasmosis, Cryptococcal meningitis, CNS lymphoma, HIV dementia, Cryptosporidiosis. CMV retinitis, Non-Hodgkin's lymphoma, Coccidioidomycosis, Aspergillosis

Immunological Response and Immune Evasion

Both humoral and cellular immune responses develop after the infection. Detectable anti HIV antibodies appear early. Neutralizing antibodies that can prevent the virus to host cell fusion (and thus, decrease the disease progression rate) however, are formed only after 12 weeks of infection. All structural proteins of HIV are strongly immunogenic and induce antibodies. IgM antibodies appear early and remain detectable for a short time; IgG appear late and are long lasting. Diagnostic tests are mainly designed to detect IgG. IgA detection is helpful in new-borns. HIV-specific Cytotoxic T cells (CTL) are the main contributors to decrease the viral set point after acute phase [15].

Immune evasion is the mechanism of the virus to counter the immune response of the host. HIV can undergo mutations rapidly. There is high frequency of recombination during reverse transcription, and the high rate of replication of the virus, together

contribute to enormous genetic diversity. Antibody and CTL recognition sites on the viral envelop are repeatedly changed to protect the viral epitope from the immune system [15].

Virus causes down regulation of surface receptors for CTL in the infected cells (MHC class I not expressed on the infected cell). Thus, the virus can multiply un-noticed by the immune cells [15]. In the latent phase, the virus becomes undetectable by the HIV-specific immune response.

Diagnosis

Screening Tests

ELISA (Enzyme Linked Immunosorbent Assay)

This test is done as a screening test to detect HIV-specific antibodies present in the patient serum. Specific antigens are coated in the well of a microtitre plate. Test sera are added in different wells. Specific antibodies, if present in test serum, bind to the antigen in the microtitre well. Colorimetric detection of these complexes is then done. It takes 2 to 3 hours time. Many

serum samples can be tested at a time. Different systems can be used, i.e., different HIV antigens can be employed. Different modifications of ELISA like capture ELISA, Immunometric assays can be employed to increase sensitivity and specificity. For testing single samples quickly, Rapid test and Simple test are employed.

Rapid Test

These are also used for screening, however, in contrast to ELISA, the results can be obtained sooner, i.e., within 30 minutes. These include Immunochromatographic test, Immunoconcentration test (Dot Blot Assay), etc.

Simple Test

These are tests based on ELISA principle and are easier to perform (These do not need much expertise in contrast to ELISA). Results can be obtained within 1-2 hours. But these tests do not produce results as fast as the Rapid tests.

ELISA is relatively inexpensive and highly sensitive, but has high rate of false positive results. ELISA can detect HIV-I/ HIV-II and variants. Regularly maintained sophisticated equipment, a constant electricity supply and skilled technicians are key requirements for performing ELISA. ELISA is best for testing in laboratories with more sample load as well as in blood banks or for surveillance studies. Rapid tests/Simple tests are more suitable for emergency testing and in smaller laboratories with low numbers of tests per day [18].

Confirmatory/Supplementary Tests

These are performed on serum samples reactive to screening tests. For this purpose, the serum sample has to be tested again by a different

system, i.e. a different HIV antigen or a different principle of test to confirm diagnosis. If the sample is reactive in two different systems, a third test ELISA / Rapid/ Simple test using a system different from previous is performed.

Western Blot

This is the most commonly used supplementary test for validation of positive screening tests. It detects individual antibodies, present in serum, separately against different HIV antigens in the form of bands (formed by antigen-antibody complexes) on nitrocellulose strip.

Viral RNA Detection

This is the 'gold standard' for confirmation of HIV diagnosis. Plasmaviral RNA is typically not detectable until 10 days after infection [19]. It is, however, the method that can detect HIV infection at the earliest amongst all the methods that are employed. It is prognostically valuable for viral load detection. It is also the best tool for diagnosis in the window period. It is also important for typing and drug resistance gene detection [20, 21].

p24Core Antigen Detection

It is less sensitive and becomes positive only after about 17 days post-infection [19]. However, it can be used as a confirmatory test, and can detect HIV infection in the window period.

DNA PCR

Provirus is extracted from peripheral lymphocytes. LTR-gag regions are selectively amplified and studied.

Isolation of Virus

Viral isolation is a less sensitive method. It is also time consuming and expensive.

CD4 Count measurement

This is done by flow cytometry. It acts as a guide for initiation of ART, prediction of opportunistic infections and monitoring the response to drugs.

As PCR and Western Blot are expensive and cannot be employed for all samples reactive to screening tests NACO (National AIDS Control Organisation, India) suggests following strategies for HIV diagnosis in India [22,23].

Strategy 1

It is followed for transfusion and transplantation screening. Test serum is subjected to any one test ELISA/Simple/Rapid tests. If the test result is negative (non-reactive), serum is considered to be free of HIV; and if the test result is positive (reactive), then the sample is considered as HIV infected.

Strategy 2

It is followed for surveillance and in diagnosis too when some AIDS indicator disease is present. If serum is found to be reactive in first ELISA, it is subjected to second ELISA that uses different system from first one. If it is also reactive in second test, it is reported positive; otherwise, it is reported as negative.

Strategy 3

This strategy is followed for diagnosing asymptomatic individuals. Three tests based on different principles are employed. Here the 1st test positive is highly sensitive and 2nd and 3rd tests are more specific. If 1st is positive, 2nd and 3rd tests are done. If both 2nd and 3rd tests are positive, then the case is reported to be positive; if both are negative, then the case is reported to be

negative. If there is any discrepancy in the result of both the tests, then the result is reported as indeterminate and follow up is planned.

Treatment

Highly active antiretroviral therapy (HAART) is using a combination of atleast 3 drugs to suppress maximally possible the virus load as long as possible and stop progression of the disease.

NACO recommends 2 Nucleoside Reverse Transcriptase Inhibitors (NRTI) combined with 1 Non-nucleoside RTI, with Lamivudine in all regimens. The most preferred regimen is Lamivudine + Zidovudine + Nevirapine. In other commonly used regimens, Stavudine can replace Zidovudine and Efavirenz can replace Nevirapine [23].

According to NACO, for clinical stages I and II, ART is to be initiated if CD4+ T-cell count is < 350 cells/mm³. However, for clinical stages III and IV ART should be started irrespective of the CD4 count. Opportunistic infections should be treated prior to initiating HAART [23].

Toxicities associated with the drugs, risk of development of resistance and spread of resistant virus, limited therapeutic options and high cost are the major problems associated with ART. Immune reconstitution inflammatory response is another important concern where due to starting of ART, exaggerated immune response occurs to previously acquired opportunistic infections.

Post-Exposure Prophylaxis

A 28 days course of treatment consisting of 2 NRTI drugs (Basic regimen: Zidovudine + Lamivudine), or 3 (2NRTIs + 1 Protease inhibitor) drugs (Expanded regimen: Basic regimen + Indinavir/Nelfinavir) is recommended by NACO. PEP must

be initiated as soon as possible preferably within 2 hours but not beyond 72 hours. Baseline HIV testing recommended at time of exposure and to be repeated serially at 6 weeks, 3 months, 6 months and 1 year following exposure [4, 23].

Prevention

Prevention of the infection is the corner stone of the entire edifice to deal with AIDS. Safe sexual practices - use of condoms, mutually monogamous (only one sex partner) relationship with a person who is not infected with HIV (be it heterosexual or homosexual) is achievable by educating the community regarding the danger and consequences. Most important recommendations for preventing transmission of HIV infections are - Prompt treatment of STDs, ART for infected persons, ART along with Caesarean delivery in case of infected women who conceive. Mandatory testing of blood and organ donors is also pivotal to prevention of transmission of the disease. Proper disposal of sharp instruments, Personal Protection Equipment use by health workers and laboratory staff, strict standards for aseptic handling techniques for potentially infective material, can help preventing accidental infection in health care set up. Interactive educational communication and sex education can help in prevention in young adults. Similarly, IDUs are another group that should also be targeted [23,24].

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Vaccine

Chemotherapy can never eliminate HIV infection. The development of an effective vaccine can be the ultimate answer for control of AIDS. Unfortunately, even after 35 years of discovery of HIV, no vaccine has yet been developed. For HIV vaccine to be effective, it should elicit strong humoral and cell mediated immunity [25]. Various approaches and trials have been undertaken, but in vain. High mutations of the virus, long latent period, lack of ideal animal models, ethical issues and many more problems have to be overcome.

Conclusion

Forming a basic understanding of the disease is important, so as to help spread awareness and take precautions to prevent HIV infection in any form. ART is presently the answer only for delaying the progression of the disease once infection has occurred, however there is no cure. The increased morbidity and mortality associated with HIV/AIDS is a major problem. There is increased prevalence because increased use of ART has led to increased survival of HIV infected individuals. Till an effective vaccine is developed, research work for the same should be ardently pursued. Awareness for the prevention of transmission, early diagnosis and treatment, and improving the quality of the patients' lives are the crying needs of the hour.

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The screenshot shows the homepage of the Indian Institute of Sexology (IISB) in Bhubaneswar. The header features the IISB logo and the motto "for ecstasy of mankind". A navigation menu includes Home, About IISB, Services Points, Experts, Academics, Get Involved, and Contact Us. The main content area has a blue background with the text "Building on a vivacious tradition" and a Sanskrit quote: "मा निषाद प्रतिष्ठां त्वमगमः शाश्वतीः समाः। शकौषमिथुनादेकम् अवधीः काममोहितम्।". Below this, there are sections for "Centres" (listing Sexual Medicine, Marital Therapy & Counselling, Social Studies, and Communication & Health Education), "Welcome to our Website!" (describing the institute's research focus), and "News & Events" (announcing a mid-year publication on HIV infection/AIDS and Sexuality).

HIV and Stigma



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Abstract

Stigma is mainly associated with diseases which are incurable and are perceived by the society as caused by the violation of societal norms. HIV/AIDS is an example of one such disease. HIV/AIDS related stigma is present at all levels: family, community and health services. This stigma prevents people from getting tests done, from disclosing their seropositivity and taking proper treatment, which in turn, increases the chances of infection and causes various mental health problems. Various programmes have been developed at the national and international levels to address the stigma and provide proper treatment to such individuals.

Introduction

Stigma is defined by Erving Goffman (1963) [1] as a “significantly discrediting” attribute possessed by a person with an “undesired difference”. In recent times stigma came to be defined as a social process that involves recognizing and using “differences” between groups of people to create and legitimize social hierarchies and inequalities (Horizons 2002) [2]. Stigma has been associated with mainly those diseases which are incurable, especially those that society perceives are caused by the violation of social norms like HIV/AIDS [3].

Two types of stigma are mainly spotted, namely, felt stigma and enacted stigma. Felt stigma is defined as anticipation of stigma and discrimination and internal sense of shame while enacted stigma is actual experience of stigma. Felt stigma is not present overtly, it refers to the fear of being treated differently and labeled by others. Enacted stigma is overt and visible, e.g., hesitating in shaking hands with an HIV-infected person or not letting HIV positive individual to work at their workplace. HIV/AIDS-related stigma prevents people from seeking counseling and testing, disclosing about their seropositivity to others, taking adequate medical care as well as complying with medications [4]. The stigma also destroys social lives, leads to depression and other conditions that lowers mental health status, reduces support groups and income due to job loss [4]. HIV infection is often perceived by people, in general, as being associated with immoral acts of an individual which further enhances the complexities of stigma.

Stigma about HIV infection also varies in different sexuality, gender, race, cultures. For instance, HIV infection is related to sexuality as AIDS is mainly a sexually transmitted disease. Thus, it has reinforced pre-existing sexual stigma associated with sexually transmitted diseases.

The associated stigma for HIV positive people might become a source of chronic stressor and might also result in coping with problems, improper self-care, and disturbances in mental health [6]. They face discrimination in workplace, health-care, and housing-related settings [7]. These collectively contribute to stress and adjustment difficulties in persons with HIV infection [8-10]. Thus, understanding the effects of stigma and approaching the treatment from different perspectives is important in dealing with such people.

Stigma and Discrimination Confronted by PLHA

Family & Community

Family and community is the place where people live and spend most of their times. Thus, stigma at such places has a significant effect on people living with HIV/ AIDS (PLHA). In family setup, the form of discrimination reported mostly are separation of utensils, other family members' avoiding sharing food, or not allowing them to cook and denial of use of common areas like toilet, etc. [11]. Other studies have found that PLHA were denied rights in property, care and treatment resources [12-14].

The daughter-in-laws were often not allowed to live in their matrimonial homes, sometimes even when their HIV-positive husbands are alive. The women are even devoid of any rights in the husband's property after his death. It is also found that in some households, HIV positive women are not even allowed to reach to their children [12,14, 17]. Due to stigma and discrimination present in the community women even fear disclosing their HIV-positive status [18-20].

However, studies do report support for PLHA from families. It is reported that in most developing countries, people do get a supportive environment of care, management and treatment of illness. Pradhan et.al reported that the current attitude of the spouse/family was supportive for 58% of the sample in the study [21]. Similar finding was also observed in a study where 70% of the respondents were willing to care for their relatives with HIV/AIDS [22].

It has also been seen that once people disclose their HIV infection status, most of them have received support from their family members

although gender discrimination could be seen in the care provided. Men were more privileged when it comes to be taken care by family than women. The female counterparts including daughters, wives and daughters-in-law experience higher levels of discrimination than men [14]. Thus, in the study gender was observed to be a strong determinant for the type of response one may receive from the family.

Behaviour of families towards the HIV-infected individuals is seen to be affected, to some extent, by the community's attitude and perception towards HIV/AIDS. The family members will treat the PLHA with care, support if they know that the presence of a positive person in family will not result in isolation and banishment from the community.

The most common type of stigma present in the community is that of labelling and shaming [11,12,14]. In a study on Nigerian population, Dahlui M (2015) found that 50% of people agreed with the view that PLHA should be ashamed of themselves, this view was held more by men (60%) as compared to women (50%). In another community based study in Nicaragua approximately 86% participants echoed similar views. Around 54.2% of respondents in the Nicaragua study also believed that even individuals who work with PLHA should feel ashamed about themselves [23].

Other forms of discrimination also exist in community which are more extreme such as barring HIV-positive individuals from social functions, expulsion of children of HIV-positive parents from schools, prohibit social visits to homes, physical isolation, and denial of last rites and burial plot upon death [12,14].

Thus the fear, ignorance and denial associated with HIV/AIDS lead to stigma and discrimination,

which in different ways cast an adverse effect on an HIV-positive individual's daily life and create a hidden epidemic of HIV/AIDS.

There is fear of transmission from infected persons present in the community and visible signs enhance such stigma and discrimination [12]. There is enacted stigma present in the community wherein PLHA are neglected, isolated, verbally abused. The individuals are not allowed to participate in Mahila Mandals, Panchayats and they are also refused house for renting. The marriage of their sibling is affected once people came to know that they are infected with HIV/AIDS. Some other studies also provide evidence for reactions like ostracism, differential treatment at death, and discrimination in schools towards children of PLHA [14,26]. The children of PLHA are not allowed to play with other children or enter anganwadi centres and are also debarred from public amenities.

Gender

The lack of education and patriarchal system puts women in a submissive position. As a result, women have lesser control over their own bodies and lack negotiating skills for their protection [27-29] which is reflected in the manner family and society deals with a HIV-positive woman.

Although discrimination exists for both the genders, women were found to face more discrimination as compared to the men by Bharat et al. (2001)—a finding that is also reported by Greeff et al. (2008) [14, 15]. The study found that Malawian and South African women reported more incidents of stigma than men. Studies observed that family oriented, cultural beliefs of India too contribute in greater acceptance and support for HIV positive men as compared to women [16].

Being HIV positive, women are blamed and named in various ways. Often married women are blamed for bringing the disease to the family. Often due to the existing social hierarchical system, it was observed in several antenatal clinics that women were blamed for bringing the infection into the family, especially, when they are tested HIV positive before their husbands [30].

In majority of cases, men would accuse the women for being unfaithful partners and blame them for bringing the disease [31]. A study on the management of HIV sero-discordant couples in Ibadan, Nigeria, reported that out of 1,000 couples who participated in the study, 30% responded that they would not allow their sons to continue in a marital relationship if their partner is tested positive for HIV infection and sons are not HIV positive [32].

