"A CLINICAL STUDY OF CO-INFECTION OF HEPATITIS B AND HEPATITIS C VIRUS IN HIV INFECTED PATIENTS IN CORRELATION WITH CD4 COUNT AND LIVER ENZYMES"

By

Dr. SHASHANK.S.GOWDA. MBBS

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Under the guidance of

Dr. SANJEEV KUMAR N. BENTOOR M.D

PROFESSOR

DEPARTMENT OF MEDICINE

BLDE UNIVERSITY'S, SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR, KARNATAKA.

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Date:

Place: Vijayapur **Dr. SHASHANK S GOWDA**

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Date:

Dr. SANJEEV KUMAR N. BENTOOR M.D

Professor

Department of Medicine

Place: Vijayapur

Shri B.M. Patil MedicalCollege, Vijayapur.

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Seal & Signature of
HOD of Medicine

Dr. M. S. MULIMANI
M. D. (Medicine)
Shri B.M. Patil
Medical College, Hospital &
Research Centre, Vijayapur
Date:
Place: Vijayapur

Seal and signature of
the principal

DR. S.P.GUGGARIGOUDAR
M.S. (ENT) BLDEU's
BLDEU's Shri B.M. Patil
Medical College, Hospital &
Research Centre, Vijayapur.
Date:
Place: Vijayapur

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& RESEARCH CENTRE, VIJAYAPUR

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Date:

Place: Vijayapur **Dr. SHASHANK S GOWDA**

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Dr. SHASHANK S GOWDA

ABSTRACT

Background:

Human immunodeficiency virus (HIV) and Hepatitis B and C virus (HBV&HCV) are three most common chronic viral pathogens of major public health concerns. These viruses have similar routes of transmission, namely through blood and blood products, sharing of needles to inject drugs and sexual activity. People at high risk for HIV are also likely to be at risk for other infectious pathogens, including HBV or HCV enabling co-infection with these viruses a common event.In co-infection, the presence of one virus impacts the natural history of the other virus. HIV accelerates the natural course of HBV and HCV infection and facilitates faster progression of liver disease to cirrhosis and hepatocellular carcinoma.

Objective:

To study the prevalence, risk factors (probable mode of acquisition), signs and symptoms of co-infection of Hepatitis B and Hepatitis C virus in HIV infected patients in correlation with CD4 count and liver enzymes.

Methodology:

The information for the study was collected from HIV positive patients admitted to BLDEU'S Shri B.M Patil Medical college Hospital and Research center,Vijayapur from November 2015 to June 2017.Information was collected through prepared proforma from each patient. All patients were interviewed as per the prepared proforma and then complete clinical examinationwas done.

Inclusion Criteria:

• HIV infection diagnosed as per NACO guidelines–

Diagnosed by 3 spot tests:1. Coombs AIDS test

2. Triline test

3.Qualpro test

Exclusion Criteria:

1. HIV negative patents.
2. Patients not willing to take part in the study**.**

Results:

Common age group for HIV positive patients was 31-40 years (41.7%). Males out number females with ratio of 13:7 [64.9% : 35.1%]. Multiple sexual partner behaviour as a risk factor for HIV accounted for about 46.4%. HbsAg positive in HIV positive individuals were recorded in 31 patients (14.7%). Anti HCV antibodies status in HIV positive individuals were recorded in 8 patients (3.8%). The age group of 31-40 years was the commonest period for HBV and HCV co-infection in HIV individuals. Mean CD4 counts were 154.3 and 135.5 for HbsAg and Anti HCV positive HIV patients respectively. Mean SGOT and SGPT levels were 229.4 and 168.4 respectively in HbsAg positive HIV patients. Mean SGOT and SGPT levels were 467.6 and 239.8 respectively in Anti HCV positive HIV patients. Mean Serum bilirubin was 1 and 2.4 in HbsAg positive and Anti HCV positive HIV patients respectively.

Conclusion:

Co-positivity with HBV in HIV positive patients was found to be 14.7%. Co-positivity with HCV was found to be 3.8%. Age group 31-40 years showed highest co-positivity for HBV. Age group 31-40 years showed highest co-positivity for HCV. Study showed Male preponderance. Study also shows that highest co-positivity is seen in patients whose CD 4 count was <200. Liver enzymes are markedly elevated in co-infected cases. Study of viral DNA levels could not be done because of non-availability of facilities in our set up; this could have been more sensitive in finding the exact co-positivity of HbsAg and Anti HCV with HIV infection.

Key Words: Human immunodeficiency virus (HIV), Hepatitis B virus (HBV), Hepatitis C virus (HCV), CD4 Count, Liver enzymes.

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INTRODUCTION

The three very common chronic viral infections seen in the world are Human immunodeficiency virus (HIV), Hepatitis C virus (HCV), and Hepatitis B virus (HBV).These viruses have the same routes of transmission and therefore there is high chance of their co-existence in the same host. As there is immunosuppression due to HIV infection, it leads to harmful effects on the natural history, pathophysiology, diagnosis, therapeutic responses to hepatitis B and hepatitis C viruses. HBV vaccination has less efficiency in persons with HIV infection. Co-infection of HIV with HBV and HCV is likely to become a major health care concern in the future.

With the advent of HAART, survival among HIV positive patients is increasing and mortality due to HIV infection is reducing, where as mortality because of liver diseases are increasing in HIV infected patients.

Viral hepatitis has become one of the major causes of morbidity and mortality in HIV-infected patients all over the world.[50]

There are increased levels of HCV RNA in patients with Co-infection of HCV with HIV. This leads to accelerated progression of HCV-related liver disease. In HCV infection, co infection of HIV, consumption of alcohol, increase in the age, and CD4 count <200 cells/mm are the risk factors for increased rate of progression of fibrosis.[50]

There is increased risk of cirrhosis, end stage liver disease, hepatocellular carcinoma in patients with HIV-HCV co-infection and -occurs at a younger age.[50,51]

Co-infection of HBV and HCV in HIV may lead to faster progression to liver cirrhosis and a greater risk of anti-retroviral therapy induced hepatotoxicity.[53]

Co-infection of HIV with HCV and HBV is common because of similar routes of transmission and risk groups. Human immunodeficiency virus infection seems to affect the natural history of infection with certain hepatitis viruses. There is interaction between the HIV and concurrent infection with hepatitis viruses, which may change the natural history and response to the treatment of both diseases.[1]

There is increased epidemiological similarity between HIV and HBV infection as regards to high risk groups, mode of transmission and the presence of virus in body fluids.[2]

Currently Hepatitis C virus infection is being identified as a main problem. A well-known route of transmission of HCV is blood transfusion. HCV infection in large number of patients is caused by intravenous drug abuse or administration of blood products.[3]

The significance of transmission of HCV through sexual intercourse is controversial.

