A person who is infected with both the hepatitis B and the HIV viruses is said to have a HBV/HIV co-infection. Approximately 10% of the HIV-infected population worldwide is infected with hepatitis B.

From the high risk groups of the patients attending the HIV clinic Jawahar Lal Nehru Hospital, Ajmer. Out of 256 samples we found 12 samples positive for HBV i.e. 4.68%. Highest prevalence among age group 36-45 years (50.0%), while not a single HBV positive case were recorded in age group <15 years. 6 out of 12 patients were between 36-45 year of age, which implies that HBV infection is more common in adult males.

This could be because of the increased exposure of this population to the various risk factors like promiscuity, blood transfusion, needle injury etc. In present study, we got 12 cases of accidental needle injury. Patients accidentally get infection with needles due to lack of awareness.

Since both the hepatitis B virus and the HIV virus share similar transmission routes, there is a high frequency of their co-infection. Sexual activity and/or injection drug use are the most common routes of transmission of the hepatitis B virus among those also infected with HIV.

Gilson et al., (1997) reports that hepatitis B virus (HBV) and HIV infections share risk-factors; therefore co-infection is common. Their study concludes the significant effect of HIV infection on the natural history of chronic HBV infection, which by prolonging the period of infectivity could have an important impact on the epidemiology of HBV infection in regions, or patient groups,
with high HIV seroprevalence. There was no evidence of an important effect of HBV carriage on HIV disease progression. Rodriguez et al. (2000) aimed to study the serological pattern of past HBV infection (presence or absence of anti-HBs) and the course of past HBV infection (changes in anti-HBs status, and HBV reactivation) in two cohorts of IDUs with and without HIV infection. The prevalence of HBV infection (either active or past) was higher in HIV-positive than in HIV-negative cases (90% vs 62%, p<0.001). HBV infection (either active or past) is particularly frequent in HIV-positive IDUs. Most cases have markers of past infection. Isolate detection of anti-HBc (absence of anti-HBs) is more common in HIV-positive than in HIV-negative IDUs. Despite their progressive immunosuppression, both anti-HBs loss and HBV reactivation are rare in HIV-infected IDUs. Human immunodeficiency virus (HIV)-infected persons are hyporesponsive to hepatitis B virus (HBV) vaccination.

The main goal of treating HBV/HIV-co-infection is to stop or slow down HBV viral activity as much as possible and for as long as possible.

Copper et al., (2008) stated that the immunostimulatory properties of CPG 7909 present an important strategy in achieving long-term protection in HIV-infected patients and other HBV vaccine-hyporesponsive populations. Cruciani et al., (2009) suggested that in HIV-infected patients with relatively high CD4 count, response to high dose of HBV vaccine is suboptimal. Rate of response may be increased by vaccine boosts, but antibody titers are significantly lower in non-responders than in responders to primary vaccination. Since persistence of anti-HBs titers appears significantly related
to antibody titers after the immunization procedure, monitoring of anti-HBs, particularly in patients with low level of protective antibody titers after primary vaccination or boosters, seems more than justified.

All HBV positive patients should be counseled about reducing the risk of HBV transmission to the close contacts and those who share injection drug equipment with the patients should be screened for HBV and vaccinated if they are not actively infected. Prevention strategies that work for viral hepatitis include immunization against HBV and education on safer sex for everyone and on harm reduction for IDUs. Safer blood and blood products and medical and medical practices are also important.

**HBV Therapy**

The goal of HBV treatment for persons with HIV co-infection is to suppress HBV replication and minimize ongoing hepatic damage. The key determinants for need of treatment for HBV are the HBV DNA level and the CD4 cell count. In HBV monoinfected patients, there is good correlation between high HBV DNA levels, long term histological progression to cirrhosis and the role of Hepatocelullar carcinoma. The accepted HBV DNA threshold for consideration for treatment is now>2000 IU/ml. The CD4 cell count is integral deciding when to initiate HIV therapy.