Women are also blamed by the in-laws for the wrong behaviour of their husbands. They are also accused of not being able to control their husbands resulting in such misery to the family. Married women are also denied their rights in their matrimonial homes. They are not allowed to stay in the home and sometimes they are not also allowed access to their children. After the death of their husband, they are forced to leave and are denied any right in the husband's property [12,14]. In a study by Paxton et al.(2005), it was found that approximately 10% of HIV-positive women were forced for abortion. Sometimes women are also forced to have sexual relations with an HIV-positive husband.

Workplace

It has been seen that while HIV infection is not readily transmitted in the majority of workplace settings, still the supposed risk of transmission has

been used by numerous employers to terminate or refuse employment. Thus, people fearing social isolation and loss of job often restrain from disclosing their HIV/AIDS status at their workplace. The major reason for hiding their HIV/AIDS status is fear of job loss. Besides this apprehension, PLHAs also fear facing unfair practices when they are hired at a lesser pay, biased promotion policies, discrimination in work allocation and denial of benefits like loan, insurance or health benefits [26].

These findings reiterate the discrimination faced by HIV-positive individual in various situations. For instance, in a study on HIV/AIDS related stigma and discrimination against PLHA in Nigeria, 40% of the participants responded that teachers who are infected by HIV should not be allowed to teach even if they are not sick [22]. This has also been supported by Oyediran et.al. (2005) who found that about two-third the Nigerian population were in agreement that colleagues who are found to be HIV positive should not be allowed to work further [33]. In another study on unemployment, participants who lost their jobs in past 12 months 50% of them attributed their job loss to their HIV sero-status [34].

Apart from these practices by the employers, the attitude of the co-workers also influences their decision for status disclosure. Many HIV-positive people report of experiencing discrimination in the form of isolation, non-sharing of food by co-workers and non-sharing of the same glass for drinking water etc. More than one-third of the respondents in the study by Porter (1993) refused to dine and work with HIV-positive people [35]. Colleagues avoid sitting close to HIV-positive individuals and sometimes even showed hostile behaviour towards them. Most of the employees

with HIV/AIDS reported in various studies reported discriminatory behaviour from their supervisors and colleagues in the form of social isolation and ridicule [36, 37].

Often co-workers pressurize the employer to terminate the duties of HIV-positive workers. Frequently, due to lack of knowledge regarding HIV infection other people feel anxious because they feel that they will also be highly at risk of being infected by working with a seropositive person.

The fear of image getting tarred, sense of low esteem, being called by names in workplace, often makes PLHAs to apprehend that they would be thought of as people of low character. Hence, they stop going to work, take voluntary retirement or they refrain from declaring their HIV positive status till medical/physical signs start showing up. All these lead to problems in compliance of treatment. Stigma and discrimination often force HIV-positive people to switch jobs frequently. At times, taking up jobs that are less paying, or jobs that are less demanding due to their physical capacities or the working conditions might be such that it would expose them to things that hamper their health and make them ill, as recorded in a case study. The case study published in "India: HIV and AIDS-related Discrimination, Stigmatization and Denial" by S. Bharat (2001) reported the problems faced by the workers in transport department. While working in buses, they often use to fall sick. So they have to request for lighter jobs in place of their current workplace.

Many organization do not have defined policies or guidelines for HIV-positive workers. Thus, they are unable to deal with the discrimination faced by HIV-positive people. Since no special benefits are provided to HIV-positive employees most of them find it difficult to stick to their treatment regime.

This also affected the precautionary measures taken up by them as the special safety measures might make other co-workers curious.

However, very few companies in developing countries seem to have developed policies to deal with fear, stigma and discrimination in the workplace, and some had also defined the responsibilities of employers towards workers with HIV/AIDS [26]. A process has been initiated for facing the HIV/AIDS in organizations by the name of Industrial Response to AIDS (IRTA) still there are very few takers for these steps [38].

Health Services

Health-care facilities have been reported by various studies as the place where HIV-positives experience discrimination the most. [12,14, 39]. HIV-positive individuals feel that presence of special secluded wards or units propagate stigmatization and people who visit such places are subjected to discrimination by others.

Individuals reported discrimination in the the form of denial of admission or treatment services. The government hospitals and private clinics blame each other for negligence of patients on learning about their HIV-positive status. It is often reported by patients that they are offered treatment services at a higher cost and predefined conditions or clauses. Also, if the positive status of an individual is found out during treatment of some other ailment, the patients are then subsequently denied further treatment. Often HIV/AIDS status is not disclosed to the patient instead they are referred to some other medical facility. If, at all, they are provided medical care, they are kept in separate wards, their movement is restricted and sometimes their beds are categorized as "AIDS patient" etc.

HIV-positive patients often experience poor quality of treatment and segregation in hospital wards [40]. It has been seen in two sites (Bangalore and Mumbai) of S Bharat (2001) study that in India pregnant women and people who come for surgery were denied treatment facilities by hospital when their HIV infection test results came out to be positive.

Various studies report the discriminatory practices carried by health care facilities in the form of denying help to HIV-positive pregnant females during delivery, delay in treatment or asking for additional payment for health services [14,17,41]. Kurien et al. (2007) in their study reported similar discrimination faced by PLHA; The study found that 20% of doctors denied treatment to PLHA, 24% isolated HIV-positive people for care from others and 13% doctors changed treatment or postponed it.

Another major issue concerning testing of HIV infection is to take consent for testing and maintain confidentiality of the result. It has been learned during the S. Bharat (2001) study that in most of the cases pre and post-test counselling is not done and in some cases, it was mandatory for people coming in for surgery and for pregnant women to take up HIV/AIDS test. Paxton et al. (2005) found that 52% of positive respondents were told about the HIV test before beginning the test and approximately similar percentage of participants were counselled when they were given their HIV test results. The studies by Mahendra et al (2007) and Pisal (2007) found that health care staff do not consider taking consent of the patient before testing for HIV as important [39, 43]. A National AIDS Research Institute-Yale University (USA) study conducted in Maharashtra, India, observed that most of the

healthcare providers associated patient's HIV infection to immoral conduct of the individual. They also reported presence of fear of touching HIV/AIDS patients. The study also found that healthcare service providers also indulge in malpractices of testing the individual without his or her consent as well as disclosure of his/her HIV sero-status on open charts [44].

Recently, however, Indian courts have started looking into matters of refusal of treatment to HIV-positive patients. In November 2002, the Delhi High Court issued notices to both the Union Government and the Delhi Government seeking their replies on the refusal of several city hospitals to treat an HIV-positive person. Notices were also issued to several hospitals where the person with HIV infection was refused treatment and denied admission.

Discrimination due to HIV infection related stigma is also observed in the way the dead body of an HIV-positive person is treated. In majority of cases, hospital staff refuse to touch the body. It is also reported that often the dead body is wrapped in plastic sheets and even accessing a transport for a dead body is a difficult task for the family members. Respondents of the study conducted by socio-economic impact of HIV/AIDS (2006) by Pradhan et.al reported problems in cremation of the body in form of non-cooperation by the staff at cremation ground or by the community [21].

Stigma Measurement

Scales were developed mostly for research purposes which mostly looked into the attitude of respondents pertaining to areas of negative and hostile feelings towards people with HIV infection,

attribution of HIV-AIDS to vulnerable groups, avoidance intention or social distancing, and support for punitive actions and denial of rights. Responses were obtained on 3 or 4 point scale (No/ Yes/ Maybe/ Don't Know). However, earlier tests lacked standardisation and their reliability and validity scales were not known.

Validity and reliability of measures of HIV/AIDS stigma are important to make sure the effectiveness of prevention and treatment program. Standardized measures of stigma can help trace stigma burden across varied regions and over time [45-47]. Such measures can help trace how stigma is affecting the treatment and testing procedures. Further they can help in development of programmes which aim for stigma reduction as per different cultures and populations [46].

Keeping this in mind, various efforts were made to measure HIV/AIDS stigma with a view to understand stigma in a better way than only as attitude [11, 29, 48, 49]. All the scales were based on prior qualitative research in various setups. One such significant scale was developed by Zelaya et al. (2008). It was a 24-item stigma assessment scale based on a male sample of unknown HIV infection status from the wine shops in Chennai. The scale focused on measuring perceived stigma. The strength of the scale was that it was completely standardised with psychometric properties. Thus, the final scale measured four major domains: "fear of transmission and disease, association with shame and blame, personal support of discriminatory actions towards people living with HIV infection, perceived community support for discriminatory actions or policies towards HIV-infected people, and perceived community support of discriminatory actions or

policies towards HIV-infected people". These domains were assessed using 24 items. Since the scale was based on a low-income group which are comparatively high HIV-risk men, the scale might not apply to the general population.

The scale developed by Mahendra et al. (2007) during a stigma intervention project in three Delhi hospitals, traced the stigma of AIDS among the health workers in hospitals. It aimed to measure fear of contamination and moralistic attitudes with 21 items. Other than the above mentioned areas, it also assessed the dimensions of human rights and health management practices as aspects of discrimination within health settings, thus, proving to be an important tool for assessment of stigma among health practitioners.

To assess four different stigma forms, Steward et al (2008) developed four new and separate tools which assessed enacted, felt, internalised and vicarious stigma each with acceptable reliability 0.92, 0.94, 0.83 & 0.88 respectively. These scales took in to consideration the cultural and context-specific aspects of stigma which could be useful for future research work with HIV-positive individuals.

Enacted stigma could be assessed using the questionnaire developed by Paxton et al. (2005). It measures discrimination with respect to denial of human rights of the HIV-positive individuals as described in the Universal Declaration of Human Rights (right to health, privacy, security, freedom from degrading treatment, marry and found a family, employment, education and right to self-determination and association). The questionnaire thus developed measures discrimination in five areas—health sector, family, community, employment and education. This tool is applicable for measuring discrimination in

Asia at institutional and structural level. However, the drawback of the instrument is that it lacks in psychometric properties and needs testing on subsets of PLHA.

An indirect approach was taken up by Green et al. (2007) to assess stigma in the cultural context of care giving in India. The method used was observation, to record the relationship of the attendants of positive patients with their visitors. The study found that absence of caregivers was an indicator of strained relationships. A major setback of this method was lack of standardization and it was limited in scope as it assessed only the married population.

Another significant test which is applicable to the Indian culture is Implicit Association Test (IAT) developed by NARI, Pune in collaboration with Yale University, USA. It is a self-administered test assessing the thoughts and unconscious feelings of health care providers that are present during treatment of patient with HIV infection [44]. The test traces stigma associated with HIV/AIDS being a sexually transmitted disease and a fatal disease.[44, 50].

Intervention for Stigma and Discrimination Related to HIV/AIDS

Stigma and discrimination are the results of lack of awareness about the illness at the social and individual levels. Thus, various programs and policies are made across various countries to address the issues of acceptance and respect for PLHA at various levels. Policies address the issues both at individual and community level. Most of them target stigma by focusing on providing

comprehensive care, support and treatment to eligible PLHA. They also aim to target stigma and discrimination through greater involvement of PLHA. Policies look into maintaining confidentiality of HIV/AIDS status of the individuals and condemn unauthorised disclosure of their HIV positive status. The policies also take care that HIV infected individuals are not discriminated against due to their seropositive status. Efforts towards raising public awareness are also being made, so that it facilitate acceptance, empathy and respect for PLHAs in the community, at the workplace and at places providing health services and various other services.

Conclusion

Stigma related to HIV/AIDS pose a great threat for the proper implementation of prevention and treatment programs. Existing stigma restrains the individuals from disclosing their status or getting tested for HIV infection which further aggravates the spread of infection. Not taking proper treatment because of the fear of getting noticed and looked down upon is a major concern for health services. Fear of facing loss of job and negative attitude from the family members and society too makes the individual pull himself into exile. In spite of various initiatives taken up by various authorities to mainstream the individuals who have been marginalized, prevalent stigma continues to play the spoil sport. Stigma and discrimination prevalent in the society presents a big challenge for programs associated with prevention and rehabilitation of people living with HIV/AIDS.

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HIV Infection and AIDS: Indian and Global Scenario



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Abstract

Acquired immuno-deficiency syndrome (AIDS) also called "slim disease" has evolved from a mysterious illness to a global pandemic which has infected millions of people across the world. On the verge of the fourth decade of AIDS epidemic, the world has turned the corner; it has halted and begun to reverse the spread of HIV/AIDS infection. Although the prevalence of HIV infection is increasing worldwide as people living with HIV/AIDS (PLHA) are living longer due to effective antiretroviral therapy (ART), due to which new HIV infection and AIDS related deaths were decreased in the last ten years. India ranks 3rd in the world in terms of the number of HIV infected people, although ART and preventive measures are being taken to curb the spread and transmission of HIV/AIDS in high risk groups. Nevertheless, highly sustained economic, social and political motivation is required to reduce the burden of HIV/AIDS in India and worldwide.

Introduction

Acquired immuno-deficiency syndrome (AIDS) is one of the most destructive illness humankind has ever faced. It causes profound social, economic and public health consequences and has become one of the world's most serious health and development challenge. The first case of AIDS was reported in

1981 and since the beginning of the pandemic more than three decades ago, approximately 30 million HIV infected people worldwide have succumbed to AIDS-related illnesses [1].

Global Problems

During early 1980s, AIDS was an emerging illness, and since then it has alarmingly grown in number and evolved into a global pandemic. As per the United Nations Programme on HIV and AIDS (UNAIDS) 2014, the total numbers of people living with AIDS worldwide stands at 36.9 million, of which 34.3 million are adults, 17.4 million are females and 2.6 million are children. During 2014, the total number of newly infected people with HIV was 2 million, out of which 1.8 million were adults while 0.22 million were children. Total deaths from AIDS during 2014 were 1.2 million of which 0.15 million were children [2].

Maximum prevalence of AIDS is reported in the Sub-Saharan Africa and Asia, with both the regions contributing around 85% of total cases worldwide followed by Latin America and Eastern Europe. As per UNAIDS, about 5,600 people got infected with HIV everyday of which about 66% hailed from Sub-Saharan Africa, 600 were children under 15 years of age, 5000 were adults, of whom almost 48% were women and about 30% were young people (15-24 years of age) [2].

Global Trends in Last Decade

The number of HIV infected population has jumped from 31.7 million in 2003 to 35.3 million in 2013, as a result of continuing new infections, people living longer with HIV and population growth. The global prevalence rate which was 0.8 % in 2013 has levelled since 2001. The number

of people newly infected with HIV has declined in the last decade, contributing to the stabilization of the epidemic. The estimated numbers of children acquiring HIV in low- and middle-income countries have also decreased since the year 2000: from 5,36,000 in 2000 to 3,20,000 in 2012. The number of AIDS-related deaths has also declined in the last decade. The number of AIDS-related deaths fell down from a peak of 2.2 million in the mid-2000s to 1.6 million in 2012, due to the more widespread availability of antiretroviral treatment (ART), since its introduction in 1996. By the end of 2009, since the advent of Highly Active Antiretroviral Therapy (HAART) in 1996, it is estimated that HAART has saved an estimated 14.4 million life-years worldwide. In 2012, 62 % of pregnant women living with HIV in low- and middle-income countries received the medicines they needed to prevent transmission of HIV to their babies. In the 22 top priority countries of the Global Plan, to eliminate new HIV infections among children, overall mother-to-child transmission rates have declined from an estimated 26 % in 2009 to 17 % in 2012. In the low- and middle-income countries, the availability and uptake of HIV testing has increased considerably in recent years. Yet, a large proportion of people infected with HIV are still unaware of their HIV status. Approximately 9.7 million HIV cases in low- and middle-income countries were receiving ART, a 50% jump over 2010 [2].

In America, the Caribbean countries and the Western the Europe, cases of new HIV infection has remained relatively stable since 2001 while in Eastern Europe and Central Asia, it has increased since 2008. There has been 25% drop in new cases in Eastern Asia during last decade [2].

The number of new HIV infections globally declined 19% over the past decade. In 15 high

burden countries, HIV prevalence declined more than 25% among young people aged 15-24 years. These declines are largely attributable to the expanded and improved HIV programmes related factors like access to antiretroviral therapy in low- and middle-income countries which increased from only 0.4 million people in 2003 to 5.25 million receiving the therapy by end 2009; significant reductions in the price of first-line antiretroviral medicines enabling low-income countries to provide a year of antiretroviral therapy at a low cost; and as high as 53% of pregnant women living with HIV gaining access to antiretroviral medicines in 2014 to prevent transmission of HIV to their infants, up from 45% in 2008 [2].

Indian Scenario

National adult (aged 15–49 years) HIV prevalence is 0.26% in 2015. The prevalence rate among males stands at 0.30%, while among females it is 0.22%.