There is increased viral load of Hepatitis viruses in patients with co-infection of HIV with HBV/HCV which results in increased amount of liver damage.[1]

The presence of HBV/HCV in HIV may advance quickly to liver cirrhosis and lead to an increased threat of antiretroviral therapy induced hepatotoxicity. The studies on co-infection of HCV and HBV in HIV infection are scarce in our country.

There is more chance of liver disease, cirrhosis, and mortalities in a person having co-infection of HIV with HCV/HBV when compared to a person who is having infection of only one of these viruses. Hence, identifying HCV and HBV in HIV infected individuals is important in order to take care of these infections and allocate resources in health plans so that all HIV positive individuals should be tested for both HCV and HBV.[52]

Therefore this study was undertaken to find the prevalence of co-infection of HCV and HBV in HIV infected patients, and need for prevention of HCV by education and HBV through immunization to be stressed in practice for better outcome in HIV patients.

OBJECTIVE OF THE STUDY:

To study the prevalence, risk factors (probable mode of acquisition), signs and symptoms of co-infection of Hepatitis B and Hepatitis C virus in HIV infected patients in correlation with CD4 count and liver enzymes.

REVIEW OF LITERATURE:

HIV INFECTION

History of HIV infection: The emergence and pandemic spread of acquired Immunodeficiency disease has led to a big challenge to public health in present times. The full result of this phenomenon was not evident for several years because of the silent spread and slow evolution.[4]

There are scientifically un-authenticated cases having similar clinical features in Ayurveda in 800 BC described by Susrutha, Charaka & Vaghabhatta of HIV.[5]

This new syndrome was first identified in the summer of 1981, reported from New-York and California of a sudden rise in the occurrence of two very infrequent diseases. Pneumocystis carinii pneumonia's and Kaposi's sarcoma in young individuals who were addicted to heroin or other IV narcotic or had sex with people of their own sex.[6]

The first case of AIDS was reported in India in 1985 at Madras.[5]

HIV has now been identified in more than 193 nations across the world with largest distribution of HIV infected individuals in Africa followed by Asia.[7,8]

Isolation of etiological agent was first reported in 1983 by Luc Montagnier and colleagues from Pasteur Institute. They isolated a retrovirus and called it Lymphadenopathy Associated Virus (LAV).[6]

Retrovirus from AIDS patient was isolated by Robert Gallo and Colleagues from National Institute of health, USA, in the year 1984 and they called it Human T cell lymphotropic virus III. Other isolates of same kind were reported under other names like AIDS related Virus etc.[9]

To resolve this nomenclature confusion the international committee on virus nomenclature in 1986 decided on generic name Human Immunodeficiency virus for these viruses.

DEFINITION[10]

AIDS case definition has been recommended by WHO for use in adults and adolescent (>12 years) for surveillance. The recommended case definition depends on whether testing for HIV is available or not.

1. WHO case definition for AIDS surveillance where testing for HIV is not available

Case definition for AIDS is fulfilled in the presence of at least 2 major signs and 1 minor sign.

MAJOR SIGNS:

a. Fever for >1 month.

b. Loss of weight> 10% of body weight. Diarrhea for > 1 month.

c. Diarrhea for > 1 month

MINOR SIGNS:

a. Generalized pruritus.

b. Cough > 1 month.

c. Candidiasis of the oropharynx

d. Herpes zoster history

e. Disseminated or chronic progressive herpes simplex infection.

f. Generalized peripheral lymphadenopathy.

(Cough for >1 month should not be considered as a minor sign for TB patients)

This clinical definition is relatively specific and relatively insensitive.

II. WHO case definition for AIDS surveillance where testing for HIV is available

The AIDS case definition is fulfilled if test for HIV is positive and there is presence of one or more of the following-

1. Chronic cachexia with fever or diarrhea or both, or loss of weight > 10% of body weight for > 1 month unknown to be secondary to a disease not related to HIV.

2. Kaposi's Sarcoma

3. Cryptococcal meningitis.

4. Candidiasis of oesophagus

5. Neurological impairment, unknown to be secondary to disease with no relation to HIV infection which hinders to perform daily routine activities

6. Invasive Ca cervix.

7. Tuberculosis

8. Recurrent episodes of pneumonia or life threatening pneumonia.

The case definition of AIDS in current use as laid down by the centre of disease control is for surveillance purpose – 1993.[7]

A. Indicators of disease definitively diagnosed in the absence of other immunodeficiency causes and without a laboratory evidence of HIV infection.

1. Cryptococcosis.

2. Candidiasis of trachea, bronchus, lungs, oesophagus.

3. Cryptosporidiasis with diarrhoea persisting for more than 1 month.

4. Cytomegalovirus disease.

5. Herpes simplex virus infection persisting more than 1 month.

6. Kaposi's sarcoma in a patient < 60 years.

7. Lymphoma of a brain in patient <60 years of age.

8. Lymphoid interstitial pneumonia or pulmonary lymphoid hyperplasia in a child< 13 years.

9. Mycobacterium avium complex and mycobacterium Kansassii disease.

10. Brain toxoplasmosis in patient > 1 month of age.

11. Pneumonia caused by Pneumocystis carinii infection.

B. Indicator of disease diagnosed irrespective of presence of laboratory evidence of HIV and other immunodeficiency causes.

1. Any disease listed in A.

2. Infection in children < 13 years of age (Multiple or recurrent) caused by haemophillus, streptococcus or other pyogenic bacteria.