A threshold of 350 Cells/μl is recommended by British HIV Association Guideline (BHIVA, 2010) and other international guidelines as a level below which antiretroviral are indicated in HIV monoinfected persons (Gazzard B et
al., 2008). Coinfected patients with CD4 count between 350 and 500 cells/μl should also be treated with drugs active at suppressing both viruses (Soriano V et al., 2008).

**Indications for treatment of HBV in HIV infection**

HBV therapy is recommended for all HIV/HBV co-infected patients with abnormal Alanine aminotransferase (ALT) values of HBV DNA levels of >2000 IU/ml. Many experts recommend treatment of HBV for all HIV co-infected patients in whom any HBV replication is present. Any HIV/HBV co-infected patient with an indication for ART should be started on HIV treatment that includes effective anti-HBV treatment. Hepatitis B vaccination is the most effective measure to prevent hepatitis B virus (HBV) infection and its consequences, including cirrhosis of the liver, liver cancer, liver failure, and death.

The combination of (TDF) Tenofovir with Lamivudine (3TC) or FTC (emtricitabine) is recommended as a highly effective first line treatment for HBV (Centre for disease control and prevention, 2009). Lamivudine and emtricitabine, Entecavir, Telbivudine, Adefovir, Tenofovir use for HIV/HBV medication according to patients condition. In HIV/HBV co-infected individuals who require treatment for HBV infection, ART should be initiated irrespective of CD4 cell count or WHO clinical stage. On the question of when to start ART in HIV/HBV co-infection, there are no trials comparing early versus late initiation of ART. However, observational data support, that those with
HIV/HBV co-infection risk have an increased risk of fibrosis and cirrhosis. Some studies found 3 to 6 fold and 17 fold risk of developing chronic HBV in the individuals (Bodsworth, 1991; Hadler, 1991; Gatanaga, 2000).

While HBV has minimal effect on the progression of HIV, those with HIV/HBV co-infection have an increased risk of fibrosis and cirrhosis. Some studies found three to six fold risk of developing chronic HBV (Bodsworth, 1991; Gatanaga, 2000) and 17 fold increase risk of death (Thio, 2002) in HIV/HBV co-infected patients as compared with HIV negative individuals.

Management is complicated by limited availability of HBV testing and other markers of disease activity (HBV DNA, HBeAg). More laboratory needs for adequate HBV screening, evaluation of disease activity. These components can be implemented in areas where HIV programmes are already in place. HBsAg is available as an ELISA test and can use the same platform of HIV testing. HBV DNA can use the same lab platform of HIV viral load. Liver biopsy is commonly used in developed settings for evaluation of liver fibrosis. Common drugs used to treat HIV, HBV and some common epidemiological aspects of management of both diseases, suggest an integrated programmatic approach as the best strategy.

HCV seropositivity is associated with higher risk or death or developing AIDS related illness. Coinfection with hepatitis C virus (HCV) is common in human immunodeficiency virus (HIV)-infected individuals as a result of shared routes of transmission, and this coinfection represents a special challenge. For HIV-
HCV-coinfected individuals, the burden of disease is largely related to their HCV diseases, including a faster progression to liver fibrosis, cirrhosis and liver-related deaths (Clausen, 2014).

Studies find that HIV infection is associated with an increased risk of liver disease in the patients infected with HCV and HIV/HCV co-infection which may lead to a more rapid progression of liver fibrosis cirrhosis and liver related deaths than in those with HCV infection alone (Lesens et al., 1990; Makris et al., 1990; Eyster et al., 1993; Bierhoff et al., 1997; Soto et al., 1997; Pol et al., 1998; Benhamou et al., 1999). In one of the studies a median fibrosis progression rate (calculated by the fibrosis score divided by the duration of HCV infection) was found to be greater in HIV/HCV co-infected patients as compared to HCV monoinfected patients (Benhamou et al., 1999). Potential beneficial interaction between HAART and HCV include slowing of HCV related disease in patients on HAART. Existing data suggest that the long term use of protease inhibitor containing HAART regimen may have beneficial impact on HCV related disease independent of CD4 response (Benhamou et al., 2001).