Among the States/Union Territories, in 2015, Manipur has shown the highest estimated adult HIV prevalence of 1.15% followed by Mizoram (0.80%), Nagaland (0.78%), Andhra Pradesh & Telangana (0.66%), Karnataka (0.45%), Gujarat (0.42%) and Goa (0.40%). Besides these States, Maharashtra, Chandigarh, Tripura and Tamil Nadu have shown estimated adult HIV prevalence greater than the national prevalence (0.26%), while Odisha, Bihar, Sikkim, Delhi, Rajasthan and West Bengal have shown an estimated adult HIV prevalence in the range of 0.21– 0.25%. All other States/UTs have levels of adult HIV prevalence below 0.20%. The adult HIV prevalence at the national level has continued its steady decline from an estimated peak of 0.38% in 2001-03 through 0.34% in 2007 and 0.28% in 2012 to

0.26% in 2015. Similar consistent declines are noted both in males and females at the national level [3].

The total number of people living with HIV (PLHIV) in India is estimated at 21.17 lakhs in 2015 compared to 22.26 lakhs in 2007.

Children (aged < 15 years) account for 6.54%, while two fifth (40.5%) of total HIV infections are seen among the females. Undivided Andhra Pradesh and Telangana have the highest estimated number of PLHIV (3.95 lakhs) followed by Maharashtra (3.01 lakhs), Karnataka (1.99 lakhs), Gujarat (1.66 lakhs), Bihar (1.51 lakhs) and Uttar Pradesh (1.50 lakhs). These seven States together account for about two thirds (64.4%) of total estimated PLHIV in India. The estimated number of PLHIV in India has been more or less stable during 2013-15 [3].

India is estimated to have around 86,000 new HIV infections in 2015 showing a 66% decline in the number of new infection cases from 2000 and a 32% decline from 2007- the year being set as the baseline in the National AIDS Control Programme (NACP). Children (aged < 15 years) accounted for 12% of total new infections while the remaining cases of new infections were among the adults. Andhra Pradesh & Telangana, Bihar, Gujarat and Uttar Pradesh currently account for 47% of total new infections among adults in 2015. New infections among adults have declined by 50% or more during 2007-2015 [3].

Since 2007, when the number of AIDS-related deaths started to show a declining trend, the annual number of AIDS-related deaths has declined by 54%. In 2015, 67,000 people died of AIDS-related causes nationally. This decline is consistent with the rapid expansion of access to ART in the country. It is estimated that the scale-

up of free ART since 2004 has saved cumulatively around 4.5 lakh lives in India until 2014 [3].

It is estimated that around 35,000 HIV-positive pregnant women needed prevention of parent-to-child transmission services (PPTCT) in 2015. The overall number of pregnant women needing PPTCT has declined in the country from 52,000 in 2007 to 35,000 in 2015 [3].

Nearly, 13.45 lakh PLHIV needed ART in 2015. This includes 12.71 lakh adults and 75,000 children. These results reaffirm, in no uncertain term the country's success story in responding to the HIV/AIDS epidemic. India has successfully achieved the 6th Millennium Development Goal (MDG) of halting and reversing the HIV epidemic. Between 2000 and 2015, there is a reduction of 66% against a global average reduction rate of 35% in new HIV infection cases. By 2007, AIDS-related deaths started to decline, falling by 54% from 2007 to 2015 against a global average decline of 41% during 2005-15 [3].

Trends in Modes of Transmission

As strategized in National AIDS Control Program (NACP) - IV, prevention will continue to be the core strategy as more than 99% of the people are HIV negative. The epidemic continues to be concentrated in subgroups of population that are likely to engage in high-risk behaviour, making them vulnerable to HIV infection. Such groups are referred as high risk groups (HRGs). In India, Female Sex Workers (FSW), Men who have Sex with Men (MSM), Transgender (TG) and Injecting Drug Users (IDU) have been identified as the core HRGs. Further, it has been observed that two other population groups, long distance truckers and

migrant workers play a key role in the spread of HIV infection [4].

Considerable decline in HIV prevalence has been recorded among FSW at the national level (5.06% in 2007 to 2.67% in 2011) and in most of the states where long-standing targeted interventions have focused on behaviour change and increasing condom use. Declines have been achieved among MSW (7.41% in 2007 to 4.43% in 2011) also, though several pockets in the country have shown higher HIV prevalence among them with mixed trends [4].

Prevalence of HIV in pregnant females is 0.35% (2012-13), migrants is 0.99%, truckers is 2.59% and transgender is 8.82% (2011). In some of the North Eastern States, Injecting Drug Use (IDU) has been identified to be the major vulnerability fuelling the epidemic. Stable trends have been recorded among Injecting Drug Users at the national level (7.23% in 2007 to 7.14% in 2011) [4].

Unprotected sex (88.2% heterosexual and 1.5% homosexual) is the major route of HIV transmission followed by transmission from parent to child (5%) and the use of infected blood and blood products (1%). While injecting drug use is the predominant route of transmission in north eastern states, it accounts for 1.7% of HIV infections whereas 2.7% account for unknown causes [5].

The majority of the reported AIDS cases (83%) have occurred in the sexually active and economically productive 15 to 44 year age group followed by 13.5% in the age group of > 45 years and 3.5% in the under-15 year age group.

It is reported that HIV prevalence and incidence is more among the urban population in most parts of the country. However in Maharashtra and Tamil Nadu, HIV prevalence is found to be higher among the rural population [6].

Programmes for Prevention of HIV/AIDS and Unmet Needs

Targeted Interventions (TI) are meant for prevention of HIV infection among high risk groups in defined geographical area. Coverage of TI for high risk groups in India is still not satisfactory. As per National AIDS Control Organization (NACO)'s 2015 estimates, the targeted interventions (TI) coverage of FSW is 80.18% followed by IUD 74.58%, MSM 68.35% and transgender 25.71%. Under the TI projects, the attendance of HRGs has improved in reproductive tract infection (RTI) /sexually transmitted diseases (STD), clinics but their numbers treated is still very low. During 2014-15, among HRGs 8.53% truckers and 7.1% migrants were treated for STI/RTI while treatment of FSW, MSM, TG, and IDU remained quite low, ranging from 1.92% to 2.8%. At Integrated Counselling and Testing Centre (ICTC) during 2014-15, HIV testing was better in MSM (70.96%),FSW (69.93%), IDU (66.81%) and TG (52.92%) as compared to migrants (14.93%) and truckers (8.68%), while HIV positivity remained quite low around 1% or less. Condom distribution was highest among FSW, followed by MSM, while the same was unsatisfactory among TGs and IDUs. In order to reduce HIV transmission among IDUs, under targeted intervention, syringes and needles were distributed to IDUs. During 2014-

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15, 71% of all IDUs could return syringes and 64% could return needles [7].

As of March 2014, 425 ART centres, 870 link ART centres,10 centres of excellence, 37 ART Plus centres , 224 care and support centres and 7 paediatrics centres of excellence were functioning in the country [8].

Conclusion

In the high-risk groups, HIV prevalence has been declining significantly due to targeted interventions. However, India still continues to be in the category of countries with concentrated epidemic. Although HIV prevalence have begun to decline in high HIV burden states but pockets of high HIV transmission still continue. However, new areas of high prevalence are emerging in low prevalence states. Economic losses due to HIV/AIDS are tremendous, putting high burden on the country. Hence, prevention of transmission is only the key factor to reduce the socio-economic loss pertaining to HIV infection / AIDS. Apart from that, discrimination and stigma is quite high which must be curbed in every domain of life of HIV-infected people, creating a positive environment for them, thereby enabling them to gain access to the best possible health care and occupational opportunities.

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Nervous System Involvement in HIV Infection

Abstract

Neurological involvement in HIV positive patients is very common. These conditions are either caused by direct and indirect infection of brain tissue by HIV virus or secondary to immunodeficiency status produced in HIV positive patients. The Central Nervous System (CNS) involvement varies from neoplasm, opportunistic brain infections to brain diseases resulting from exaggerated immune-reaction. Clinically, they present as diffuse non-focal or focal brain diseases. To decrease the morbidity, it is very important that these conditions are suspected and diagnosed early. Multiple infections should be considered while evaluating these patients.

Introduction

Neurological involvement occurs in more than 40% of HIV patients. In up to 20% of AIDS cases, neurological symptoms are presenting features [1, 2, 3, 4]. Although in very recent years, HIV-associated CNS disease is declining, the mortality from these diseases continues to remain high [5]. Nervous system involvement may be caused either directly by HIV itself or indirectly by infectious, neoplastic or autoimmune processes secondary to immunodeficiency status of such patients. CNS diseases caused directly or indirectly due to HIV infection are: 1. HIV-associated neurocognitive disorder



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(HAND syndrome/AIDS Dementia Complex), 2. Vacuolar myelopathy, and 3. Certain peripheral neuropathies [6]. Conditions caused secondary to immunodeficiency status of such patients include 1. CNS lymphoma, 2. Kaposi sarcoma, 3. Progressive multifocal leukoencephalopathy (PML), 4. Fungal infections, 5. Tuberculous meningitis and tuberculoma, 6. Cerebrovascular diseases, 7. Certain neuropathies and myopathies, 8. Toxoplasmosis, 9. Neurocysticercosis, 10. Cytomegalovirus (CMV) encephalitis [7, 8, 9]. In addition, some neurological conditions (i.e. peripheral neuropathy) are caused by antiretroviral drugs. In AIDS, it is difficult to explain a clinical presentation with a single diagnosis. On an ongoing process a new onset neurologic complications are often superimposed with a different etiology. Clinical features reflect the sum of deficits at several anatomic sites.

Non-Focal Brain Diseases in HIV Infection

These conditions present with diffuse alterations in cognition and symmetrical motor dysfunction. In these conditions, patient's symptoms are not readily explained by one or a few macroscopic focal lesions of the brain. There are no specific neurological symptoms to provide discrete cortical localization, nor any signs to point to a lesion in a cerebral or cerebellar hemisphere. Again, these non focal disorders clinically can be further segregated into those in which cognition is altered in the face of preserved alertness and those in which these two elements are altered in parallel. The most important disorder in the first category is AIDS dementia complex (ADC) and the other categories are cryptococcal meningitis, toxic/metabolic encephalopathies, CMV/HSV encephalitis, etc.

Focal Brain Diseases in HIV Infection

Focal brain involvement may be discrete, solitary or multifocal. *Toxoplasma gondii* infection, progressive multifocal leukoencephalopathy (PML) and primary CNS lymphoma are common focal neurological lesions. *M. tuberculosis* and *C. neoformans* are other infectious focal lesions. Infection with less frequent causes include pyogenic abscess and infection with *Nocardia asteroides*, Cytomegalovirus (CMV), *Treponema pallidum*, Varicella-zoster virus (VZV) and *Histoplasma capsulatum*. In a single patient, multicentric lesions may represent more than one disease, and the inability to biopsy all lesions may lead to chances of misdiagnoses. Biopsy-associated complications are more common in these patients.

Important Brain Diseases in HIV Infection

AIDS Dementia Complex (ADC)

In the advanced stages of HIV infection, only a minority of patients develop progressive encephalopathy. This has been termed as AIDS dementia complex (ADC) or HIV dementia. The pathogenesis of the disease is only partly understood, but HIV replication in the CNS plays a key role. Soon after the primary infection, HIV appears to be present and replicating in the CNS of most, if not all, infected individuals. The macrophages in the peri-vascular spaces and multinucleated giant cells (MGCs) are the major sites of virus accumulation [10]. Clinically, this is a sub-cortical dementia characterized by disturbances in motor performance, cognition and behavior. An essential feature in the diagnosis of ADC is the presence of well documented cognitive decline and the exclusion of other neurological complications of HIV infection,

such as cerebral toxoplasmosis, lymphoma, and progressive multifocal leukoencephalopathy [11]. Therefore, cerebrospinal fluid (CSF) examination and imaging studies of the brain are mandatory. CSF analysis should exclude infectious agents other than HIV, and imaging scans should show cortical atrophy, enlarged ventricles, diffusely decreased attenuation of the deep white matter, and an absence of focal abnormalities in patients with ADC. Neuropsychological assessment may also be helpful in confirming the clinical diagnosis of ADC.

The treatment of ADC is highly active antiretroviral therapy (HAART) with drugs that have good CSF penetration. The therapeutic and prophylactic efficacy of zidovudine in ADC has been well documented. Encouraging preliminary results have been seen with lamivudine, abacavir and stavudine. At this point, it is impossible to make definitive recommendations about the optimum antiretroviral therapy for HIV dementia. Good CSF virologic suppression has been reported for regimens consisting of NRTIs plus indinavir, efavirenz, nelfinavir, or nevirapine, but not for ritonavir/saquinavir without NRTIs.

Toxoplasma Encephalitis

Cerebral toxoplasmosis is the most common cerebral mass lesion in patients with AIDS. The sero-prevalence for *Toxoplasma gondii* in HIV infected individuals is estimated to be 10% to 40%. The frequency of symptomatic toxoplasma encephalitis (TE) in seropositive HIV infected patients varies from about one fourth to one half of cases in the absence of antimicrobial prophylaxis [12]. Cerebral toxoplasmosis is due to reactivation of latent infection as a result of progressive loss of cellular immunity. The most frequent clinical manifestations of TE in HIV infected patients are headache, confusion,

fever, and lethargy. Seizures may be an initial manifestation. 50% to 60% of patients complain of or demonstrate focal neurological signs. Occasionally, TE may also present as diffuse encephalitis [13].

Cryptococcal Meningitis

Cryptococcal meningitis (CM) is the most common manifestation of systemic fungal infection in HIV infected patients and is the third most frequent neurological complication in patients with AIDS [14,15]. Patients often present with non-specific complaints such as headache, fever, altered mental status, nausea, and vomiting. Focal neurologic signs and seizures occur in about 10% of patients [14]. Elevated intracranial pressure (ICP) occurs in excess of 50% of HIV infected patients with CM, without accompanying hydrocephalus or cerebral edema [15]. The pathophysiology of increased ICP has not been fully elucidated in this condition.

Primary Central Nervous System Lymphoma

Primary central nervous system lymphoma (PCNSL) is an extranodal, non-Hodgkin's B-cell type neoplasm. In HIV infected patients, the incidence of this neoplasm is more common than in the general population. At present, PCNSL occurs in 2%–5% of patients with AIDS, and is the second most frequent space occupying lesion of the brain after central nervous system toxoplasmosis [16]. These lymphomas typically occur in HIV infected individuals with CD4+ T-lymphocyte counts less than 50 cells/ μ L. In HIV infected patients, the finding of PCNSL is considered an independent criterion for the diagnosis of AIDS. In patients with AIDS, the age of presentation of PCNSL is usually in the fourth decade. Men are more commonly affected than women (ratio 9:1). The majority of these tumors are located supratentorially. Lymphomatous meningitis is estimated to occur

in 25% of patients with AIDS and PCNSL. Single or multiple lesions may be present. The Epstein-Barr virus (EBV) is almost always found in tumor specimens from patients with AIDS-related PCNSL. The clinical presentation of PCNSL in patients with AIDS usually consists of altered mental status, focal neurologic deficits, seizures, or evidence of increased intracranial pressure, with symptoms present for a mean of approximately two months prior to presentation [17]. Patients with HIV infection in whom PCNSL is suspected should promptly be evaluated with CT or MRI of the brain.

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is an often fatal, demyelinating disease of immunocompromised patients caused by the JC virus. Although relatively uncommon at one time, PML is now encountered more frequently, mainly due to the presence of HIV and AIDS. Though survival with PML remains low, the introduction of HAART now offers some hope in improving survival with PML. Because PML is a multifocal disease, a wide variety of neurologic symptoms can occur, and PML should be considered in any HIV infected patient with neurologic symptoms. Common signs and symptoms include mono- or hemiparetic limb weakness, gait abnormality, cognitive dysfunction, and dysarthria. Less frequently, there may be seizures, sensory loss, vertigo, and visual impairment. Fever and headache are usually absent. Without intervention, most patients with PML will die within 4–6 months, though prolonged survival has been reported in a small number of patients, even in the pre- HAART era [18, 19].