3. HIV encephalopathy.

4. HIV wasting syndrome.

5. Isosporiasis with diarrhea > 1 month.

6. Kaposi's sarcoma at any age.

7. Non-Hodgkin's lymphoma of B cell of unknown phenotype.

8. Any disseminated Mycobacterial disease. 9. Recurrent salmonella septicaemia.

10. Histoplasmosis disseminated.

C. Indicator disease presumptively diagnosed in the presence of Laboratory evidence of HIV infection.

1. Kaposi's sarcoma.

2. Pneumonia caused by Pneumocystis carinii infection.

3. Candidiasis of oesophagus.

4. Disseminated mycobacterial disease.

5. Pulmonary lymphoid hyperplasia and / or lymphoid interstitial pneumonia in a child < 13 years.

6. Loss of vision due to CMV retinitis.

7. Brain Toxoplasmosis in patients > 1 month of age.

D. Indicator disease definitively diagnosed in the presence of negative results for HIV infection and in the absence of other immunodeficiency causes.

1. Other indicator disease in sec A with a CD4+ count<400/ mm3.

2. Pneumonia caused by Pneumocystis carinii infection.

E. Expanded definition of 1993 includes

1. All HIV positive persons who have < 200 CD4+ T lymphocyte count per mm3 or a CD4+ T Lymphocyte % of total lymphocyte < 14%.

2. Invasive Ca cervix.

3. Recurrent pneumonias.

4. Pulmonary tuberculosis.

EPIDEMIOLOGY OF HIV AND AIDS[11,12]

- HIV infection/AIDS cases have been reported from almost all nations. Hence it has become a pandemic disease.
- The Joint United Nations Programme on HIV/AIDS (UNAIDS) has reported that about 33.3 million people were living with HIV infection at the end of 2009.
- Greater than 95% HIV/AIDS patients are from developing; 50% of them belong to female sex, and 2.5 million of them are <15 years of age.
- Since 1990, the global prevalence has raised nearly fourfold since 1990, showing that the incidence of new HIV infections are increasing and antiretroviral therapy has been useful in prolonging their lifespan.
- In India by Dec. 2003 the estimated people infected by HIV virus was 5.1 million cases. By Aug 31, 2004, 86028 cases of full blown AIDS were detected of whom 10 males were 62050 and females 23978. Tamil Nadu tops the list with more than half the cases. Maharashtra is next with 21% of cases.
- The epidemic of HIV infection has taken place in "waves" in various parts of the world, and there was rather unlike features in each wave determined by the demographics of the nation and part of the globe in question and the time at which HIV has been instituted into the population. Even though the epidemic of AIDS was initially identified in The United States and little later in Western Europe, it most probably started in sub-Saharan Africa, which was especially ruined by the epidemic. Although sub-Saharan Africa accounts for only 10–11% of the world's population, greater than two-thirds of HIV infected people (22.5 million) reside in that part of the world. Amongst them most of them are from southern Africa.

According to the sero-prevalence data which is present it is noted that >10% of the adult population that is 15–49 years of age is HIV positive in all the nine southern African nations. The sero-prevalence of HIV infection is presently more than 50% in few nations amongst the high risk population that is patients attending STD clinics, sex workers residing

in urban regions of Sub-Saharan Africa. Sub-Saharan Africa's HIV regional epidemics vary significantly, with most appearing to have stabilized, although frequently at very high levels.

Heterosexual exposure is the primary mode of HIV transmission in sub-Saharan Africa, with women and girls disproportionately affected, accounting for60 percent of all HIV infections in that region. In 2009, an estimated 460,000people were living with HIV in the Middle East/North Africa region. Cases are largely concentrated among IDUs, men who have sex with men, and sex workers and their clients.

- In east, south, and south-east Asia, an estimated 4.9 million people were living with HIV at the end of 2009. In this region of the world, the trends between different nations vary largely and in the south-east Asian nations the national HIV prevalence is the highest.

Among Asian countries, only Thailand has an adult sero-prevalence rate of >1%. Even low infection and sero-prevalence rates lead to more number of HIV infected people in many Asian nations like India and China because of their huge population. Although Asia's epidemic has been concentrated for some time among specific populations—sex workers and their clients, men who have sex with men, and IDUs—it is expanding to the heterosexual partners of those most at risk. While the regional epidemic appears to be stable overall, HIV prevalence has increased in certain nations like Bangladesh and Pakistan.

- At the end of 2009 it was found that 1.4 million of the population in Eastern Europe and Central Asia were HIV infected. This shows that the epidemic is increasing in these regions.

The Russian Federation and Ukraine account for the majority of HIV cases in the region; the Ukraine has an adult sero-prevalence rate of 1.1%, the highest in all of Europe. There is drastic rise in the number of new infections in this part of the world since past 10

years as it was steered in the beginning by injection drug use and later more by heterosexual transmission.

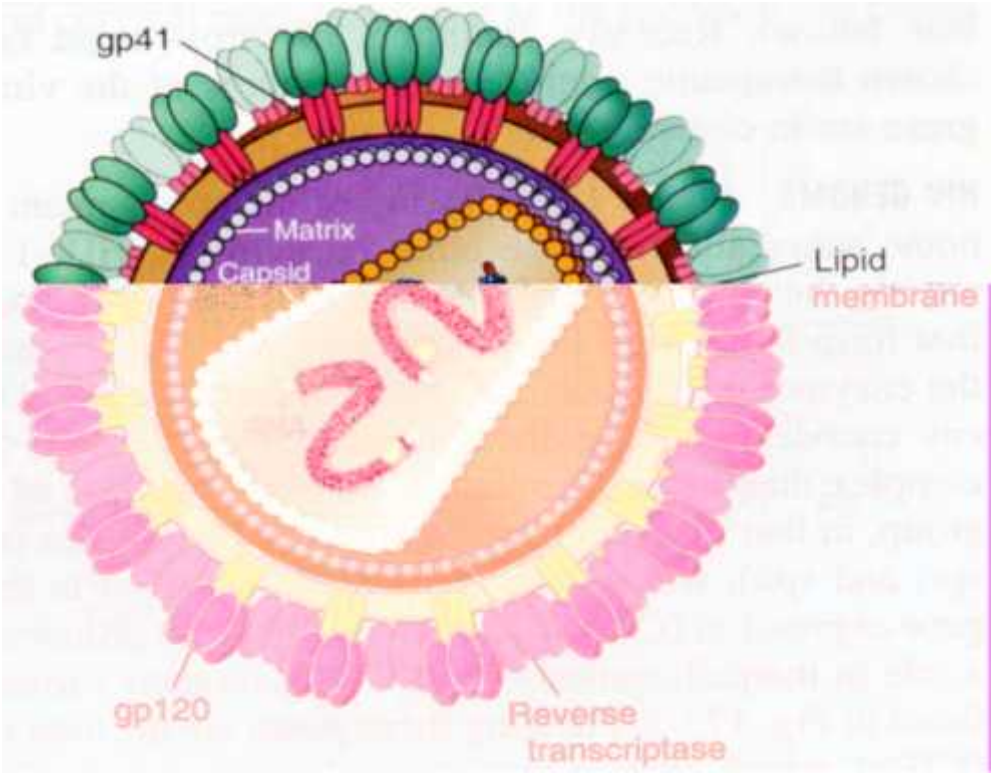
- HIV infection is present in nearly 1.6 million people in West Indies, Central and South America. In this part of the world, most number of HIV infected individuals are residing in Brazil. Nevertheless the epidemic has decreased in that country because of proper use of preventive methods and efficient treatment. Men who have sex with men account for the largest proportion of HIV infections in Central and South America.