Studies since year 2000 reports hepatitis C virus (HCV) infection in HIV-positive men who have sex with me in the US, Canada, Europe, Japan and Australia (Van de laar et al., 2010). The evidence points to blood as the medium of HCV exposure in sexual transmission (Schmidt et al., 2011; Pasquier et al., 2003).
HIV/HCV co-infected patients also demonstrated more rapid progression to clinical AIDS or death as compared to patients infected with HIV alone (Greub et al., 2000). The impaired recovery of CD4 cells in HIV/HCV co-infected patients was demonstrated in another study of patients’ recovery of CD4 cells in HIV/HCV co-infected patients was demonstrated in another study of patients initiating HAART as part of clinical trials. Further studies are clearly needed to understand the role of HCV on HIV disease progression.

In HIV-infected patients, acute HCV infection is more likely to become persistent, and HCV treatment is less likely to result in cure (Boesecke et al., 2011). The risk of advanced liver fibrosis increases almost double in co-infected patients and HIV treatment is complicated in the presence of HCV-related liver damage (Graham et al., 2001). Liver disease and hepatocellular carcinoma have become leading causes of death in HIV-infected individuals (Ghany et al., 2009) and the risk of hepatocellular carcinoma is three to eight times higher among co-infected vs HCV mono-infected patients (Sigel et al., 2011). In general, HIV/HCV co-infection is characterized by decreased response to therapies and increased rates of both HIV and HCV disease progression (Hadigan et al., 2011).

Another study by Eze et al. (2014) reports on one hundred and eighteen HIV-infected children, aged between eighteen months to fifteen years reports, eight of the HIV infected subjects positive for HCV, giving an HIV-HCV co-infection prevalence of 6.8%. Co-infection was more prevalent among males and in those in age group 11-15 years. Sagnelli et al. (2014) reports that Liver
histology remained substantially unchanged in human immunodeficiency virus/hepatitis C virus patients non-responder to anti-hepatitis C virus therapy over 4 years observation, suggesting an effective anti-hepatitis C virus early treatment for all hepatitis C virus/human immunodeficiency virus coinfected patients who can reasonably tolerate therapy.

By far studies indicate that, HIV infected individuals have a high probability of getting co-infected with HBV or HCV. HIV diseases progression and enhanced immunosuppression has a direct bearing on the natural history and pathogenesis of these infections. Sexual transmission of both HBV and HCV also appears to be significant and its epidemiological importance in the light of heterosexual transmission of HIV in India. Monitoring of HIV infected patients for concurrent infection with HBV and HCV is therefore necessary.

In HIV-infected individuals, coinfection with HBV and/or HCV in common because of shared modes of transmission. It is known that HIV accelerates progression of liver disease and results in increased morbidity and mortality associated with viral hepatitis, but it is less clear if viral hepatitis has a direct effect of HIV. Treatment of viral hepatitis improves outcomes and should be considered in all HIV-infected patients. Treatment of HBV without concurrent treatment of HIV is risky because resistance can occur both viruses if regimens are not carefully chosen (Petty et al., 2014). Another study of Matthews et al. (2014) states that there is a preponderance of HIV/HBV coinfection compared to HIV/HCV and significant caveats exist regarding the accuracy of published HCV seroprevalence surveys.
Few other studies taken up in Brazil, Myanmar & Nigeria reached to the Inferences that woman are less likely to become coinfected than men and the chance of coinfection increases with age (De-Oliveira et al., 2014). Men who have sex with men are at the highest risk of being co-infected with hepatitis B while intravenous drug users are at the highest risk of being co-infected with hepatitis C. It is important to screen for hepatitis B and C in HIV infected people in order to provide quality care for HIV patients with co-infection (Zaw et al., 2013). A study conducted by Tremewan et al. (2012) found that 7.9% were infected by 71 HBV, 2.3% with HCV and 0.7% with HBV & HCV i.e. the overall hepatitis-HIV prevalence is 10.8%.