Cytomegalovirus Infection

Cytomegalovirus (CMV) infection of the central and peripheral nervous system in patients with

HIV infection and AIDS may result in various clinical syndromes. Encephalitis, polyradiculitis, and polyradiculomyelitis, and peripheral neuropathies can occur due to CMV infection. CMV encephalitis usually occurs in patients with very low CD4+ T-lymphocyte counts (<50 cells/ μ L), and CMV infection is often present at other sites (retina, adrenal glands, gastrointestinal tract, or blood) at the time of presentation. Two distinct clinical and neuropathological entities of CMV encephalitis have been described [20]. The first, encephalitis with dementia, is characterized by subacute dementia with periods of delirium, confusion, apathy, and focal neurologic deficits. Autopsy in these patients reveals diffuse microglial nodules in the grey matter of cortex, basal ganglia, brain stem, and cerebellum. The second form of CMV encephalitis is a ventriculo-encephalitis. CMV infection of the ependymal cells lining the ventricles typically results in a rapidly progressive syndrome of delirium, cranial nerve deficits, and ventriculomegaly. Death due to CMV encephalitis usually results within 4 to 6 weeks of presentation [20, 21].

Tuberculosis

Tubercular involvement may occur at any stage of HIV infection. Multiple infections should always be kept in mind. The presenting signs and symptoms vary according to the stages of the disease. Two patterns of focal involvement have been described. Tuberculomas are small foci of caseation surrounded by a collagenous capsule, inflammatory cells, and few bacilli. The MRI appearance may vary, showing multiple or solitary lesions smaller than 1cm in size, with nodular or ring enhancement, and no associated mass effect or edema. In contrast, tuberculous abscesses are large solitary lesions with central liquefaction containing numerous tubercle bacilli. They appear hyper-intense on T2-

weighted MRI images and are generally associated with mass effect, ring enhancement, and edema. Lesions may be located in the supratentorial area, posterior fossa, or brain stem. Hydrocephalus, basilar meningitis, or cerebral infarction develops in at least one third of the patients diagnosed with CNS tuberculosis [22].

Neurologic Immune Reconstitution Inflammatory Syndrome

Neurologic immune reconstitution inflammatory syndrome (NeuroIRIS) is a newly recognized complication which usually occurs after starting combination antiretroviral therapy. As host immunity improves after starting ART, the body immune system produces an exaggerated immune reaction against underlying subclinical opportunistic infectious agent. In a recent retrospective study of 461 patients started on combination antiretroviral therapy, 7 patients (0.7%) developed Neuro IRIS [23]. In general, the risk of IRIS appears to be high in patients whose CD4+ lymphocyte count is below 50 cells/mL at the start of antiretroviral therapy [24].

Diagnostic Approaches

The basic principle of the diagnostic approach is neuro-anatomic localization. The advantages of this approach is to differentiate disease processes including opportunistic infections which have a tendency to damage particular structures and thus, causing anatomically defined syndromes. Anatomic localization helps for further diagnostic evaluation like electrophysiological testing in diseases of peripheral nervous system and neuroimaging in case of CNS diseases. The time course of the evolution of symptoms and signs are the important diagnostic elements. The temporal

profile reduces the different possibilities. The third important variable in diagnostic approach is the patient's risk background. Here, the stage of systemic HIV infection and the resultant immune suppression are the most important variables. Severe compromise of cell-mediated immunity increases vulnerability to a group of disorders that dominate the course. Bacterial meningitis, tuberculosis, syphilis, etc., can occur at any CD4 count. However, progressive multifocal encephalopathy, cryptococcal meningitis, toxoplasma encephalitis and CMV encephalitis occur in AIDS patients with CD4 count <200/ cubic mm. CNS involvement may be diffuse like HIV encephalopathy, or associated with more discrete solitary or multifocal lesions.

Conclusion

CNS involvement in HIV infection/AIDS is common. To decrease the morbidity, it is very important that these conditions are suspected and diagnosed early. Multiple infections should always be kept in mind while evaluating these patients. Diagnostic approach includes staging of HIV and whether disease is nonfocal or focal. If disease is non-focal, without constitutional symptoms, and major defect is cognitive, then the possible diagnosis is ADC. In the presence of constitutional symptoms, headache and some alteration of sensorium, cryptococcal or tuberculous meningitis should be ruled out. In the presence of focal disease with constitutional symptoms, mass effect and ring enhancement on imaging, toxoplasmosis, tuberculosis and primary CNS lymphoma should be considered. CSF toxoplasma serology and PCR for tuberculosis can be helpful investigations. A typical MRI brain finding with JC virus serology or PCR is suggestive of PML. A diagnostic approach, thus, minimizes both empiricism and the need for brain biopsy.

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Dermatological Manifestations : A Clinical Predictor of AIDS



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Abstract

AIDS (Acquired Immune Deficiency Syndrome) was first described as a distinct clinical entity in 1981. Since then, AIDS, which is caused by HIV (Human Immunodeficiency Virus) infection, has been spreading its tentacles and has come to assume the status of a global pandemic at present. HIV is associated with a wide spectrum of clinical disorders ranging from an asymptomatic infection to AIDS-related complex. The virus produces a panorama of muco-cutaneous manifestations. In resource-poor settings where methods for early detection and management of HIV/AIDS are not readily available, muco-cutaneous disorders may be used as reliable predictors of disease progression and the underlying immune status of the patients, thus, allowing initiation of appropriate antiretroviral therapy and modifying the clinical course of the disease.

Introduction

Ever since its recognition in 1981, HIV/AIDS continues to ravage humanity across all the continents of the world. Within two years of AIDS being defined as a distinctive syndrome, human immunodeficiency virus (HIV) was identified as the causative agent [1]. According to the National AIDS Control Organization (NACO) annual report 2014-15, the total number of people living with HIV/AIDS in India was estimated at

around 20.9 lakh in 2011, 86% of whom were in 15-49 years age-group [2].

Role of Skin Diseases as a Marker of HIV/AIDS

Since the discovery of HIV infection, a number of skin diseases are described to be associated with it. Dermatological manifestation can serve as a window to other systems. Hence, a thorough examination can guide to a diagnosis with severity or stage of affliction. Cutaneous manifestations can be classified into five groups: infectious, auto-immune, drug-induced, HIV-related, and cutaneous malignancies. The prevalence of

muco-cutaneous manifestations varies between 68.8% and 90% [3].

Skin diseases have proved to be sensitive and useful measures through which HIV progression can be monitored. Although skin lesions may be seen in the general population, their occurrence in HIV-infected patients is often atypical and more severe, explosive, extensive or resistant to therapy. The number and severity of dermatoses increase with the advancement of AIDS and at a relatively low CD4 count. However, with the advent of HAART in 1995, there has been a temporal reduction in muco-cutaneous manifestations [4]. The various clinical manifestations are tabulated in Table 1.

Table 1. Cutaneous Manifestation in HIV-Positive Patients

INFECTIOUS DERMATOSES	NON-INFECTIOUS DERMATOSES
<p>1. VIRAL</p> <ul style="list-style-type: none"> • Herpes simplex virus • Herpes zoster virus • Human papilloma virus • Epstein bar virus • Molluscumcontagiosum virus • Cytomegalovirus 	<p>1. PAPULOSQUAMOUS</p> <ul style="list-style-type: none"> • Psoriasis • Seborrheic dermatitis • Reiters disease • Ichthyosiform dermatoses • Xerosis • Pruritus • Eosinophilic folliculitis • Popular pruritic eruption • Eczemas • Papularurticaria
<p>2. BACTERIAL</p> <ul style="list-style-type: none"> • Pyodermas: staphylococcal, streptococcal, pseudomonas. • Mycobacterial infections • Bacillary angiomatosis • Treponema pallidum • Others 	<p>2. PIGMENTARY CHANGES</p> <ul style="list-style-type: none"> • Diffuse pigmentation • Pigmentation of oral cavity

<p>3. FUNGAL</p> <ul style="list-style-type: none"> • Superficial Fungal Infections Candida Dermatophytoses Pityrosporum infections • Deep Fungal Infections Cryptococcus neoformans Histoplasma capsulatum Penicilliummarneffeii Pneumocystis jirovecii Others 	<p>3. NEOPLASMS</p> <ul style="list-style-type: none"> • Kaposi sarcoma • Melanomas • Lymphomas • Squamous cell carcinomas
<p>4. PARASITIC INFESTATIONS</p> <ul style="list-style-type: none"> • Sarcoptesscabiei • Demodicidosis • Toxoplasmosis • Leishmaniasis 	<p>4. HAIR CHANGES</p> <ul style="list-style-type: none"> • Diffuse alopecia • Alopecia areata • Sparseness of hair • Long eye lashes
	<p>5. ORAL CHANGES</p> <ul style="list-style-type: none"> • Oral candidiasis • Oral ulcerations • Oral hairy leucoplakia • Xerostomia • Melanotic hyperpigmentation • Salivary gland enlargement • Linear gingival erythema • Necrotising ulcerative gingivitis • Necrotising ulcerative periodontitis • Acute necrotising ulcerative gingivitis
	<p>6. NAIL CHANGES</p> <ul style="list-style-type: none"> • Onychomycosis • Melanotic band

Spectrum of Skin Conditions

1. Acute seroconversion syndrome

Patients may present with fever, sore throat, cervical adenopathy. A symmetrical

maculopapular erythematous rash is found in 75% of people [1]. Painful oral ulceration, genital ulceration, erythema multiforme and Stevens-Johnson Syndrome may occur [5,6].

2. Bacterial infections

Pyodermas

Staphylococcus aureus (including methicillin-resistant *S. aureus*), *Pseudomonas* species, *Escherichia coli* and *Streptococcus pyogenes* are the commonest isolates in that order; polymicrobial infection may be present in up to 40% of cases [7]. Besides folliculitis, manifestations of staphylococcal infections in HIV include bullous impetigo, ecthyma, cellulitis, abscesses, botromycosis and staphylococcal scalded skin and toxic shock syndromes [1]. Subcutaneous abscesses due to staphylococci may complicate injection or intravenous line sites. Group A *Streptococcus erysipelas* and lymphadenitis has been reported [1]. *Pseudomonas aeruginosa* causes Ecthyma gangrenosum and panniculitis in advanced HIV [8].

Bacillary Angiomatosis

The causative agents are *B. quintana* and *B. henselae*. It is most commonly seen in patients with CD4 <100/ μ L, but cases occurring in the first year following seroconversion with CD4 count > 500/ μ L have been reported [8, 9]. It presents as single or multiple red-purple nodules on the eyelids, mucosae, liver or spleen. Histology shows lobular capillary proliferation. Warthin-Starry stain demonstrates clumps of tangled bacilli [8, 9].

Mycobacterial Infections

HIV-TB co-infection is a serious problem worldwide, but especially of concern in India where background rates of TB are the highest in the world [10]. Prevalence of HIV among patients with radiologic or bacteriologic confirmation of TB in India ranges from 2.8 to 9.4 percent [11].

According to an estimate, around 5.1 million people are infected with HIV and about half of these cases are co-infected with tuberculosis [12].

The clinical presentation is diverse as shown in Table 2 and Figure 1.

Table 2. Clinical Presentation of Cutaneous Tuberculosis

1.	Scrofuloderma
2.	Palmoplantar keratoderma
3.	Acute military tuberculosis of skin
4.	Keratotic papules
5.	Tuberculids
6.	Scattered violaceous papules
7.	Tuberculous lymphadenitis
8.	Atypical mycobacterial infection
9.	Buruli ulcer



Figure 1. Massive lymphadenopathy with suppuration in HIV-positive infant.

Mycobacterium avium intracellular complex (MAC) is one of the most frequent atypical mycobacterial infections in HIV-infected patients. This occurs as part of a disseminated infection

in up to one-third of patients at CD4 T-cell counts below $50 \times 106/L$ (rare below $200 \times 106/L$) [1]. Cutaneous manifestations include violaceous papule, nodule and ulcer. *M. ulcerans* induced aggressive multifocal Buruli ulcer has also been reported [1].

3. Viral infections

Herpes simplex virus

There is a synergistic relation between Herpes Simplex Virus (HSV) and HIV infection. Although ano-genital involvement is frequent, any site can be affected. Atypical presentations include large deep ulcers extending to perineum, buttocks and abdomen, nodular, hyperkeratotic and condylomatous lesions [1]. According to CDC guidelines, any non-healing ulcers of HSV lasting for more than 1 month is an AIDS-defining illness and indicate active HIV [13].

Varicella zoster virus

Varicella may run a prolonged course (>10 days) in HIV-infected children and is frequently associated with complications like pneumonitis, bacterial superinfection and meningitis [3,14]. Von Seidlein et al. documented an association between increasing numbers of episodes of VZV infection and a low CD4 count at the time of primary infection [15].

Herpes zoster occurs in about 6–31.5% patients with HIV-AIDS [16]. Multi-dermatomal lesions are more frequent in advanced HIV disease. Atypical features such as necrotic punched-out ulcers or hyperkeratotic ulcerated nodules have been reported [17]. Systemic complications including fulminant hepatitis and acute meningoencephalitis may occur [3].

Human papilloma virus

Human papilloma virus (HPV), which causes oral, genital and anal warts, has been reported in 29% of buccal mucosal cells and 63% of cervical cells in female sex workers in Kolkata [18].

Both verruca vulgaris and condylomata acuminata are common in HIV disease. There is an increased incidence of facial and intra-oral warts. The extent of disease and the number of HPV types tends to increase as the CD4 count drops. In the anogenital area, condylomata acuminata may form large vegetating masses (Figure 2) or may extend into the anal canal where squamous cell carcinoma may develop. Recent study suggests routine anal cytology to all HIV-infected men especially in those with low CD4 [19].

The most important known determinant of human papilloma virus (HPV) persistence and progression to cancer is viral type, notably the presence of HPV16 [19]. Immune suppression by HIV infection also appears to worsen the outcome of HPV infection. Women infected with HIV are at significantly increased risk for invasive cervical cancer [20].



Figure 2. Condyloma Acuminata in a 25-year old HIV-positive male.

Molluscum contagiosum

The incidence of Molluscum contagiosum (MC) in HIV varies from 5% to 18% in adults and 21% in children [21]. MC should be considered as a first sign of HIV infection, especially extragenital and eruptive MC [22]. Typical lesions are shiny, umbilicated, pearly, dome-shaped papules ranging from 2 to 5 mm [22]. However, atypical lesions including florid, extensive, genital lesions with cellulitis, several papular and nodular lesions lacking the characteristic central umbilication, giant lesions larger than 1 cm, periorbital and intraoral lesions have been reported [1,3]. Disseminated *Penicillium marneffeii* infection, which can be confused with molluscum contagiosum has been reported in Manipur [23].

Cytomegalo virus infection

Reactivation of Cytomegalo virus (CMV) in HIV infection occurs with a CD4 count below $50 \times 10^6/L$. Skin involvement with CMV is relatively uncommon in HIV, but when CMV affects the skin, the mortality can be about 85% in 6 months [1]. Muco-cutaneous manifestation can be variable including painful ulcers of the perineum, thigh, buttocks & oral cavity, purpura, papules, nodules, verrucous plaques, coagulopathies and nodular prurigo [1].

4. Fungal infections

Superficial fungal infections

Candidiasis

Oral candidiasis occurs frequently in individuals with HIV infection; it has been reported as the most common HIV-associated condition, occurring in up to 70% of cases [24]. *Candida albicans* is the most frequent species isolated, however, other species like *C. glabrata*, *C. tropicalis*, *C. krusei* have also

been isolated and these may have decreased susceptibility to fluconazole [25].

Pseudomembranous disease is the commonest oro-pharyngeal manifestation but erythematous (atrophic), chronic hyperplastic, papillary hyperplasia, median rhomboid glossitis may also be seen [1]. *Candida* can also be responsible for paronychia, onychodystrophy, angular cheilitis and intertriginous candidiasis [26]. It may present as a generalized cutaneous eruption of papules and nodules [27].

Dermatophytoses

Tinea corporis, *tinea capitis*, *tinea faciale*, and *onychomycosis* are particularly common. Cases of severe and recurrent *tinea capitis* have been observed. Widespread dermatophytosis, atypical presentation, and unusual forms of nail infection in the form of proximal subungual white onychomycosis (PSWO) have been described in association with HIV infection [28]. Kumarasamy et al. in their study from south India, found 8% of HIV-infected patients to be having dermatophytosis [29]. In severely immuno-suppressed patients with AIDS, lesions have little inflammation and often lack the elevated border and central clearing typical of tinea (Anergic tinea). They are recognized as sharply marginated areas of hyperkeratosis resembling dry skin. Onychomycosis in HIV infection commonly involves the toe nails. Proximal subungual onychomycosis and superficial white onychomycosis are commonly observed in HIV patients [30].

Deep fungal infections

Histoplasma capsulatum, *Cryptococcus neoformans*, *Coccidio desimmitis*, *Aspergillus fumigatus*, *Penicillium marneffeii*, *Sporothrix schenckii*, and

others can cause opportunistic infections in HIV-infected adults.