The West Indies region has the largest number HIV infected population, second to Africa. This is because there are large number of people residing with HIV infection in Haiti amongst these West Indian countries. Heterosexual transmission, often tied to sex work, is the main driver of transmission in the region.

- In North America, western and central Europe, and Oceania, nearly 2.4 million HIV infected individuals are residing. The number of new infection among homosexual men has become more in the last 10 years in these mostly high-income areas, while rates of new infections among heterosexuals have stabilized and infections among IDUs have fallen.

ETIOLOGY[12]

- The etiological agent of AIDS is Human Immunodeficiency Virus (HIV), which belongs to the family of human retrovirus and a subfamily of lentivirus.
- The four groups which are human T lymphotrophic viruses, HLT - I and HLT- II and the human immunodeficiency virus HIV I and HIV II.
- The most common cause of HIV worldwide is HIV I. HIV II was first identified in1986 in West Africa and its number is increasing worldwide.



MORPHOLOGY[12,13,14]

Figure 1: Structure of HIV

- Electron microscopy suggest that the shape of a HIV virion is icosahedral having large number of outside spikes which is constituted by gp120 and gp4l, the two major envelope proteins. The virion buds from the exterior layer of the host cell and assimilates few proteins including Major Histocompatibility Complex (MHC) class I and II antigens.
- Internally, it is electron dense central core which in turn encloses 2 similar copies of a lone typical viral RNA genome.
- Alongside the RNA genome the DNA polymerases, known as reverse transcriptase are found which is a distinguishing feature of retrovirus. The core antigen p24encloses the viral genomes and is highly antigenic.

PATHOPHYSIOLOGY AND IMMUNOPATHOGENESIS[7,8,10,15]

There are 2 major targets of HIV, the CNS and the immune system.

IMMUNE SYSTEM

Intense immunosuppression mainly acting on the cell mediated immunity is the characteristic feature of AIDS. This is mainly due to a severe suppression and infection of CD+4 cells as well as loss of functioning of surviving T helper cells.

It is seen that CD4+ molecule has high affinity for HIV. The HIV binds to the cell and then HIV gp120 binds to other surface molecules (co-receptors) and gains entry into the cell. This is served by 2 cell surface molecules CCR5 and CX CR4. Binding of gp120 to CD+4 cells is the starting step of infection. The second step is gp41 glycoprotein attaining conformational changes. This will lead to the introduction of a fusion peptide at the end of gp41 into the cell membrane of the macrophages or target T cells. The virus core consisting of the HIV core, after the fusion, invades the cytoplasm of the cell. There is strong evidence that HIV binding to its co-receptors is important in the pathogenesis of AIDS. Early in the course of the disease, HIV colonizes the lymphoid organs (Spleen, lymph nodes, tonsils).

The first cells affected are CD4+ T cells. There is production of nearly 100 billion viral particles daily and 1-2 billion CD4+T cells die daily. Early in the infection the body can regenerate the dying T cells, so the loss of CD4+ counts is low.

This is also low as the cells die in the lymphoid organs and not the peripheral circulation. Next affected are the macrophages. These are also infected in the body tissue and not in the circulation. Infection of the macrophages has 3 major implications, First macrophages and monocytes act as a reservoir for HIV to body’s different parts. Secondly in later stages when the amount of CD4+ T cells reduces, macrophages are the major site of viral replication. Third, the macrophages carry HIV virus to the CNS. Infection of monocytes is generally low.

HIV is known to thrive when host macrophages and T cells are physiologically activated from within due to antigenic stimulation by other infecting micro-organism like CMV, EBV, Hepatitis B Virus and M. Tuberculosis. In US the increased risk of HIV is attributed to recurrent sexually transmitted diseases while in developing countries like African and Indian sub-continent socio-economic conditions play a role to chronic microbial infections. The multiple infection to which the patient are prone due to decreased T helper cells leads to increase production of pro-inflammatory cytokines, which in turn stimulates more HIV production which in turn causes decrease CD4+ T - helper cells, thus making a vicious cycle.

PATHOGENESIS OF CNS INVOLVEMENT

HIV infection mainly affects central nervous system. Monocytes and macrophages are the major cells involved in CNS infection of HIV. It is thought that infected monocytes transport HIV to the brain. The way by which brain is damaged by HIV is not known.

TRANSMISSION OF HIV

HIV is transmitted by both heterosexual and homosexual contact by blood and blood products, by infected mothers to infants either intra partum, perinatally or via breast milk. There is no evidence that HIV can be transmitted by casual contact or through insect bites.

1. SEXUAL TRANSMISSION[10,16]

HIV is a predominantly sexually transmitted disease worldwide. In developing countries heterosexual transmission is most common form of transmission while in countries like USA homosexual contact accounts for half the cases.

HIV has been identified in seminal fluid, vaginal and cervical secretions. There is high chance of HIV transmission in individuals receiving anal sex as the seminal fluid and the cells beneath are separated only by a thin mucous membrane, and also trauma is frequently

associated with anal intercourse. The chances of transfer of HIV infection from male to female is 20 times higher than that of female to male.