Fernandez et al. (2012) suggests that since interaction between viruses generally amplifies liver damage, increasing the risk of developing end-stage liver disease and hepatocellular carcinoma. HIV-HCV co-infection is associated with poorer response to antiviral therapy. New antivirals against HCV are eagerly awaited for this population. HBV-HCV-dual infections are less common. The principles guiding indication of therapy in monoinfected patients should be followed considering which virus replicates in persons with serological markers of dual HBV-HCV infection. Although there is growing evidence supporting the use of direct acting antivirals (DAA) in dually infected patients with active HCV replication, prospective trials should be conducted to demonstrate their benefit, assessing carefully the rate and clinical consequences of HBV rebounds.
Syphilis is a global public health problem, resulting in an estimated 12 million new infections per year. Both pathogens are sexually transmitted, and the presence of one may facilitate infection with the other. The incidence of syphilis is rising all over the world, partly due to the increased transmission in HIV patients. Recognition of both the diseased and their complex interactions need importance and cautious treatments as manifestations of syphilis in co infection with HIV make difficult diagnosis, management and cure, with various problems leading due to the co infection. Similar to the HIV epidemic, the current syphilis epidemic predominantly affects three groups: men who have sex with men (MSM), injection-drug users, and individuals who engage in sex for money or drugs. Women account for a much smaller proportion of cases, but the incidence in this group has been increasing during the past 10 years.

HIV and *Treponema pallidum* coinfection is relatively common, accounting for approximately 25% of cases or primary and secondary syphilis reported in the U.S. Syphilis facilitates both HIV transmission and HIV acquisition, reflecting the complex interplay between the two diseases. Chancre cause epithelial and mucosal breaches, facilitating the transmission of HIV virions (Sellati et al., 2000). It is clear that apart from other infections, HIV infected individuals have high probability of getting co infected with syphilis. Multiple infection pose a small but definite risk to the recipients of blood product, voluntary donation are safer as compared with replacement ones and need to be encouraged (Kaur et al., 2010). Mathai et al. (2002) reported that 31942
donors screened over a 6 year period, mixed infections were seen in only 10 donors (0.03%).

Kaur et al. (2010) found that Syphilis infection can increase the susceptibility to HIV infection. HIV can alter the clinical course of Syphilis, increase the likelihood of relapse and co-found the diagnosis of neurosyphilis.

Swai and colleagues had reported age specific seroprevalence of Syphilis, in which the individual on the higher age group had higher seroprevalence. (Swai et al., 2006)

Whereas our study infer that HIV seropositivity was higher among the subjects aged between 15-34 years. This may be associated with higher sexual activities within these age groups. This suggests that women who are at the peak of their reproductive years are more prone to HIV infection. In all epidemiological studies, younger age has always proved to be the most important factors. The age of acquiring the infection is the major determinant of the incidence and prevalence rates. Since HIV prevalence among young pregnant women (15-24 years) is used as a proxy for measuring rates of new infections in population (Federal Ministry of Health Nigeria, Report, 2009). A risky sexual behavior are very common in India, while condom use remain low. The implications of HIV infection in pregnancy are serious. HIV seropositive pregnant women are significantly more likely to have recurrent vulvovaginitis, perineal tear, post partum hemorrhage, birth asphyxia and increased perinatal mortality (Obi., 2005). There is also great risk of vertical
transmission during parturition and breast feeding (Fawole et al., 2002). Swai and colleagues reported age specific seroprevalence of syphilis, in which the individual in the higher age group had higher seroprevalence. It seems that people start having unprotected sex at young age and have extramarital relationships (Swai et al., 2006). Having Syphilis once does not protect a person from getting it again. Following successful treatment, people can still be susceptible to re infection, because syphilis sores can be hidden in the vagina, rectum, or mouth it may not be obvious that a sex partner has syphilis. Detection and treatment of syphilis can, therefore, help to reduce HIV transmission. Syphilis may present with non typical feature in the HIV positive patients. There is a higher rate of symptomless primary syphilis and proportionately more HIV positive patient present with secondary infection. Secondary infection may be more aggressive and there is an increased rate of early neurological and ophthalmic involvement. The surest way to avoid transmission of sexually transmitted diseases, including syphilis is to abstain from sexual contact or to be in a long term mutually monogamous relationship with a partner who has been tested and in known to be uninfected. Rate of infection have increased since the turn of millennium in many countries often in combination with HIV. This has been attributed partly to unsafe sexual practices among men who have sex with men, increased promiscuity, prostitute.