Extra pulmonary infection with histoplasmosis is an indicator condition in the case definition of AIDS [1]. A wide morphological spectrum of lesions is seen. Macules, crusted/eroded/ulcerated papules and plaques mainly located on the face and chest, as well as oral involvement with erosions and ulcers, is the commonest presentation [1].

Cutaneous involvement can occur in 6–20% of cases of disseminated cryptococcosis [31]. It presents as papulonodular necrotizing skin lesions with central umbilication, like molluscum contagiosum in the context of neurological or pulmonary disease [1].

Penicilliosis is an AIDS-defining diagnosis, caused by dimorphic fungus *Penicillium marneffei*. Patients with CD4 count less than 100/ μ l are at an increased risk [32]. The characteristic skin lesions of disseminated infection are umbilicated papules with or without central necrosis. Nevertheless, penicilliosis can manifest as ulcers, nodules, maculopapules, acneiform lesions or folliculitis [32].

5. Parasitic infestations

Scabies

Scabies occurs frequently in HIV-infected patients. Clinical presentation often depends on the degree of immunosuppression. Atypical clinical features such as involvement of head and neck which is highly unusual in non-HIV-infected adults, may be encountered with progressive immunosuppression. Norwegian/crusted scabies is highly contagious and its diagnosis should arouse suspicion of underlying HIV infection. Lesions may be in the form of classical thick-crusted plaques, psoriasiform plaques or hyperkeratotic yellow

papules resembling Darier's disease [3]. Crusted scabies in HIV infection may be localized to the soles or genitals [1].

Demodex

Demodex folliculorum is a saprophytic mite of human pilosebaceous unit. During HIV infection, demodicosis occurs with CD4 count lower than 200/ μ l. Manifestations range from follicular pityriasis, rosacea like demodicosis, pustular folliculitis, blepharitis and granulomatous rosacea [3].

6. Seborrheic dermatitis

The prevalence of SD in HIV-positive and AIDS patients is 34–83% as opposed to 3% in the general population [33]. In these patients, SD tends to occur early in the course of the disease (CD4+ T-cell count range 450–550 cells/ μ L) and is usually more severe and difficult to diagnose and treat than in the general population [34].

Its incidence and severity are closely related to the stage of HIV infection and inversely correlate with the absolute CD4 and helper T cell counts. More severe, widespread disease, with more erythematous, hyperkeratotic and psoriasiform lesions, has led to the suggestion that the clinical picture seen in HIV infection/AIDS should be termed as SD-like dermatitis of AIDS and should be regarded as a distinctive entity caused by immunological defects [33]. Histopathology shows a deeper lymphocytic infiltrate and a more perivascular neutrophilic (with occasional leukocytoclasia) and plasma cell infiltrate in HIV compared with classic SD [1].

7. Papular pruritic eruptions (PPE)

This is a very common cutaneous manifestation

of HIV-AIDS with Indian reports suggesting an incidence varying between 2% and 35.8% [35, 36]. It is usually a diagnosis of exclusion manifesting as chronic, sterile pruritic papules and pustules on the extensor surfaces of the arms, dorsa of the hands, trunk, and face with sparing of the palms and soles. The condition tends to wax and wane. Sometimes lichenified patches and plaques may be seen. It is associated with eosinophilia and elevated IgE levels. Lesions heal with disfiguring scarring and hyperpigmentation. Interestingly, in a study, 75% of these patients had circulating bullous pemphigoid autoantibodies [37].

It has to be differentiated from prurigonodularis, prebullous pemphigoid, scabies, papulo-necrotic tuberculid, drug eruption, photo-dermatitis, secondary syphilis, oncho-dermatitis and eosinophilic, seborrhoeic, bacterial and acneiform folliculitis [1].

8. Eosinophilic folliculitis

Eosinophilic folliculitis (EF) is a HIV-specific disorder occurring at CD4 T cell counts of 250–300/ μ l [38]. It presents as peri-follicular erythematous papules and pustules, commonly seen over the face and central trunk with sparing of acral sites. The lesions are pruritic and chronic but may display periods of improvement, unlike PPE. Histopathology is an important tool in differentiating PPE from EF and many other conditions that can mimic it. In EF, sterile inflammatory infiltrate consists of perifollicular eosinophils; unlike in PPE, which shows perivascular mononuclear cell infiltrate [38].

9. Psoriasis

The overall incidence of psoriasis is probably not increased in HIV infection, however, its clinical presentation tends to be more severe. The severity varies inversely with the underlying immune status and CD4 counts. A rapid onset of eruptive psoriasis can serve as a clue to underlying HIV infection [1,39]. Palmoplantar, flexural involvement and psoriatic arthritis is common in HIV psoriasis than in the general population of people with psoriasis [1, 39].

10. Pruritus, xerosis and ichthyosis

The incidence of xerosis has been found to be between 22.6% and 100% [3]. Workup of pruritus should include a careful examination of the skin, hair, nails, and mucous membranes to establish a primary dermatologic diagnosis. If no dermatologic cause is found, a systemic cause or medication-related etiology should be sought. Idiopathic HIV pruritus is a diagnosis of exclusion and should only be considered when a specific diagnosis cannot be established. Pruritus and xerosis are also side effects of Anti-retroviral drugs, especially the protease inhibitors [1].

11. Drug reactions

Patients with HIV infection are particularly prone to hypersensitivity drug eruptions.

Table. 3 enumerates various drugs implicated in erythema multiforme, Stevens–Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), hypersensitivity syndrome, and drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome in patients with HIV/AIDS [1].

Table 3: Drugs Implicated In Erythema Multiforme, SJS, TEN, DRESS Syndrome in HIV/AIDS

1.	Abacavir
2.	Allopurinol
3.	Amprenavir
4.	Carbamazepine
5.	Co-trimoxazole
6.	Efavirenz
7.	Fluconazole
8.	Griseofulvin
9.	Indinavir
10.	Nevirapine
11.	Nitrofurantoin
12.	Phenytoin
13.	Probenacid
14.	Pyrimethamine
15.	Saquinavir
16.	Streptomycin
17.	Sulfadiazine
18.	Sulfadoxine
19.	Thioacetazone
20.	Vancomycin
21.	Zidovudine
22.	Traditional Chinese medications

12. Neoplasms

The AIDS-defining malignancies are neoplasms that consistently correlate with the presence of

AIDS in HIV-infected persons. Over the years, Kaposi sarcoma, squamous cell carcinoma of the uterine cervix and high grade non-Hodgkin's lymphomas have been listed as AIDS-defining malignancies. The non-AIDS-defining malignancies appear to occur at a much younger age in HIV-infected persons compared to those who are HIV negative, the neoplasms show atypical features, and a higher grade and stage at the time of diagnosis [39].

Kaposi sarcoma

Kaposi sarcoma is a multifocal, systemic tumor of endothelial origin. It has four clinical variants, enumerated in Table 4 [1,40]. Human herpes virus 8 (HHV8) is thought to be the initiating factor in the pathogenesis of KS [41]. It is transmitted sexually, more by faeco-oral route or the ejaculate than by blood, in HIV positive homosexual men. However, the predominant mode of HIV transmission in India is heterosexual and this might explain relatively low prevalence of KS in India [1,41].

It generally affects skin, mucous membranes, gastrointestinal tract, lymph nodes, and lungs. The oral mucosa is the initial site of localization in 10–20% of all HIV associated KS, frequently involving palate. The classical lesion in HIV is a purple patch, plaque or nodule, which may ulcerate.

Table 4. Clinical variants of Kaposi sarcoma in HIV positive patients

Variant	Risk Group	Median Survival
1. Classical sporadic KS	Elderly men of Eastern European or Mediterranean descent	Years or decades
2. African endemic KS	African children and adults	Months or years
3. Iatrogenic- immunosuppression or transplantation associated	Organ transplant recipients	Months or years
4. AIDS- related KS	Persons infected with HIV	Weeks or months

Lymphomas

The incidence of intermediate and high-grade B-cell non-Hodgkin's lymphomas in HIV-infected individuals is approximately 60 times greater than in the general population. Extranodal sites are usually involved, including bone marrow, Gastro Intestinal tract, and other sites that are unusual in non-HIV-associated non-Hodgkin lymphoma, such as the Central Nervous System (CNS) and body cavities (pleural, pericardial, peritoneal) [42].

Melanoma and non-melanoma skin cancer

AIDS patients have a three to five fold risk of developing a non-melanoma skin cancer. The ratio of squamous cell carcinoma to basal cell carcinoma in HIV is 1:7 as compared to 1.8:1 in renal transplant patients. Melanoma may present atypically and behave more aggressively. Squamous cell carcinoma may present at unusual sites like the nail folds, are multifocal with a high risk of metastasis and recurrence and thus results in a high mortality rate [1].

13. Oral manifestations

Oral manifestations of HIV disease are common and are among the first signs of HIV infection and immunosuppression. Oral lesions are important

not only in early diagnosis but also in monitoring the progress of the disease.

The various oral lesions encountered in HIV include:

- a. Oral candidiasis
- b. Oral ulcerations
- c. Oral hairy leucoplakia
- d. Xerostomia
- e. Melanotic hyperpigmentation
- f. Salivary gland enlargement
- g. Linear gingival erythema
- h. Necrotising ulcerative gingivitis
- i. Necrotising ulcerative periodontitis
- j. Acute necrotising ulcerative gingivitis

Conclusion

Early recognition of the muco-cutaneous manifestations of HIV infection is an important challenge. Careful examination of the skin and mucosa may be highly rewarding in evaluating the stages of HIV disease. An increasing number of dermatoses in an HIV-infected individual point toward progression of HIV, and dermatological evaluation may detect prognostic indicators. By recognizing the spectrum of skin conditions associated with HIV infection and performing appropriate diagnostic tests, treatment can be administered in a timely fashion and outcomes optimized.

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HIV Infection in Injecting Drug Users



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Abstract

Injecting drug users (IDUs) living with Human Immunodeficiency Virus (HIV) have been major vectors of HIV infection transmission. Rates of HIV seropositivity are higher in communities with greater prevalence of IDUs, owing to unsafe injection and sex practices. Despite effective anti-retroviral therapy (ART) and behavioural interventions in reducing the transmission of HIV, the healthcare service utilization has been poor among IDUs. IDU population need to be encompassed in early testing for seropositivity and prompt inclusion in treatment process which includes provision of ART, substance abuse treatment, harm reduction measures, counselling and other behavioural interventions through outreach programmes.

Introduction

HIV infection continued to constitute a major health burden globally. Despite the decline in the number of new HIV infection cases, about 2 million people get infected by HIV around the world annually in the present days. India alone is estimated to have around 21.17 lakhs people living with HIV, as of year 2015; with more than 85 thousand newly emergent HIV infected cases annually [1]. Substance abuse disorders have a well-known association with higher prevalence of

HIV infection in population [2]. Moreover, injecting drug use has been implicated and is a major mode of transmission of HIV infection. IDUs along with men who have sex with men (MSM), transgenders, and female sex workers constitute the higher riskgroup (HRG) for acquiring the infection. Truckers and migrant workers are often considered as the bridge population who, by the virtue of their work condition, are prone to develop substance abuse and subsequent acquisition of HIV infection. Moreover, despite the declining trends of overall HIV prevalence, the estimates have been stable among IDUs.

Globally, the commonly injectable drugs of abuse include opioids, cocaine and methamphetamine etc. [3]. In India, opioids (heroin, buprenorphine, dextropropoxyphene, pentazocine) are the substance of abuse most commonly used through injection by IDUs [4]. These may be abused admixed with benzodiazepines (diazepam, lorazepam) or antihistaminics (chlorpheniramine or promethazine). Over the last few decades, the abuse of opioids by means of injection has escalated from its conventional usage through smoking or chasing. This accelerated the chances of HIV transmission through shared use of injecting equipment.

Epidemiology of HIV among IDUs

The World Drug Report (2014) reported HIV prevalence of 13.1% among IDUs which corresponds to a global figure of 1.7 million (range: 0.9-4.8 million) HIV infected IDUs [5]. There exist great variations worldwide in the prevalence rates of IDUs living with HIV. Estimated prevalence of HIV among IDUs in South-West Asia and Eastern Europe was over 20% [5]. HIV Sentinel Surveillance

(HSS) conducted in India (2010-11) reported HIV infection in 7.14% of IDUs compared to 0.40% from ante-natal clinic attendees (crude representative of general population) [6]. North-eastern states of Manipur and Nagaland reported higher prevalence of IDUs with HIV infection, although the rates are declining with the intervention programmes coming into effect. However, new pockets of higher seroconversion among IDUs have emerged in various states and metropolitan cities in India [1].

Transmission of HIV in IDUs

HIV may get transmitted among IDUs due to shared use of needle, syringes and drug paraphernalia. Transmission risk through parenteral route is higher, as compared to sexual route [7]. Owing to unsafe sex practices, IDUs are often susceptible to acquire infection from individuals of HRGs. Moreover, IDUs with HIV infection may themselves be source of infection transmission through unprotected sex with their female or male sex partners. Unsafe sexual practices have been reported to be higher among IDUs. In comparison to opioids, cocaine and amphetamine abuse is associated with impulsivity, elevated levels of disinhibition and sexual risk taking [7]. Condom use by IDUs is also reportedly low, with higher rates of sexual relationship being established with sex workers. Moreover, the phenomenon of MSM may be higher among the IDUs [8].

Risk Factors and Barriers in Management

Various physical, social, economic, and political factors influence the spread of HIV infection among IDUs. Strathdee et al. (2010) in their review

enlisted an array of risk factors with an attempt to classify them in terms of micro-environmental and macro-environmental [9].

A review by Milloy et al. (2012) emphasized that natural history of HIV infection as well as treatment outcomes in illicit drug users have a strong association with endogenous host and viral characteristics. Lack of legal income among IDUs at baseline was reported as a strong predictor of reduced survival. Early exposure to highly active ART (HAART) and good adherence to treatment have been found to be strongly associated with illness outcomes [10]. Besides, Knowlton et al. (2006, 2007) reported good socio-emotional support, stable housing, and healthy interaction with healthcare providers as predictors of viral load suppression among IDUs on ART [11, 12].

Myriad of factors related to the patients, their social environment, and the care-providers have been identified that might act as barriers to HIV treatment in IDUs. These include stigma and marginalization, incarceration, criminalization, homelessness, economic deprivation, psychiatric and physical co-morbidity, poor ART service facilities and utilization as well as lack of trained healthcare professionals and complicated appointments [13].

Management

Evaluation and assessment

IDUs may not be revealing information about the substance use as well as other high-risk behaviours. Thus, an empathetic approach directed towards rapport building might be helpful in getting relevant information. Moreover, multiple needle marks, scarred blood vessels, abscesses, tattoo marks and bands over venous sites may be useful clues and should be looked for in

patients using substance. A thorough assessment encompasses deriving information regarding the substance use disorder (SUD), high-risk behaviours, sexual activity, other co-morbidities, legal history, premorbid personality/temperament and support systems of the individual. IDUs might require being motivated regarding undergoing investigations for HIV infection as well as initiating and adhering to treatment for SUD and HIV infection as discussed below.

Treatment

Antiretroviral therapy has been used in treatment of HIV infection. The other proposed role of ART has been its use as an infection prevention strategy. ART reduces virus load among infected patients, thus, lowering the risk of infection transmission to others. Combination anti-retroviral treatment (cART) has been shown to lower the incidence of HIV among IDUs [14]. Moreover, Kato et al. (2013) proposed regular testing and early ART for IDUs as the most economical and effective measure to reduce new HIV infections and related deaths in the general population [15]. However, ART utilization among IDUs infected with HIV had consistently been lower as compared to the rest of the HIV infected population, and mortality rates being significantly higher [16]. Behavioural interventions reduce risk behaviour and have been associated with decline in drug use (injection as well as non-injection), increased drug treatment entry, heightened condom use and diminished sex trading. Thus, prompt testing for HIV and treatment initiation along with behavioural interventions, de-addiction strategies and other HIV preventive interventions among IDUs may be helpful in curbing HIV spread [17].

Harm reduction strategies employed among

IDUs unwilling to practice complete abstinence include among others the needle-syringe exchange programs. Sterile needle and syringes are provided to the IDUs through syringe-needle exchange programmes (SNEPs), so that repeated needle use could be prevented with no requirement to share contaminated needles. Since its first application in Amsterdam (1984), SNEPs has been effectively used in various countries worldwide, developed as well as in economically deprived nations. SNEPs have been shown to be efficacious in reducing risky injection behaviour and HIV infection transmission [18]. Similarly, condoms are distributed (free or at nominal price) and its use is encouraged among IDUs while establishing sexual contact with others, so as to prevent transmission of HIV and other sexually transmitted infections (STIs) [18].