Risk of HIV transmission has been estimated in different sexual behaviors as follows. In receptive anal it is highest that is 1:30-1:100. In insertive anal it is 1:1000, receptive vaginal 1:1000 and in insertive vaginal it is 1:10000. Infection is higher in people with genital ulcerations.

Oral sex appears to be a less effective mode of transmission but is no way a safe from of sex as HIV transmission has been estimated to be 1:1000 in people who had only oral sex.

2. BLOOD AND BLOOD PRODUCTS[10,16]

HIV infection can be transferred among people who share contaminated needles and syringes in 1:150 and by those who receive contaminated blood and blood product transfusion there is 95% risk of transmission. Over the last few years with strict screening procedures the transmission by this route is reduced and it is 1 in ten lakh which is usually because of transmission in window period.

In I.V. drug users, more the needle sharing frequency where many people share the same needle and duration of use of injection more is the chance of transmission of HIV infection.

3. OCCUPATIONAL TRANSMISSION OF HIV[10,16]

The health care workers, lab workers and people who handle specimens infected by HIV especially the sharp objects are definitely prone to HIV transmission even though the risk is less. The risk of infection from a needle prick which is contaminated is approximately 0.3%. The chance of transfer of infection from infected health care workers to patients is very-very low.

4. MATERNAL - FETAL / INFANT TRANSMISSION[10]

This is significantly a main mode of transmission in countries with low socio-economic status.

In the perinatal period, maternal to fetus transmission is most common. Caesarian section reduces this mode of transmission considerably. 25% of children born to infected mothers get infected and this increases to 45% with prolonged breast feeding. Thus colostrum and breast milk have been implicated as vehicles of transmission.

5. TRANSMISSION BY OTHER BODY FLUIDS

It is believed that HIV can be transmitted by other body fluids like saliva, tears, sweat or urine. But there is no clear evidence.

DIAGNOSIS AND LAB MONITORING OF HIV INFECTION

Before HIV - I was identified diagnosis was purely on clinical grounds. In 1984 HIV was discovered as the causative organism for AIDS and since then rapid development has taken place in screening for HIV infections. Since March 1985 donors have been routinely screened for HIV and since June 1996 the P24 antigen capture assay has been added in the USA. At present a wide array of tests are available for diagnosing HIV infection. Enzyme linked immune-sorbent assay (ELISA) which has a sensitivity of over99.5% but not optimal with regard to specificity is the standard screening test for HIV. Western blot is the most commonly used confirmation test. In patient with positive ELISA but negative western blot, it can be concluded that the ELISA reactivity was false positive, but western blot is truly a confirmation test. If western blot is intermediate positive then it should be repeated after 1 month.[17,18,19]

For screening a patient the first test is ELISA. If negative the HIV infection is ruled out. If positive first it should be done again. If it is negative on 2 occasions after first positive

HIV infection is ruled out. If the test is intermediate positive then western blot should be done and it decides if patient is positive or negative.[20,21]

OTHER TESTS[12]

P24 antigen capture assay.

HIV RNA by PCR.

DNA PCR – Detecting HIV Proviral DNA.

LAB MONITORING OF PATIENT WITH HIV[12]

1. CD4 + T cell counts.

2. Direct culture of HIV from plasma or peripheral blood

3. Direct measurement of HIV RNA

4. ß2microglobulin levels.

5. Neoptrin levels.

The lab test which is usually preferred is the CD4+ T cell count.

CLINICAL MANIFESTATION OF HIV[12,22]

CLINICAL CATEGORIES

Category A: Constitutes one or more of the disorders mentioned below in an individual aged >13 years with documentation of infection by HIV. Disorders mentioned in categories B and C should not exist.

HIV infection without any symptoms.

HIV infection which is primary and acute with accompanying illness or history of acute HIV infection.

Lymphadenopathy which is generalized and persistent.

Category B: Constitutes disorders which are symptomatic in an individual aged > 13 years who are infected with HIV which are not mentioned in clinical category C and which meets one or more of the criteria mentioned below:

(1) The disorders indicating a defect in cell-mediated immunity or attributing to infection by HIV.

(2) The conditions needing management or having a course clinically which is regarded by the physicians to be complicated by HIV infection. Examples comprise the conditions mentioned below, but are not limited to the following:

Dysplasia of cervix (moderate or severe) or Ca cervix in situ.

Diarrhea or fever (38.5°C) lasting >1 month

Oral candidiasis or pharyngeal candidiasis.

Vulval candidiasis or vaginal candidiasis; frequent, persistent or poorly responsive to treatment.

Epithelioid-angiomatosis (Bacillary angiomatosis).

Immune thrombocytopenic purpura.

Peripheral neuropathy.

Pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess.

Listerioris.

Oral Hairy leukoplakia.

Shingles (Herpes zoster) involving more than one dermatome or not less than two distinct episodes or more than one dermatome.

Category C: Disorders mentioned in the AIDS surveillance case definition.

Chronic intestinal cryptosporidiosis for more than 1 month duration.

Chronic intestinal isosporiasis for more than 1 month duration.

HIV related encephalopathy.

Chronic ulcer(s) for more than one month duration caused by herpes simplex virus; or oesophagitis, bronchitis, or pneumonia.

Extrapulmonary or disseminated Histoplasmosis.

Invasive Ca cervix.

Multifocal progressive leukoencephalopathy.

Pneumonia caused by Pnemocystis jiroveci.

Recurrent pneumonia.

Primary lymphoma of brain.

Extrapulmonary or disseminated coccidiomycosis.

Extrapulmonary cryptococcosis.

Pumonary or extrapumonary tuberculosis caused by Mycobacterium tuberculosis, Mycobacterium kansasii, or Mycobacterium avium complex.

Extrapulmonary or disseminated tuberculosis caused by other or unidentified species of Mycobacterium.

Primary lymphoma of brain.

Burkitt's lymphoma.

Kaposi's sarcoma.

Brain toxoplasmosis.

Recurrent septicemia caused by Salmonella.

Cytomegalovirus disease involving liver, spleen or nodes.

Bronchial, tracheal or pulmonary candidiasis.

Oesophageal candidiasis.

Cytomegalovirus retinitis (with loss of vision).