Congenital syphilis in the new born can be prevented by screening during early pregnancy and treating those who are infected. World Health
Organization (WHO) recommends that all women are tested at their first antenatal visit and again in third trimester. If they are positive they recommend that their partners also be treated. A number of measures to increases testing appear effective at reducing rates of congenital syphilis in low to middle age groups.

HIV seropositive pregnant women are significantly more likely to have recurrent vulvovaginitis, perineal tear, post partum hemorrhage, birth asphyxia and increased perinatal mortality (Obi, 2005). This is also great risk of vertical transmission during parturition and breast feeding (Fawole et al., 2002).

In both the sexes *T. palladium* can spread throughout the whole body, infecting major organs. Brain damage and other serious health problems can occur, most of them are difficult to be treated. A woman who is pregnant and has not been effectively treated is at higher risk of putting her baby in danger. Untreated syphilis can also cause major birth defects. Syphilis also increases the risk of HIV infection because HIV can enter the body more easily when there is a sore present.

Early stages of syphilis can be cured with antibiotics. If some person in infected will be required the treatment for a longer duration. Once the damage occur in the body from the late stage of Syphilis, it is difficult to treat. Failure to diagnose and treat these devastating disease agents at an early stage may result in serious complications and sequela including infertility, fetal wastage, ectopic pregnancy, and genital cancer and premature death as well as
Discussion

neonatal and infant infection (WHO Guideline, 2003). HIV-infected patients with syphilis should receive the same treatment as HIV-uninfected patients. All HIV positive patients should be treated with penicillin based regimen that is adequate for the treatment of neuro-syphilis. Relapses are more likely to occur in HIV positive patient and careful follow up in required.

The recent syphilis epidemic among HIV-infected individuals reminds clinicians of an old pathogen with new features. The complex immunologic interplay between syphilis and HIV results in subtle but clinically relevant differences in presentation, diagnosis, and management strategies that must be recognized by those caring for HIV-infected patients.

Syphilis facilitates HIV transmission and acquisition as a result of local and systemic immune activation.

Syphilis may progress more rapidly and follow a more aggressive course in HIV-infected individuals. Neurosyphilis is common among HIV-infected patients and often presents atypically (e.g. asymptomatic infection, ocular syphilis). Serologic testing remains the primary tools for diagnosing syphilis in both HIV-infected and HIV-uninfected patients. Clinicians must maintain a high level of suspicion for asymptomatic neurosyphilis in HIV-infected individuals and should obtain a thorough history, perform a careful physical exam, and use clinical parameters (CD4-cell count and RPR titer) to help guide the decision to perform lumbar puncture. Treatment of syphilis does not vary between those are HIV-infected and those who are not.
The immunologic consequences of HIV infection may result in delayed serologic responses to nontreponemal tests following syphilis treatment. HIV-infected and HIV-uninfected patients experience comparable clinical responses to treatment.