Researches have demonstrated treatment of substance abuse in IUDs as a measure to reduce high-risk behaviour and the spread of HIV infection. Opioid substitution therapy (OST) using buprenorphine and methadone maintenance therapy is used with the objectives to treat opioid dependence; and reduce substance abuse, prevent use of needle and syringes and transmission of blood-borne infections [18]. Moreover, OST in IUDs is associated with enhanced adherence to ART.

IUDs are highly prone to contract other sexually transmitted infections (STIs) as well as blood-borne infections like hepatitis B and hepatitis C. Moreover, owing to unhygienic injection practices, chances of developing abscesses remain high in this population. Additionally, IUDs living with HIV/AIDS tend to develop opportunistic infections in due course of illness. These illnesses and comorbidities should be addressed and adequately

managed for the general well-being of the patient [19].

People vulnerable to transmission of HIV from IDUs should be identified and assessed for their serological status. Interventions in this population might help in preventing further spread of infection to other members of the group or family. Family members often endure an enormous physical, mental and economic burden besides encountering social discrimination [20]. Thus, support, care and education should be provided to the patients as well as their families. It further helps in maintaining treatment adherence in IDUs.

Targeted Prevention Strategies in India

National AIDS Control Programme launched by Government of India in 1992, which has entered currently its fourth phase (2012-17), focuses on strengthening and consolidating HIV prevention services in the country. Working objectives of the programme include prevention of new infection and treatment, comprehensive care as well as support to all people suffering from the infection [1]. Interventions are also targeted towards IDUs through an outreach-based service delivery model. Government had introduced needle-syringe exchange programme (NSEP) and opioid substitution therapy (OST) for IDUs apart from general HIV treatment services. Additional services include prevention and management of STIs and abscesses, promotion of condom and safe sex strategies, linkage to health care and rehabilitation agencies, schemes for reduction of stigma as well as building a supportive environment [1]. National AIDS Control Organisation has also developed guidelines specific to female sex partners of

IDUs, and outreach services are also undergoing to address needs specific to this subgroup.

Conclusion

IDUs living with HIV suffer from various health hazards and are a potential source of HIV transmission. Despite various efforts being made to limit the new HIV infections and transmission through IDUs, the outcomes are not entirely satisfactory and successful. Apart from availability and accessibility issues, utilization of healthcare services by IDUs also remains poor. IDUs often

held themselves back due to the stigma and discrimination they face for their drug abuse behaviour. With known effectiveness of ART in reducing virus-load and diminishing HIV transmission, measures should be taken to strengthen the healthcare system and enhance service utilization by IDUs. Healthcare agencies need to sustain and enhance their efforts towards fulfilling the target set by United Nations Programme on HIV/AIDS (UNAIDS) for making accessible HIV combination prevention services to 90% of key populations including IDUs [21].

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HIV Infection in Pediatric Population



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Abstract

Human Immunodeficiency Virus (HIV) infection causes higher mortality and morbidity in children. The important route of acquisition trails to the mother. It casts a bad impact on the health of the mother-child duo. With the active support of organisations like WHO, UNICEF and, more importantly, ongoing research in the field of HIV diagnosis and treatment, there has been lot of visible changes in the field which help to reduce transmission from mother to child, better early infant diagnosis, and effective treatment of the mother and the child. All these have been directed at the possible elimination of mother-to-child transmission of HIV.

Introduction

Human Immunodeficiency Virus (HIV) infection is a deadly infection that is taking a heavy toll on the human population. In a majority of cases, infection in the pediatric age group is the result of mother-to-child transmission. As of 2014, there were 2.6 million children under the 15 years age group out of 37 million persons living with HIV, and 88 percent of the affected children were from Sub-Saharan Africa. In 2014, it as recorded that 2.2 lakh children have got new HIV infection, and approximately 600 children fail victims to new infection

every day [1]. HIV infection has been seen to be on a rise in the developing countries like India. The other routes of infection are due to blood and blood-product transfusion and, rarely, as a result of sexual abuse and adolescent sexual contacts. This article attempts to recognize the patterns of HIV disease, diagnosis, treatment and the challenges in the management of HIV in children.

Disease Manifestations in Children

Most of the patients are recognized because of their mothers' HIV infection status during antenatal period. Sometimes, the children are recognized with manifestations of HIV infection primarily, and then investigations of the mother support the diagnosis. The children present with failure to thrive, hepatosplenomegaly, persistent or recurrent pneumonia, and recurrent diarrhea. They run high risk of dental caries, recurrent aphthous ulcers, parotid enlargement, dermatophytosis, aggressive and resistant bacterial and fungal infections. They also exhibit features of opportunistic infections like pneumocystis jiroveci, cytomegalovirus (CMV) infections, lymphocytic interstitial pneumonitis, and invasive candidiasis. A higher incidence of neoplasia is spotted in children with HIV infection. B-cell lymphoproliferative diseases, including non-Hodgkin lymphoma, Burkitt lymphoma, and smooth muscle tumors, have also been identified. Motor delay, hypotonia, hypertonia, and/or pyramidal tract signs may indicate opportunistic infection of the central nervous system (CNS) or progressive HIV encephalopathy. Cardiac failure, nephropathy due to virus is rare feature of pediatric HIV infection. Indian studies indicate common clinical manifestations including fever

>1 month duration, weight loss, severe protein-energy malnutrition (PEM), skin manifestations, hepatomegaly and tuberculosis [2-12].

Diagnosis of HIV in Children

The disease progression is rapid among babies acquiring infection perinatally and almost 50% death occurs by 2 years. It is very vital to differentiate babies infected with HIV from HIV exposed but uninfected ones. The diagnosis of babies born to HIV positive mothers includes early virological testing (HIV-DNA-PCR) polymerase chain reaction in Integrated counseling and training centre (ICTC) at 6 weeks of age or at the earliest opportunity thereafter [10, 14]. A confirmatory test using whole blood in an ART centre is needed in positive cases, however, ART needs to be started without waiting for results [14]. In case of those who are breast fed, definitive test needs to be done after 6 weeks of stopping breast feeding. If a baby born to an HIV-positive mother presents for first time in 6-18 months of age, a rapid antibody test is done. If rapid test comes negative and the baby has not received breast milk in the last 6 weeks, then HIV-DNA-PCR testing need not be done, however, the definitive diagnosis (rapid antibody test) is to be done after 18 months. If the rapid antibody test is positive, dried blood spot needs to be done for HIV-DNA-PCR. If DNA-PCR test is negative, the guidelines to be followed are similar to earlier one. If the DNA-PCR sample is positive, then the whole blood specimen is sent for DNA-PCR. It is evident that DNA-PCR is to be done after 6 weeks of last exposure (delivery or breast feeding) for confirmation. The US Panel in 2016 has clarified that recommended virologic testing at 1–2 months of age is preferably scheduled

2-4 weeks after stopping the antiretroviral (ARV) prophylaxis. In such situations, the test would be obtained at 6 weeks (in those receiving 4 weeks ARV prophylaxis) or at 2 months (in those with 6 weeks ARV prophylaxis) [14,15]. Every case should have rapid antibody test at 18 months for definite diagnosis. The positive patients should have their CD4 count (absolute and percentage count) done at baseline and every 6 months. The children should be monitored for clinical, immunological, and virological changes periodically.

The Feb 2016 guidelines details the updates on the use of NAT (nucleic acid amplification tests) and point-of care testing for early and easy detection [16].

Prevention of Mother-to-Child Transmission

The updated recommendations support the use of ART on every pregnant mother and during lactation, irrespective of their HIV clinical stage or CD4 counts, and this should be continued lifelong [17].

The breastfed infants should receive Nevirapine once daily from birth till 6 weeks. Those babies who are on replacement feed, shall receive Nevirapine once daily (or Zidovudine once or twice daily) till 4 to 6 weeks [14].

It is recommended that either the baby receives exclusive breast feeding and ARV prophylaxis or no breast feeding at all. In breast fed infants, they can introduce complimentary foods along with breast feeding upto 12 months. The chances of HIV transmission decreases from 35% at baseline (without any intervention and breast feeding) to <5% (with ARV prophylaxis and breast feeding).

Treatment of Pediatric HIV Infection

Initiation of ART

Based on data from the multi-national START and PENPACT1 trials, the Panel now recommends antiretroviral treatment (ART) for all HIV-infected children (includes adolescents), irrespective of clinical symptoms, CD4 T lymphocyte count or viral load [14, 18, 19]. Experts recommend Accelerated ART to reduce the time between diagnosis and initiation of ART. In cases of combined HIV and TB disease, TB treatment should be started first, followed by ART as early as possible within the first 8 weeks of treatment. HIV-positive TB patients with profound immunosuppression (e.g. CD4 counts less than 50 cells/mm³) should be started on ART within the first 2 weeks of TB treatment [13, 14].

What to start?

It is prudent to have good supportive care in the form of balanced nutrition, adequate micronutrients (vitamin A, D, and Zinc), vaccination and growth monitoring [20]. The recommended ART options are as follows:

The first line ART includes Abacavir (ABC) or Zidovudine (AZT) with Lamuvudine (3TC) and Lopinavir/ritonavir (LPV/r) for children less than 3 years of age. Older children should receive Abacavir with Lamuvudine and Efavirenz. The recent updates recommend the use of tenofovir disoproxil fumarate(TDF) for adolescents with Efavirenz (EFV) and Lamuvudine or Emticitabine (FTC). The alternative regimens included Nevirapine instead of Lopinavir or Efavirenz.

What ART to switch to?

Table 1. The following are recommendations for switching of ART drugs[13].

First Line Regimen	Second Line Regimen	Third Line Regimen
2 NRTIs with LPV/r	Less than 3 years: 2 NRTIs with RAL	DTG with 2 NRTIs DRV/r with 2 NRTIs DRV/r with DTG+1–2 NRTIs
	More than 3 years: 2 NRTIs with EFV or RAL	
2 NRTIs with EFV	2 NRTIs with ATV/r or LPV/r	

NRTIs: Nucleoside reverse transcriptase inhibitors, DRV/r =darunavir/ritonavir, RAL=raltegravir, ATV/r=atazanavir/ritonavir.

Prophylaxis and Management of Opportunistic Infections [13]

- 1) Co-trimoxazole prophylaxis is recommended for HIV-exposed infants between 4 and 6 weeks of age and should be continued until HIV infection has been excluded by an age-appropriate HIV test to establish final diagnosis after complete cessation of breastfeeding.
- 2) Co-trimoxazole prophylaxis is recommended for infants, children and adolescents with HIV, regardless of the clinical and immunological conditions.
- 3) In settings where malaria and/or severe bacterial infections are highly prevalent, co-trimoxazole prophylaxis should be continued until adulthood whether or not ART is being taken. In settings of low prevalence for both malaria and bacterial infections, co-trimoxazole prophylaxis may be discontinued for children of 5 years of age and older who are clinically stable and/or virologically suppressed on ART for at least 6 months and CD4 >350 cells/mm³.

ART toxicity

The toxic effects of drugs used in ART are depicted in the Table 2.

Table 2. Side effects of ART Drugs in Pediatric Population

Drugs	Side Effects
Zidovudine	Headache, asthenia, nausea, anemia, neutropenia, lactic acidosis, lipodystrophy. If Hb<8gm%, shift to Abacavir.
Abacavir	Safe, but occasional hypersensitivity reaction (HSR)
Lamivudine	Low side effects
Lopinavir	Nausea, vomiting, hypertriglyceridemia, diarrhea
Tenofovir	
Emtricitabine	Low side effects
Dolutegravir	Insomnia, headache, hepatotoxicity
Nevirapine	Elevated transaminases, hepatotoxicity, Steven-Johnson syndrome (SJS)

Efavirenz	CNS(Insomnia, dizziness, vivid dreams), headaches, elevated transaminases, hepatotoxicity, SJS
Darunavir	Skin rash, nausea, diarrhea, SJS
Raltegravir	Headache, diarrhea, fever, elevated CPK.
Atazanavir	Indirect hyperbilirubinemia, 1st degree AV block, nephrolithiasis, dyslipidemia
Ritonavir	GI intolerance, taste perversion, dyslipidemia, hepatotoxicity, SJS. Coadministration even at low doses with antihistamines, antihypertensives, ergot, hypnotics result in severe adverse effects.

A study from Wadia hospital, Mumbai, reported 43 HIV positive children from the age group of 5 months to 14 years who were started on antiretroviral therapy (ART). 30% reported adverse effects related to the ART. 16% had hepatotoxicity, 12% had raised serum amylase without symptomatic pancreatitis, 12% had zidovudine (AZT) induced anemia, 9% had Nevirapine (NVP) induced rash [21]. The children on ART need frequent monitoring of LFT (liver function tests), complete hemogram to avert the serious side effects and maintaining compliance [19].

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Conclusion

It may be observed that it is super critical in maintaining ART compliance, administering appropriate formulation, decrease dose burden, monitoring by recall, maintaining easy accessibility to drugs, counseling regarding testing, giving and interpretation of test results, psychological counseling of patients, parents/caregivers, and maintaining good level of nutrition, healthy lifestyle, exercise, vaccination appropriately to attain good level of acceptance and coverage. But it is possible now to eliminate mother-to-child transmission with drugs and appropriate practices.

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Our Vision

Harmonious existence between male and female leading the mankind towards ultimate bliss

Our Goals

INDIAN INSTITUTE OF SEXOLOGY BHUBANESWAR (IISB)

- *Aims to facilitate the integration of knowledge and expertise across various disciplines like medicine, psychology, sociology, law and ethics for greater understanding of complexities of human sexuality*
- *Aims to adequately address the individual sexual problems and social issues*

Objectives

- *To bring experts of different disciplines to a common platform for sharing of knowledge and views on human sexuality*
- *To promote research on human sexuality*
- *To impart training on 'Sexology' and strengthen the discipline of 'Sexual Medicine'*
- *To encourage medical professionals to choose 'Sexual Medicine' as a career*
- *To create public awareness on human sexuality and gender issues*
- *To advocate any social change for betterment of mankind*

Mental Health Perspectives of HIV Infection and AIDS



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Abstract

The human immunodeficiency virus (HIV) epidemic continues to be a major public health problem after more than 30 years of the initial discovery of the infection and the delineation of the routes by which it is spread. Psychiatric disorders play a role in the epidemic by increasing risk behavior for infection and decreasing access to treatment. Many individuals are at increased risk because of addictions, personality vulnerabilities, mood disorders, impulse-control disorders, cognitive impairment, social isolation and disenfranchisement, or other barriers to behaviour change. HIV-infected patients with psychiatric illness may experience great difficulty in modifying risk behaviours. Psychiatric disorders too may adversely impact the treatment of HIV infection primarily through undermining treatment adherence. The same psychiatric disorders that prevented patients from reducing their risk prevent them from obtaining benefit from treatment. Untreated patients with high viral loads are more infectious, leading to an increased potential for spread of the HIV epidemic.

Introduction

HIV was originally recognized through a series of cases of young homosexual men with Pneumocystis carinii pneumonia in the early 1980s in California. Current global statistics suggest that nearly 750,000 infants are born each year with HIV infection, while some estimate that 16,000 new infections occur each

day globally, with one individual being infected about every 10 seconds [1]. AIDS-related stigma and mental disorders are the most common conditions seen in people living with HIV (PLHA). AIDS-related stigma and discrimination impede millions of PLHA from accessing and benefiting from effective prevention and treatment services. Mental disorders are also found to be among the most common problems in the life of PLHA, regardless of gender or race/ethnicity, and can impact their health status, healthcare seeking behaviours, and quality of life. Depression, alcohol use disorders, and neurocognitive disorders are the most prevalent mental problems in this vulnerable population [2]. The purpose of this brief review is to prepare readers who may be unfamiliar with HIV-associated neurocognitive disorders (HAND).