ASYM**PTOMATIC INFECTION**[23]

Many individuals are infected with HIV but have not yet developed symptoms. Although the proportion of those individuals who will ultimately progress to AIDS is not known the vast majority will eventually develop signs of infection.

Most if not all asymptomatic individuals remain infectious and can transmit HIV to susceptible persons.

ACUTE VIRAL SYNDROME[12,23]

A majority of patients infected with HIV develop an acute mononucleosis-like illness characterised by fever, headache, lymphadenopathy, pharyngitis, macular rash, and malaise within several weeks of exposure. Aseptic meningitis, hepato-splenomegaly, extreme fatigue, weakness, arthralgia, myalgia are frequently associated with this syndromes. Symptoms usually resolve within 2 to

4weeks, but rarely rapid progression with early development of an AIDS defining condition has been described. At this stage HIV screening test may not demonstrate antibody. However, test for HIV P24 antigen are usually positive early and should be suspected in any individual at risk in whom at risk an unexplained febrile viral-like illness exists.

PERSISTENT GENERALISED LYMPHADENOPATHY[12,23]

Lymphadenopathy, defined in this setting as enlargement of the lymph nodes in at least two extra inguinal sites for a minimum of 3 months in the absence of any illness or drug known to cause lymphadenopathy, is usually present and may be caused by immunologic response to infection with HIV. A progressive decrease in the size of the involved nodes correlates with the onset of AIDS and portends a bad prognosis.

SYMPTOMATIC INFECTION[10,12,23]

The mean time taken for the evolution of AIDS from a primary HIV infection is nearly 10 years. Even though there is mounting of vigorous responses from humoral and cell-mediated immunity after primary infection it is the unique quality of HIV infection to be not cleared entirely from the body of the host with very less number of exceptions.

Instead the virus with varying degree of replication exists in the body of the host for nearly 10 years after which the patient presents with clinical illness. Nevertheless long term survival and non-progression of the diseases is encountered in less than 5% of patients infected by HIV.

A defect in the virus in some, a variety of host factors including genetic factors or combination of both in others explains the lack of progression of the disease.

NEUROLOGICAL DISEASE[12]

The neurologic problem may be the first to occur in the overall process of infection pathogenically or may be second to the occurrence of malignancies or opportunistic infections. In the primary infection state neurologic symptoms include. Headache, Nausea and vomiting, seizures. Encephalopathy and neuropathy. Rare manifestations: Aseptic meningitis. Encephalitis. Myositis. Acute Rhabdo-myolysis.

SECONDARY INFECTIONS[24]

I. Bacterial Infections

a. Mycobacterial Infections

Worldwide mycobacterial infections head the list of secondary bacterial infection in HIV patients. The main difference' between the western countries and developing countries is that in the former atypical mycobacteria predominates (MAIC and M. Kansasii) where as in the later M. Tuberculosis is rampant.

b. Non-mycobacterial Infections:

Streptococcus Pneumonias, Haemophilus influenza, Pneumococcus,

Staphylococcus and enteric organisms.

c. Syphilis

II. Fungal Infections

a. Candida - oral thrush, oesophagitis, oropharyngeal candidiasis.

b. Cryptococcus neoformans.

c. Histoplasma capsulatum.

III. Viral Infections:

a. Herpes simplex virus infection.

b. Varicella zoster virus infection

c. Cytomegalo virus infection.

d. Ebstein Barr virus.

e. Hepatitis virus; B. C. and D virus.

IV. Protozoan infections

a. Pneumocystis carnii

b. Cryptosporidium parvum

c. Toxoplasma gondii

Other, microsporida, Isospora belli, E. Histolytica, G. Lamblia and Visceral leishmaniasis.

V. NEOPLASTIC DISEASES[12]

a. Kaposi's Sarcoma.

b. Lymphoma 3 types.

1) Grade III and IV immunoblastic lymphoma.

2) Burkitts lymphoma.

3) Primary CNS lymphoma.

c. Cervical and oral intraepithelial dysplasia.

OTHER CONDITIONS

a. HIV associated enteropathy

b. Hematologic problems-neutropenia, thrombocytopenia, anemia.

c. Dermatologic manifestations – seborrheic dermatitis, scabies, psoriasis,

d. Icthyosis, molluscum contagiosum.

e. Cardiac diseases - cardiomyopathy.

f. Ophthalmic manifestations.

g. Psychiatric disorder.

SEROLOGICAL TESTS TO DETECT HIV 1 AND 2 ANTIBODY

1. ELISA[7,13]

Procedure of the test

The immune-dominant HIV-1 AND HIV-2 epitopes are represented by synthetic peptides. The microplates's wells are coated by these synthetic peptides, to which diluted serum of plasma samples are added. There will be formation of stable complexes with the peptide antigen of HIV of the well if the sample contains specific HIV-1 and / or HIV-2 antibodies.

A horseradish peroxidase labeled goat anti-human IgG is added. There is binding of the peroxidase conjugate and it stays in the well if there is presence of antigen/antibody complex.

Observations & Interpretation

Then enzyme substrate is put. In proportion to the quantity of antibody present there is formation of a blue color during incubation. Samples prevail to be colorless if there is absence of anti - HIV antibody. An acid stop solution is added to each well and the yellow colour read on a micro plate reader at 450 nm.

2. WESTERN BLOT TEST (W.B.)[7,13]

W.B. is used to detect unknown antibodies by known antigens. It is the most commonly used confirmatory test for HIV. It is also known as Immunoblot test. In W.B. test HIV is broken down (separated) into protein fragments in polyacrylamide gel. The separated proteins are blotted electrophoretically from the gel into a strip of nitrocellulose sheet. These strips are incubated with test sera.

There will be binding of anti HIV antibodies existing in test serum to any or all HIV fragments. Enzyme conjugated antihuman gamma globulin are used to wash and treat the strips and are allowed to react. Bands with colors are produced after an appropriate substrate is added. The HIV antigen fragment to which the anti HIV antibodies in the serum have bound is indicated by the location of the color bands. Usually bands with multiple proteins are formed in a typical case. Antibodies to core proteins

(p-24, p-31) and envelope proteins (gp-41, gp-120 and gp-160) are commonly detected. A fall in the titre of anti p-24 antibodies indicated poor clinical prognosis.

