INTRODUCTION

Human Immunodeficiency virus (HIV) is the most significant emerging infectious pathogen of the 20th century. Since the Acquired immunodeficiency syndrome was first recognized in 1981 from north America, the HIV/AIDS epidemic continues its expansion across the globe. HIV infection in humans is now pandemic. As of January 2006, the Joint United Nations Programme on HIV/AIDS (UNAIDS) and the World Health Organization (WHO) estimate that AIDS has killed more than 25 million people since it was first recognized, and another 40 million people are estimated to be living with HIV/AIDS. The virus had spread to all continents of the world. Sub-Saharan Africa is most severely affected; other affected regions include south/south East Asia India has an estimated 2.6 million infection making India the country with the 2nd largest population of HIV patients next to South Africa. This condition (HIV) progressively reduces the effectiveness of the immune system and makes individuals susceptible to opportunistic infections and tumours. HIV is transmitted through direct contact of a mucous membrane or the blood stream with a body fluid containing HIV such as blood, semen, vaginal fluid, preseminal fluid and breast milk. People infected with HIV are infectious lifelong. It takes about 7–10 years for the HIV infected person to develop AIDS. Even during this relatively silent asymptomatic period they are infectious. HIV is the etiologic agent of AIDS which belongs to the family of human retroviruses (Retroviridae) and the subfamily of lentiviruses. The four recognized human retroviruses belong to two distinct groups: The Human T lymphotrophic viruses (HTLV I and II, which are transforming retroviruses; and the human immunodeficiency viruses, HIV-1 and HIV-2, which are cytopathic viruses. The most common cause of AIDS throughout the world is HIV-1. HIV-1 comprises several subtypes with different geographic distributions. In 1999, it was demonstrated that HIV-1 infection in humans was zoonotic and had originated from the Pan troglodytes troglodytes species of chimpanzees in whom the virus had co-evolved over centuries.

Morphology of HIV

HIV is a spherical enveloped virus about 90-120 nm in size. The nucleocapsid has an outer icosahedral shell and an inner cone shaped core,
enclosing the ribonucleoproteins. The genome is diploid, composed of two identical single stranded, positive sense RNA copies. In association with viral RNA is the reverse transcriptase enzyme. When the virus infect the cell viral RNA is transcribe by the enzyme, first in to single stranded DNA and then to the double stranded DNA (Provir), which is integrated into host cell chromosome. The pro virus can remain latent for long periods. At times in response to viral promoters, the provirus initiates viral replication by directing synthesis of viral RNA and other components.

During viral replication when the naked virus buds out through the host cell surface membrane, it acquire a lipoprotein envelop, which consist of lipid derived from host cell membrane and glycoprotein which are virus coded. The major virus coded envelop proteins are the projecting knob like spike on the surface and the anchoring transmembrane pedicles. The spikes, gp120 constitute the major surface components of the virus which binds to the CD4 receptors on susceptible host cell. Transmembrane gp41 pedicles cause cell fusion.

**Replication cycle of HIV**

HIV is an RNA virus whose hallmark is the reverse transcription of its genomic RNA to DNA by the enzyme reverse transcriptase. The replication cycle of HIV begins with the high-affinity binding of the gp120 protein via a portion of its V1 region near the N terminus to its receptor on the host cell surface, the CD4 molecule. The CD4 molecule is a 55-kDa protein found predominantly on a subset of T lymphocytes that are responsible for helper or inducer function in the immune system. It is also expressed on the surface of monocytes/macrophages and dendritic/Langerhans cells. In order for HIV-1 to fuse to and enter its target cell, it must also bind to one of a group of co-receptors. The two major co-receptors for HIV-1 are CCR5 and CXCR4. The reverse transcriptase enzyme, which is contained in the infecting virion, then catalyzes the reverse transcription of the genomic RNA into double-stranded DNA. The DNA translocates to the nucleus, where it is integrated randomly into the host cell chromosomes through the action of another virally encoded enzyme, integrase. This provirus may remain transcriptionally inactive (latent), or it may manifest varying levels of gene expression, up to active production of virus.

**HIV genome**

HIV-1 has genes that encode the structural proteins of the virus: gag encodes the proteins that form the core of the virion (including p24 antigen); pol encodes the enzymes responsible for reverse transcription and integration; and env encodes the envelope glycoproteins. HIV-1 contains at least six other genes (tat, rev, nef, vif, vpr, and vpu), which code for proteins involved in the regulation of gene expression. Several of these proteins are felt to play a role in the pathogenesis of HIV disease.

**Clinical manifestations**

The clinical consequences of HIV infection encompass a spectrum ranging from an acute syndrome associated with primary infection to a prolonged asymptomatic state to advanced disease.