Psychiatric Conditions Associated with HIV

Delirium

Delirium is a state of global derangement of cerebral function. It occurs more frequently in medically ill, brain-injured, or metabolically unstable patients. Prevalence of delirium in HIV-infected populations has been reported to be between 43 and 65 percent. Delirium occurs frequently in patients with advanced HIV infection [3]. Hospitalized patients with AIDS also were found to have increased mortality if delirium complicated their hospital course [4]. The clinical presentation of delirium in HIV patients is the same as it is in non-HIV-infected individuals and is characterized by inattention, disorganized thinking or confusion, and fluctuations in level of consciousness. Emotional changes are common and often

unpredictable, and hallucinations and delusions are frequently seen. The syndrome has an acute or a subacute onset and remits fairly rapidly once the underlying etiology is treated. The cause of delirium should be aggressively sought. Particular considerations in HIV patients include hypoxia with *Pneumocystis pneumonia*, malnutrition, CNS infections and neoplasms, systemic infections (e.g., mycobacteria, CMV, bacterial sepsis), HIV nephropathy, substance intoxication and withdrawal, medication toxicity, and polypharmacy. Variations in hydration or electrolyte status may also profoundly affect patients with HIV who already have cerebral compromise. HIV infection itself also may produce an acute encephalopathy similar to that reported with CMV [5]. Older age, multiple medical problems, multiple medications, impaired visual acuity, and previous episodes of delirium, patients with HIV-associated dementia are at an increased risk to develop delirium. The differential diagnosis of delirium includes HIV-associated dementia, especially with AIDS mania, minor cognitive-motor disorder, major depression, bipolar disorder, panic disorder, and schizophrenia. Delirium can usually be differentiated from the above conditions based on its rapid onset, fluctuating level of consciousness, and link to a medical etiology.

Treatment consists of three parts. The first and the foremost is the identification and removal of the underlying cause. The second is the reorientation of the patient by maintaining a normal diurnal variation of light cycles, providing orienting stimuli, such as calendars, clocks, and a view of the outside world, and active engagement and reorientation by staff members, family, and friends. The third, if necessary, is the management of behaviour or psychosis. Low

doses of high-potency antipsychotic agents are usually effective. Newer, atypical antipsychotics are currently being used with some success, but those drugs with more anticholinergic activity may worsen the condition. Benzodiazepines should be used with caution as they may contribute to delirium in some patients but are of particular use in alcohol or benzodiazepine withdrawal delirium. Physical restraint may be necessary, if the patient turns violent, however, it should be used only when alternatives are inadequate, because restraint may worsen delirium. If indicated, typical neuroleptic medications should be used at the lowest dosage and for the briefest duration possible. Atypical antipsychotics are generally preferred because of lower risk for EPS. Terminal delirium in HIV, as in other terminal diseases, is much more refractory to treatment.

HIV-Associated Dementia

Early in the AIDS epidemic, some patients presented with rapidly progressing neurocognitive disturbances, leading to an intensive search for etiology. CNS opportunistic conditions and CNS lymphoma were identified, however, a subset of patients remained for which no identifiable pathogen could be found, and it was deduced that HIV itself was the causative factor behind the dementia. Autopsy studies of demented AIDS patients revealed characteristic white matter changes and demyelination, microglial nodules, multinucleated giant cells, and perivascular infiltrates but a marked absence of HIV within neurons. Basal ganglia and nigrostriatal structures are affected early in the dementia process, with diffuse neuronal losses following. Typical late findings indicate an approximate 40% reduction in frontal and temporal neurons. Analyses of CSF

and autopsy material also have shown aberrant production of specific cytokines in patients with HIV-associated dementia [6, 7]. The cumulative prevalence of HIV dementia in the lifetime of an infected adult has been reported to be near 15 percent, although the incidence has decreased by about 50 percent since the introduction of HAART. Its frequency among patients with otherwise asymptomatic HIV infection or with CD4 cell count greater than 500 cells/mm³ is probably less than 5% in a community sample [8].

Clinically, dementia presents with the typical triad of symptoms seen in other subcortical dementias—memory and psychomotor speed impairments, depressive symptoms, and movement disorders. Initially, patients may notice problems with reading, comprehension, memory, and mathematical skills. But since these symptoms are subtle, they may be overlooked or discounted as fatigue and illness. The Modified HIV Dementia Scale is a very useful bedside screen and can be administered serially to document disease progression. Later, patients were found to develop more global dementia, with marked impairments in naming, language, and praxis. Motor symptoms are also often subtle in the early stages and include occasional stumbling while walking or running; slowing of fine repetitive movements. Impairments on tests of psychomotor speed in patients at time of AIDS diagnosis with no memory complaints have been shown to predict development of HIV-associated dementia up to 2 years later. Parkinsonian features are common in HIV-associated dementia, and clinical correlates between HIV and Parkinsonism too have been identified [9]. Apathy is a common early symptom of HIV-associated dementia. A frank depressive syndrome also commonly develops, typically with irritable mood and anhedonia instead of sadness

and crying spells. Sleep disturbances and weight loss are common. HIV-associated dementia is rapidly progressive, usually ending in death within 2 years. HIV-associated dementia has been suggested as a strong risk factor for suicide [10]. In the Multicentre AIDS Cohort Study [11], the proportion of cases of HIV-associated dementia in patients with CD4 cell counts between 201 and 350 cells/mm³ was higher in 1996–1998 compared with the figures in early 1990s. This suggests that screening for HIV-associated dementia should be extended to patients with CD4 cell counts less than 350 cells/mm³ [11]. The extended survival that antiretroviral regimens have offered patients may also increase their vulnerability to developing dementia rather than dying secondary to other fulminant complications [12].

Initial open-label studies using AZT (Zidovudine, Retrovir) showed promising results, with patients improving on neuropsychological tests [13]. Studies and reports of antiretroviral treatment of HIV-associated dementia relate the clinical improvements of patients, the reversal of confluent MRI signal abnormalities in deep white matter, and a normalization of cerebral metabolites associated with the progression of dementia after 9 months of treatment with HAART. However, controversy exists regarding the duration of treatment and outcome of dementia. Some studies suggest a dose–response relationship between duration of exposure to zidovudine and dementia-related morbidity [14]. Other evidence shows a temporary relation between zidovudine and stability of improvement of cognitive function [15]. The long-term effect of HAART on the course of HIV-associated dementia remains undetermined, with some evidence of ongoing

HIV-related cognitive damage despite more than 3 years of potent antiretroviral treatment [16]. Risperidone and clozapine have been described in case reports of HIV-associated dementia with psychosis, with significant improvement in psychotic symptoms and few EPS [17]. Quality care for patients with HIV-associated dementia is to ensure an optimal HAART regimen and to treat associated symptoms aggressively. Depression can be treated with standard antidepressants, and, in some cases, methylphenidate or other stimulants may be useful in treatment of apathy.

Minor Cognitive–Motor Disorder

HIV-associated dementia is a late-stage disorder, whereas minor cognitive-motor disorder (or mild neurocognitive disorder) is a less severe syndrome observed in earlier HIV infection. Patients with this disorder may present with a singular minor complaint, such as taking longer to read a novel, dysfunction when performing fine motor tasks such as playing the piano, an increased tendency to stumble or trip, or making more mistakes when balancing the check book. Minor cognitive-motor disorder is now regarded as part of the spectrum of HIV-associated dementia, and its description in the literature has fallen out of use. Prevalence data for minor cognitive-motor disorder are variable, often suggesting up to 60% prevalence by late-stage AIDS. Prevalence in earlier stages is not well defined. Whether minor cognitive-motor disorder inevitably leads to HIV-associated dementia is uncertain. It appears that some patients may continue to have minor problems, whereas others will progress to frank dementia. HAART may be of some benefit in slowing down progression, but this conclusion is confounded by

a lack of understanding of factors that lead some patients to progress while others remain static.

Major Depression

Depression is a significant problem in HIV/AIDS. The question of whether the incidence or prevalence of major depression increases in case of HIV-infected patients has been a controversial topic [18]. The estimated prevalence of major depressive disorder (MDD) in HIV-infected patients has been reported to be 19%–43% [19]. High rates of major depression have been found in homosexual men and patients with substance use disorders. Depression has a negative impact on adherence with medical treatments, quality of life, and finally, treatment outcome. Major depression is a risk factor for HIV infection by virtue of its effect on behavior, intensification of substance abuse, exacerbation of self-destructive behaviors, and promotion of poor partner choice in relationships [20]. HIV aggravates the risk of developing major depression through a variety of mechanisms, including direct injury to subcortical areas of brain, chronic stress, worsening social isolation, and intense demoralization. The Multicentre AIDS Cohort showed that rates of depression increased 2.5 fold as CD4 cells declined to fewer than 200/mm³ just before patients developed AIDS, suggesting that lower CD4 cell counts predict increased rates of depression [21]. This suggests that HIV is a causal factor in depression and that depression is a causal factor in HIV transmission and its morbidity, making the patients with these disorders a treatable vector for the HIV epidemic and suggesting an important role for mental health care in HIV treatment and prevention. High prevalence rates of suicide have been reported among HIV-infected patients [22].

Factors associated with HIV and suicide include depression, hopelessness, alcohol abuse, poor social support, low self-esteem, and a history of psychiatric disorder.

The diagnosis of major depression in the HIV clinic is complicated by the high frequency of depressive symptoms that are associated with chronic illness, significant losses and isolation, comorbid neurological illness, comorbid substance use, and the use of many medications that can alter mental function. Patients complaining of depressive symptoms may have their depression overlooked or discounted due to the presence of a plethora of other diagnoses. Nonspecific somatic symptoms are often the result of depression rather than HIV infection in patients who do not have concurrent medical illness. Fatigue has also been found to be associated with depression and not HIV disease progression. Worsening of fatigue and insomnia at 6 month follow-up was highly correlated with worsening depression but not CD4 count, change in CD4 count, or disease progression by CDC category. Depression is most likely to be missed when symptoms are attributed to HIV-associated dementia, fatigue, demoralization and disenfranchisement, wasting syndrome, or substance abuse. Care should be taken in distinguishing between major depression and demoralization (i.e., adjustment disorder) in patients with HIV. Approximately, one-half of the patients presenting to an urban HIV clinic with complaints of depression were found to have demoralization alone [22]. The ability to report feeling fairly normal when distracted from thinking about the precipitating event or circumstance causing distress is a hallmark of demoralization. HIV-related medical conditions and medications can cause depressive symptoms. CNS infections

such as toxoplasmosis, cryptococcal meningitis, lymphoma, and syphilis are associated with high rates of depressive symptoms. Drugs, such as efavirenz, interferon, metoclopramide, clonidine, propranolol, sulfonamides, anabolic steroids, and corticosteroids, have been reported to produce depression. These depressive symptoms often respond to withdrawal of the offending drug.

Treatment with HAART was associated with significant improvements in symptoms of depression, but did not necessarily have a causal relationship [23]. Pharmacotherapy is the mainstay of treatment for major depression. Several studies have demonstrated the efficacy of various antidepressant agents in HIV patients, but no single antidepressant has been found to be superior in treating HIV-infected patients as a group. As is the case with all depressed patients, nonadherence is the most common reason for ineffective drug treatment, and adverse effects are the most common reason for nonadherence. Since HIV-infected patients are likely to be more sensitive to side effects, antidepressants should be started at sub-therapeutic dosage and raised slowly. Psychostimulants also have been evaluated for treatment of fatigue, cognitive impairment, and depression in patients with HIV. An important issue is the interaction of antidepressants and HAART medications. As depression is associated with reductions in adherence to HAART, the risks of untreated depression must be measured against those of potential medication interactions. Psychotherapy is an important and integral part of the treatment of major depression. Treatment with medication plus psychotherapy has been shown to be more effective for patients than either modality alone. Supportive psychotherapy, group therapy and cognitive-behavioural therapy (CBT)

have all shown to be effective in patients of HIV with depression.

Bipolar Disorder

Bipolar disorder is a condition in which patients experience episodic alterations in mood that causes disorder. Manic episodes are associated with increased rates of substance abuse and impulsive behavior, and there has been speculation that bipolar disorder may be a risk factor for HIV infection. To date, there has been no unequivocal evidence to prove that bipolar illness directly increases the risk for HIV infection. Patients with pre-existing bipolar disorder may experience exacerbations because of the stresses of HIV illness. Perhaps the additional presence of CNS inflammation or degeneration secondary to HIV may also help worsen underlying bipolar disorder, and new-onset mania could be a result of the organic insult itself. The prevalence of mania has been found to be increased in patients with AIDS when compared with the general population [24]. Some have suggested that mania should be subdivided into primary and secondary types, with patients who have the secondary type showing close temporal proximity to an organic insult, no history of illness, essentially negative family history, and late age at onset [25]. Secondary mania includes cases those coming up due to HIV brain disease itself [26], those due to antiretroviral drugs [27], and those due to other HIV-related conditions (e.g., cryptococcal meningitis) or medications. Concurrent or subsequent cognitive impairment has been reported among cases of HIV-related mania. The secondary mania associated with HIV was found to be associated with low CD4 cell count [28], often lower than 100 cells/mm³. The incidence of secondary mania,

like that of HIV-associated dementia, appears to have declined after the widespread use of HAART [29]. AIDS mania is usually quite severe in its presentation and malignant in its course. AIDS mania seems to be more chronic than episodic, with infrequent spontaneous remissions, and usually relapses with cessation of treatment.

Treatment of secondary HIV or AIDS mania has not been systematically studied to date, and the optimal treatment remains unclear. Reports often have indicated a particular resistance of manic symptoms to treatment. Others have noted few differences in response in the treatment of secondary HIV mania compared with bipolar disorder [28]. The treatment of mania in early stage HIV infection is not substantially different than the standard treatment of bipolar disorder. It relies on the use of mood-stabilizing medications, particularly, lithium salts and the anticonvulsants valproic acid, lamotrigine, and carbamazepine and antipsychotic agents, now more commonly atypical agents. These medications decrease manic symptoms and may prevent recurrence.

Schizophrenia

Literature on patients with severe and chronic mental illnesses, primarily schizophrenia and bipolar disorder, reports HIV prevalence rates between 2% and 20% in both inpatient and outpatient samples [30]. There is no evidence that HIV infection causes schizophrenia, but there are data to show that schizophrenia contributes to behaviors that may lead to HIV infection. Substance abuse is very common in schizophrenic patients, including during sexual activity [31]. Patients with schizophrenia have significantly less knowledge about HIV

infection and transmission than persons without schizophrenia [32]. Even increased knowledge about HIV in schizophrenic patients may not lead to decreased risk behaviors [33]. Suicidality is found to be at an increased level in patients with both schizophrenia and HIV infection. For all these reasons put together, clinicians should evaluate schizophrenic patients for risk behaviors and for their knowledge about HIV. The principles of treatment for HIV-infected patients with schizophrenia follow the same basic principles as any other patient with schizophrenia, namely, control of symptoms with medications and psychosocial support and rehabilitation.

Substance Abuse and Addiction

Triple diagnosis refers to a patient with a dual diagnosis (substance abuse and psychiatric disorder) who also has HIV, and such patients are overrepresented in HIV treatment. Substance abuse is a primary vector for the spread of HIV in the case of those who use intravenous drugs along with their sexual partners and those who are disinhibited by intoxication or driven by addiction to unsafe sexual practices. Injection drug use is obviously a primary risk factor for contracting HIV by needle sharing. Addiction and high-risk sexual behavior have been linked across a wide range of settings. Alcohol intoxication also can lead to risky sexual behaviors by way of cognitive impairment and disinhibition [34]. Substance abuse may augment HIV replication in the CNS and increase HIV encephalopathy in early AIDS. The medical sequelae of chronic substance abuse accelerate the process of immunocompromise and amplify the burdens of HIV infection. Injection drug users are at higher risk for developing bacterial infections such as pneumonia, sepsis, and endocarditis.

Tuberculosis, sexually transmitted diseases (STDs), viral hepatitis, co-infection with human CD4 cell lymphotropic virus, and lymphomas also occur more commonly in injection drug users with HIV than in other patients with HIV. HIV infected injection drug users are at higher risk for fungal or bacterial infections of the CNS. Alcohol abuse is immunosuppressive and increases risk for bacterial infections, tuberculosis, and dementia. Heroin may worsen HIV-associated nephropathy.

Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) and its symptoms occur at greatly increased rates in HIV-infected patients [35]. PTSD increases the likelihood of engaging in destructive behaviors such as alcohol and other drug abuse, sexual promiscuity, or prostitution. PTSD is of particular concern in HIV treatment and research as it may engender or exacerbate HIV risk behaviors and worsen health outcomes. PTSD from early life trauma may predispose an individual to engage in high-risk sexual or drug behavior. On the other hand, risk behaviors such as prostitution and drug abuse increase exposure to trauma and thus the likelihood of developing PTSD. Finally, HIV infection itself may be the cause of PTSD. Rates of PTSD in response to HIV infection are higher than those in response to other debilitating illnesses [36], including cancer [37]. Persons at risk for HIV and HIV-infected individuals should be routinely screened for PTSD and psychiatric comorbidities, with treatment targeted accordingly.