Interpretation of W.B. test is sometimes difficult when bands are formed only at one or two sites, as with p-24 and gp-120. This type of result may be obtained in early infection but may be also non-specific. A completely reactive result is useful.

INFLUENCE OF HIV ON TRANSMISSION OF HCV

There is some proof that the possibility of transmission of HCV may be increased by the HIV co-infection displaying as a co-factor even though the co-relation between HCV transmission and sexual activity is arguable.[25,26,27]

There was a notably increased possibility of sero-positivity of HCV in male homosexuals who were HIV positive when compared to HIV negative men.[28]

The elevation of levels of HIV-RNA has also been related to the existence of HCV

RNA. Indeed for every unit increase in level of log HIV RNA there is 86% increased possibility of HIV RNA test positivity.[29]

Some speculate that change in the immune system rather than sexual transmission route is responsible this association sometimes detected between HIV and HCV transmission.[30,31]

The other hypothesis is that immune suppression caused by HIV aids transmission of HCV. Subjects who were at high risk of transmission of HCV positive it was found that 100% of them were positive for HCV in which CD4 count was less than 100/cu.mm and 66.6% in whom CD4 count was greater than 100/cumm.[29]

With the Co-infection of HCV and HIV there is greater possibility of mother to child transmission of any of these two viruses.

There is a definite bad effect on the response to anti-HCV treatment among HCV/HIV patients with increased viral loads of HCV. The possibility of spontaneous clearing of HCV viruses is among co-infected patients.[32]

The disease of the liver progress more quickly to liver cirrhosis, hepatocellular failure and carcinoma in HCV/HIV co-infected patients.[33]

In co-infected patients a study showed that there is an association between the reduction of viral levels of HIV with a fall in deaths due to liver diseases.[34]

It can be conceived that the chance of individuals infected by HIV surviving rises with effective viral suppression treatment and proper prophylactic treatment to opportunistic diseases, which further results in increased prevalence of diseases and deaths related to HCV.[35]

One of the main causes for mortality among individuals infected by HIV is disease of the liver related to HCV in several developed countries.[36]

INFLUENCE OF HCV ON HIV

The viral levels of HIV are probably not directly affected by HCV. Nevertheless there is a clinical showcase of worse state of deficiency of the immune system than that actually exists which may be due to the lower peripheral CD4 levels among patients in whom there is progression of the disease of the liver, portal hypertension and splenomegaly.[37]

According to the results given by the large cohorts there is a conflict in concern to the risk of advancement of individuals infected by HIV to AIDS in the existence or non-existence of the co-infection. The individuals who were infected by both HIV and HCV progressed quickly to AIDS than the HIV positive individuals who were not co-infected by HCV according to the Swiss cohort study. It was also detected in this that after the institution of ART in an effective manner the rise in CD4 levels was more in individuals not infected by HCV than the individuals infected by HCV showing that the recovery of the immune system in the HIV positive patients is hindered by HCV infection.[38]

INFLUENCE OF HCV ON ART

The treatment of HCV should be started early in HIV positive individuals as the natural history of HIV disease is affected by HCV may affect the natural history of HIV disease.

There is a notable amount of morbidity and mortality due to the multiple interruptions led by the hepatotoxicity associated with ART.[39]

It is evident in a number of studies that the patients with co-infected by HCV and HIV are more prone for ART induced hepatotoxicity, especially the patients receiving protease inhibitors.[39]

There is increased rise in the levels of transaminase in individuals infected by HCV as evident in many cohort studies.[39]

Impact of ART on HCV infection- Many studies have shown that there is rise in the viral levels of HCV after starting ART.[40]

The viral loads become normal within six months of starting ART suggesting that the rise in the viral levels is temporary.[41]

There is an association between the rise in the levels of HCV RNA and rise in the levels of AST and ALT. The reason for rise in the levels of HCV RNA in connection with the recovery of the immune system associated with ART is not clearly understood. There is rise in the levels of ALT in correlation with the fall in HBV DNA levels due to the immune mediated clearance of virus indicating that this may be true in case of HBV co-infection. Nevertheless a change in immune mediated clearance of HCV or the elevated replication in some reservoirs other than liver may be reflected by the paradoxical rise in the levels of HCV RNA seen with co-infection by HCV.[42]

Influence of HBV on course of HIV disease

Some studies have demonstrated that there is a rise in the rate of advancement of HIV to AIDS in the patients with markers of infection by HBV.

There is no variation in the advancement of HIV or survival according to other studies.

Hence the available data showing the influence of HBV on the HIV infection's course is conflicting.[44,]

Influence of HIV on course of HBV disease

There is lesser occurrence of icteric illness showing that the existence of HIV modifies acute HBV's course. The carriage rate of HBV is 25% among individuals infected by HIV while it is only 5% among individuals not infected by HIV.[45]

It is indicated that HIV has an impact on the replication of HBV markers in chronic HBV infection. There is a tendency in favour of a high rise in the viral load of HBV DNA and also the reduced clearance rate of HBV DNA and Hepatitis e antigen.[46]

There is decrease in the severity of disease of the liver which may lead to decrease in the levels of serum transaminases because of the suppression of immunity caused by HIV.[47]

Nevertheless there is also the possibility of association of suppression of immunity with HBV infection reactivation in the patients in whom there is loss of detectable HbeAg or HbsAg.[48]

Even though Anti HBs loss and symptomatic reactivation is not common in the patients infected by HIV, there is frequent reinfection and asymptomatic reactivation in HIV positive individuals who develop AIDS resulting in very high HbsAg prevalence.[49]

There is an association between the sero-positivity of HIV and significant reduction in ALT levels, increased levels of serum HBV DNA, fall in serum DNA and HbeAg clearance rate, reduced liver injury, and more anti-HBs loss. There is increased evidence of cirrhosis in patient's co-infected with HIV and HBV.

Influence of HAART on HBV disease

Patients infected by HBV may be affected by HAART. As in chronic hepatitis patients there is increase in the levels of transaminases and hepatotoxicity directly caused by almost all antiretroviral drugs, the reconstitution of the immune system caused by HAART may also lead to the increase in the levels of transaminases initially.