**The acute HIV syndrome**

It is estimated that 50 to 70% of individuals with HIV infection experience an acute clinical syndrome approximately 3 to 6 weeks after primary infection. Varying degrees of clinical severity have been reported. The typical clinical findings (Fever, Pharyngitis, Lymphadenopathy, Headache/retro orbital pain, Arthralgias/ myalgias, Lethargy/malaise, Anorexia/weight loss, Nausea/vomiting/diarrhea, Meningitis, Encephalitis, Peripheral neuropathy, Myelopathy) Symptoms usually persist for 1 to several weeks and gradually subside as an immune response to HIV develops and the levels of plasma viremia.

**The asymptomatic stage – Clinical latency**

Although the length of time from initial infection to the development of clinical disease varies greatly, the median time for untreated patients is approximately 10 years. HIV disease with active virus replication is ongoing and progressive during this asymptomatic period.

**Symptomatic disease – AIDS**

This is the end stage disease representing the irreversible breakdown of immune defence mechanisms, leaving the patient prey to progressive opportunistic infections and malignancy

Symptoms of HIV disease can appear at any time during the course of HIV infection. The more severe and life-threatening complications of HIV infection occur in patients with CD4+ T cells counts <200/uL. A diagnosis of AIDS is made in anyone with HIV infection and a CD4+ T cell count <200/uL and in anyone with HIV infection who develops one of the HIV-associated diseases considered to be indicative of a severe defect in cell-mediated immunity.

**MATERIALS AND METHODS**

The study was undertaken in Guru Govind Singh Hospital, Jamnagar from a period October 2002 to September 2003. A standardized form was filled out on each patient documenting sociodemographic information as well as related symptoms and procedures and samples were collected as follows for laboratory examination.

From collected whole blood, serum was separated on the same day. All the sera were tested on the same day and if not possible, they were stored at 2 – 8º C. All sera were screened for Anti-HIV 1/2
antibodies using third generation ELISA (ENZAIDS). Positive samples were confirmed by HIV 1 and 2 Bispot test (IMMUNOCOMB) and rapid visual band test (SD-BIO).

RESULTS

A total of 1786 serum samples were tested for HIV antibodies at Guru Govind Singh Hospital, Jamnagar. The table no 1 shows that out of total 1786 samples for HIV, 297 patients were seropositive (16.63%) and 1489 patients were seronegative (83.37%).

Table 1: Prevalence of HIV

<table>
<thead>
<tr>
<th>Total tested for HIV</th>
<th>HIV positive</th>
<th>HIV negative</th>
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<tbody>
<tr>
<td>1786</td>
<td>297 (16.63%)</td>
<td>1489 (83.37%)</td>
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</table>

The Table 2 shows that in age group of 1-10 years 14(9.92%) out of 141 were HIV seropositive. In 11-20 years age group 6(2.57%) out of 239 were seropositive. In 21-30 years age group 138(17.94%) out of 769 were seropositive. In age group of 31-40 years 102(24.52%) out of 416 were seropositive. In 41-50 years age group 31(20%) out of 155 were seropositive. In age group of 51 years and above 5(7.69%) out of 66 were found seropositive for HIV.

Table 2: Age wise distribution of patient tested for HIV

<table>
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<th>Age group in years</th>
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<th>Total Patient</th>
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<tbody>
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Although HIV is the initial causative agent in AIDS, most of the morbidity and mortality seen in immunocompromised patients results from opportunistic infections that take advantage of the lowered cellular and humoral defences of the patients. In the present study 297 out of 1786 patient tested were found positive for anti HIV antibodies showing an incidence rate of 16.63%. The studies undertaken by different workers like Tedaldi et al. (16.6%) Seme K. et al. (14.5%) and 15.0% for Slovenian and Croatian respectively coincides with the present study. Greater levels of education may also provide a framework of biological knowledge and understanding of causality into which HIV prevention messages can be assimilated. For example, children with a deeper understanding of the biological mechanisms of viruses are more resistant to myths about HIV transmission (Keselman, Kaufman, & Patel, 2004). A study in 32 countries found that literate women were more likely to know that a healthy looking person can have HIV and ways to avoid AIDS than illiterate women (Vandemoortele & Delamonica, 2000). In present study (Table 3) 59.27% illiterate patients found HIV reactive and as education increase prevalence of HIV positivity decrease.

CONCLUSION

HIV is most common in younger generation and particularly during productive period of their life. Younger peoples are sexually more active which may be the one reason of high prevalence of HIV among this group. HIV is most common in illiterate and incidence decreases with education.

REFERENCES