Personality Issues

The fact that knowledge of HIV and its transmission is insufficient to deter individuals from engaging in HIV risk behaviors suggests

that certain personality characteristics may enhance a person's tendency to engage in such behaviors. Such individuals, who report high rates of sex and/or drug risk behaviors, include HIV-infected drug users, patients presenting at HIV primary care clinics for medical treatment, and HIV-infected men who have sex with other men. This persistence of high-risk behaviors among individuals who are HIV-infected is a disturbing trend in the HIV epidemic. Educational approaches for risk reduction have proved ineffective for individuals with certain personality characteristics [38]. Effective prevention and treatment programs for HIV-infected individuals must consider specific personality factors that render them vulnerable to practicing risky behaviors that further endanger their health as well as the health of others.

Clinical experience makes us believe that unstable extroverts are the most prone to engage in practices that place them at risk for HIV. Patients present with this blend of extroversion and emotional instability. These individuals are preoccupied by, and act on, their feelings, which are labile, leading to unpredictable and inconsistent behavior. Most striking is the inconsistency between thought and action. Regardless of intellectual ability or knowledge of HIV, unstable extroverts can engage in extremely risky behavior. Their primary goal is to achieve immediate pleasure or removal of pain, regardless of circumstances. As part of their emotional instability, they experience intense fluctuations in mood. Unstable extroverts are more likely to pursue sex promiscuously. Unstable extroverts are more vulnerable to alcohol and drug abuse. They are drawn to alcohol and drugs as a quick route to pleasure. They are also more likely to become injection drug users.

Stable extroverts are also present oriented and

pleasure seeking; however, their emotions are not as intense, as easily provoked, or as mercurial. They may be at risk because they are too optimistic or sanguine to believe that they will become infected with HIV. Unstable introverts are anxious, moody, and pessimistic. Typically, these patients seek drugs and/or sex not for pleasure, but for relief or distraction from pain. Stable introverts account for the rest of the patients. These patients, with their controlled, even-tempered personalities, are least likely to engage in risky or hedonistic behaviors. Typically, these individuals are HIV positive as a result of a blood transfusion or an occupational needle stick.

Prevalence rates of personality disorders among HIV-infected patients (19%–36%) and individuals at risk for HIV (15%–20%) are high and significantly exceed rates found in the general population (10%) [39]. The most common personality disorders among HIV-infected patients are anti-social and borderline types [40]. Anti-social personality disorder is the most common and is a risk factor for HIV infection [40].

Adherence is especially challenging in HIV, which carries all of the components of low adherence—long duration of treatment, preventive rather than curative treatment, asymptomatic periods, and frequent and complex medication dosing [41]. Average rates of nonadherence to antiretroviral therapy range from 50 to 70 percent, with adherence rates of <80 percent associated with detectable viremia in a majority of patients. Clinical experience suggests that non-adherence is more common among our extroverted or unstable patients, the same personality characteristics that place them at risk for HIV. Missing doses of HAART can increase the chance of development of HIV resistance. Cognitive-behavioral approach is most effective in treating patients who present

with extroverted and/or emotionally unstable personalities.

HIV-Specific Psychotherapeutic Issues

There are a number of specific circumstances where psychosocial interventions help in HIV-infected patients. These are:

1. Pretest, test, and posttest counseling issues
2. Risk behavior reduction in patients at risk or infected with HIV
3. Issues of partner notification in patients infected with HIV
4. Impaired patients with issues of capacity and competence
5. HAART adherence issues

Studied interventions have included stress management and relaxation techniques, group counseling, education, cognitive training, negotiation skills training, psychotherapy directed at emotional distress reduction, relapse prevention models of high-risk behavior reduction, education directed at eroticizing safer sex, assertiveness training, and peer education in bars.

Adherence Counseling

The single most important factor regarding outcome of HIV treatment is the patient's ability to adhere to a prescribed regimen. The literature on adherence indicates that there are four groups of factors that affect adherence: environmental factors, treatment factors, illness factors, and patient factors. Environmental factors include medication cost, work schedules, transportation, housing issues, and lack of supportive relationships. Missed appointments are a strong predictor of treatment failure, suggesting that any factor that interferes with patients coming for treatment will interfere with adherence [42].

Treatment factors include the type of medication and amount of pill burden. Perceived side effects also correlate with poor adherence and can prevent patients from taking all required doses in an attempt to prevent adverse consequences. Illness chronicity, symptoms, and curability also affect adherence. Lifelong illnesses have the highest degree of nonadherence, as do illnesses that are asymptomatic, because the patient is unable to feel any benefit or effect from taking a medication. Patient factors associated with nonadherence including dementia, depression, psychosis, personality factors, and substance use. The current literature on HIV medication adherence focuses on technical interventions such as pill box and timer reminders, less complex pharmacological interventions, decreased pill burdens, and increased access to care. A growing literature examines psychosocial interventions, relationship with care providers, case management, and psychiatric disorders as barriers to adherence.

Conclusion

The interrelationships between HIV/AIDS and

psychiatric disorders are myriad and complex. Psychiatric disorders can be seen as vectors of HIV transmission, through associated high-risk behaviors. They also complicate the treatment of HIV infection. Use of psychotropic medications with HAART involves drug–drug interactions. Few anti-retroviral drugs like efavirenz and ritonavir can cause psychiatric symptoms and altered blood levels of psychotropic medications. Comorbid psychopathology– including major depression, schizophrenia, addictions, personality vulnerabilities such as unstable extraversion, and the effects of traumatic life experiences are highly prevalent in patients with HIV/AIDS. Each of these problems has the potential to sabotage treatment for HIV infection and its many complications. There is a profound shortage of information and availability of psychiatric care in HIV clinics. Comprehensive care for HIV patients should involve screening for psychiatric conditions and adequate management of the same. By doing so, even the most difficult patients can be successfully treated.

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AIDS : An Ayurvedic Overview



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Abstract

Ancient Ayurvedic texts described a condition called Ojakshaya/ Kshaya, meaning the loss of vital energy which can be equated with modern day AIDS. Ojas constitutes the essence of all the tissue elements and its depletion leads to symptom complex similar to that of AIDS. For prevention of Ojakshaya, comprehensive description like sadvrittapalana (ideal conduct of life with proper sexual conduct), satvavajaya (mental control therapy), acharrasayan (good conduct), abrahmacharya (ideal sexual conduct) have been prescribed. Modern day ayurvedic diagnostic approach to AIDS is independent of the patient's HIV status and does not depend on laboratory tests or imaging. It consists of the patient's history and physical examination from which a practitioner infers the state of the dosha, dhatu and agni in a patient. Traditional herbal use has been reported to be common among individuals with moderate to advanced HIV infection. Practice of Brahmacharyais considered as the most vital preventive aspect for HIV infection in Ayurveda.

Introduction

HIV infection results in AIDS (Acquired Immuno-Deficiency Syndrome) as the virus gradually starts destroying the immune system, and as a result of which, the person becomes more susceptible to opportunistic infections. HIV transmits through

unprotected sexual contact, infected syringes and needles, transfusion of infected blood and from infected mother to her child during child birth & through breast feeding. The signs and symptoms of AIDS are unexplained weight loss lasting for at least one month, diarrhea, intermittent or constant fever and cough lasting for more than one month, enlarged lymph nodes in the neck, armpits and groin. ELISA test is the most common modern test done for diagnosis of AIDS. Management of AIDS in modern medical system includes supportive care, treatment with ART and counseling.

AIDS in Ayurveda

Ayurveda appears to have actually established its identity about 2000 years ago. Although detailed description of AIDS with the line of treatment and preventive measures is not mentioned in Ayurveda, but the diseases having similarity with AIDS in the symptomology and line of treatment is definitely found in the texts of Ayurveda. Ojakshaya meaning the loss of vital energy is generally correlated in the context of AIDS.

Acharya Sushruta has used the term “Vyayama Soshā” while narrating the effects (symptoms) of perverted sex indulgence in Sushruta Samhita. The symptoms, signs and causative factors (nidān), and the treatment aspects have been described in many Ayurvedic treatises like Charak Samhita, Bhavprakash, Susruta Samhita, Astanga Hridaya, Vaidya Chintamani and Chakradatta etc. [1,2].

For the prevention of AIDS, comprehensive description like sadvrittapalana (ideal conduct of life with proper sexual conduct), sattvavajaya (mental control therapy), acharrasayan (good conduct), abrahmacharya (ideal sexual conduct) are described [3].

Importance of Ojas

It is the ojas which keeps all the living beings refreshed. There can be no life without ojas. It marks the beginning of the formation of embryo. The body of the fetus lives on ojas [4, 5]. It sustains the life and is located in the heart. It constitutes the essence of all the tissue elements [4, 5]. The kaphadosha in its natural state promotes strength in the form of ojas [4, 5].

Ojas belongs to the class of somatmakam (cooling) substances. It is cooling, oleaginous, white colored and firm and contributes to the formation and growth of flesh, maintains its integrity or holds it firm and is mobile or capable of moving about from one place to another within the organism. Loss or diminution in its natural quantity of ojas leads to the gradual emaciation and ultimate dissolution of organism [4,6].

Causes of Ojaksaya

When vata and kapha dosas are in the state of diminution, the pitta dosa, while eliminating ojas in the body, causes depression, weakness of senses, thirst, fainting and loss of action [4, 5].

Ojas is deranged by such acts as an abuse of astringent, bitter, cold, parchifying or vishambhi (indigestible food which remains stuffed in the stomach) substances, a voluntary repression of the natural urging for evacuation of the body, by excessive sexual indulgence and fatiguing physical exercise or by draining action of any particular disease [4,6]. Ojas undergoes decrease in quality also by anger, hunger, worry, grief, exertion, etc [4,7].

Signs and Symptoms of Ojaksaya

The symptoms described in Ashtanga Hridayam by Vagbhata, and its supplementary text, the

Ashtanga Sangraham, as well as another Ayurvedic classic, the Charaka Samhita are: a) drastic loss of weight, b) fatigue and lethargy, c) susceptibility to allergies and contagious diseases, d) skin irritations, e) bronchial disorders, often leading to tuberculosis of the lungs, f) damage to intestinal flora resulting in diarrhoea, dysentery, gastritis, wide fluctuations in body temperature. etc. The root causes of the disease, according to the aforementioned Ayurvedic texts, generally, are: a) unhygienic sexual indulgence, such as anal intercourse, b) having indiscriminate intercourse with many people, c) not cleaning the genitals after coitus, d) washing the body with contaminated or dirty water, e) sexual practices with animals (bestiality), and f) transfusion of contaminated blood [8, 9].

The loss of ojas produces different symptoms, such as roughness of the skin, loss of natural glow of the body, aching pain in the limbs, anemia, impaired digestive function and gradual emaciation of the body. Body fat too undergoes a change. There is gradual decay of strength of body which ultimately leads to death [4,6].

With decrease in ojas, the person becomes fretful, frequently worries much without apparent reason and feels discomfort in the sense organs [4,7]. The loss of ojas may be characterized by fits or fainting, bewilderment and distraction of the mind, delirium and the loss of consciousness [4,6].

Diagnosis and Treatment of Ojakshaya

Currently, there is no definite curative treatment for AIDS. The disorders like AIDS can be categorized under anushangi diseases, as explained in Ayurveda [3]. Another important example of

such diseases is prameha (Diabetes mellitus). Active management of these kinds of disorders is prevention only. Rajas (passionate) and tamas (agitated) states of mind are mainly responsible for mode of transmission of HIV. Satvika kind of mind is devoid of defects. Hence, the main focus should be on maintaining the satvika state of mind [10].

The Ayurvedic diagnostic approach is independent of a patient's HIV status and does not depend on laboratory tests or imaging. It consists of the patient's history and physical examination from which a practitioner infers the state of the dosha, dhatu and agni in a patient. Then, along with medicine, the patient is counseled about the "Aahar" (diet). Patients are also told about "Vihar" (behavior, movement) [9].

The medical treatment of the flowing out (external secretion) of ojas from its natural seat (visransa), as well as in the event of it becoming contaminated by the vitiated principles of the body, should consist in improving its quality by elixir and remedies possessed of rejuvenating properties, tending to increase the quantity of such fluid in the body. A patient who has lost all consciousness (owing to an excessive loss or waste of ojas) should be given up by a physician as incurable. The treatment should consist in the administration of oily or emollient drinks, use of medicated unguents or lubrications, pradeha (plasters of oleaginous substances) and pariseka (washes) and a diet comprising light cooling and well-cooked articles of food. Anyone suffering from loss of ojas naturally craves for drink and food that tend to contribute directly to the formation of matter [4,6].

The treatment for loss of ojas is the use of drugs of jivaniyagana (group of restoratives), milk, meat juice etc [4,7].

Drugs of jivaniyagana (group of restoratives) are Jivanti, Kakoli, Meda, Mudgaparni, Masaparni, Rsabhaka, Jivaka and Madhuka [4,7].

Herbal Medicine in the Treatment of AIDS

Traditional herbal use has been reported to be common among individuals with moderate and advanced HIV disease. In Africa, traditional herbal medicines are often used as primary treatment for AIDS. The most common reasons patients gave for using herbs include general wellbeing, relaxation, pain, stress, spiritualism and healing [11].

It was reported that 9% of outpatients believed that it was possible to treat HIV solely with the use of herbs, while others use it to improve energy level, to supplement dietary intake and to enhance response [11].

However, there are some challenges to collaboration between traditional herbal medicine and modern medicine especially in the developing countries mainly due to lack of mutual trust between the two health system, lack of meaningful referral between modern health providers and traditional herbal healers, exclusion of traditional healing methods from the training curricula of doctors and for the fear among traditional healers about losing their treatments secrets to scientists and researchers [11,12]. With many products prepared locally and available in the market, there is a need for patients, providers and policy makers to assess systematically the potential benefits as well as potential harms associated with these herbal medicines for AIDS [13].

Prevention

According to Ayurveda philosophy, prevention of any disease can be successfully done by

prevention of its own causative factors, i.e. nidanparivarjan (avoiding indulgence of causative factors). This nidanparivarjan is not possible without sattvavajaya, i.e., mental control therapy [3].

Thus, the ultimate and unique preventive measure for AIDS is sattvavajaya, i.e., mental control therapy without which prevention is not possible. Prevention of AIDS can be done in two ways: a) Avoidance of causative factors (sattvavajaya) and b) enhancing the immunity (ojasvrudhi) [3].

Hence, the main focus should be on restraining the mind, senses of normal healthy individuals with the means explained in the Ayurvedic classics, namely, Abrahmacharya (proper sexual conduct), Sadvrittapalana (ideal path of good conduct), Rasayan therapy [3].

Here abrahmacharya signifies unwanted sexual practice. Acharyas explains three subpillars (upstambha) for the maintenance of a normal healthy state of body. These are diet (ahara), sleep (nidra) and proper sexual conduct (abrahmacharya) [3].

Acharyas also explained that act of coitus is strictly prohibited or restricted in cases of the women who do not belong or possess the following qualities. As per the rules of the Acharyas, females lying down in any position other than supine should be avoided. Menstrual phase of women (rajasvala) should also be avoided when it comes to act of coitus. Oral, anal and other orifices which are not natural should be avoided for sexual intercourse [3].

Rasayana (vitalizers), has been advised, to be used, as they are believed to be replenishing the vital fluids of the body. Rasayana drugs not only enhance physical immunity but mental immunity also [3].

Brahmacharya–The Vital Preventive aspect of AIDS

Brahmacharya literally means achara or conduct that leads to the realization of Brahman or one's own self. The technical meaning of Brahmacharya is self-restraint, particularly mastery or gaining perfect control over the sexual organ or the freedom from lust in thought, word and deed. It must further involve a permanent abstention from indulgence in erotic imagination and reverie [14]. Practice of Brahmacharya gives good health, inner strength, peace of mind and long life. It invigorates

the mind and the nerves. It helps to conserve physical and mental energy. Hence, practice of Brahmacharyais the most vital preventive aspect for HIV infection/AIDS [3, 15, 16].

Conclusion

Ayurveda, the science of life, teaches us the art of living too. In this regard, dinacharya, ritucharya and sadvrutta play vital roles. Thus, for the prevention and reduction of the incidence of sexually transmitted diseases like AIDS, ideal life style and its benefits should be hammered upon the young generation.

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