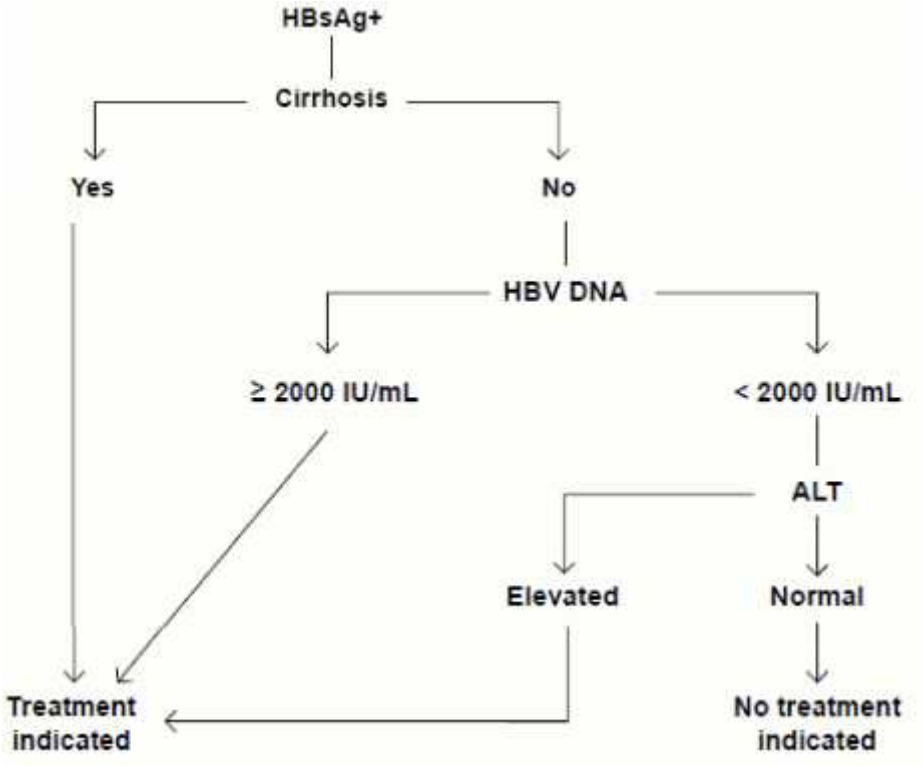
Influence of HBV on HAART

The risk of development of hepatotoxicity increases after starting ART in patients with co-infection of HBV and HIV than in HIV infected patients.61 Therefore it is advocated to start HBV treatment as soon as possible.

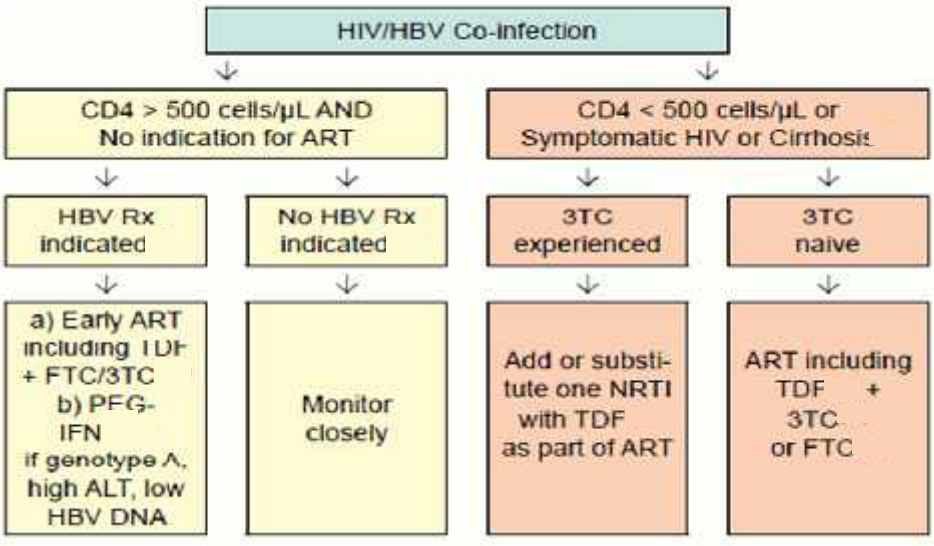
Treatment of co-infection of HIV and HBV

Choice of ART	ARVs with anti HBV activity such as 3TC (or FTC) and TDF should be included in the first-line ART regimen for HIV-infected patients who are HBsAg-positive (and HBeAg-positive, if known)
Preferred first line ART	TDF + TC + EFV TDF may be replaced by AZT in any regimen in case of toxicity or other contraindications

Assessment of Indications for HBV treatment in Patients with co-infection of HIV and HBV Co-infection-*EACS 2013*[50]



Chronic HBV Treatment in Patients with Co-infection of HIV and HBV *EACS 2013*[50]



Treatment for HBV is considered as a priority in patients with HBV/HIV co-infection due to the following two important reasons-

1. The rapid progression of the disease of the liver in HBV/HIV co-infected patients and may result in fatal complications of the disease of the liver like cirrhosis and hepatocellular carcinoma at younger ages.

2. The risk of development of hepatotoxicity increases after starting ART in patients infected by HIV also co-infected by HBV than in patients with lone HIV infection.

As HIV infection may increase the rate of progression of disease of disease of the liver, chronic hepatitis B treatment is usually advocated in persons with:

1. Inflammatory signs of liver (increased levels of ALT)
2. Fibrosis of the liver (high elastography, or liver biopsy Metavir 2)
3. Levels of HBV DNA more than 2000 IU/ml).

Treatment of HBV Patients without ART indication

1. Only the substances without activity of HIV should be used like Telbivudine, Peg Ifn, and Adefovir).
2. 3TC, FTC and Tenofovir should be avoided.
3. Entecavir should also be avoided (induction of HIV reverse transcriptase mutation M184V is possible).
4. Most of the experts recommended Telbivudine preferably than Adefovir because of more antiviral efficacy but always the possibility of early initiation of HAART which includes Tenofovir + FTC or 3TC should be checked (it is preferred - EACS 2011).

Hepatitis B treatment in HIV/HBV co-infected patients without ART indication

Treatment with **pegylated interferon** should be considered in special circumstances:

1. HIV treatment is not needed (high number of CD4 cells)

2. HBeAg positive

3. HBsAg genotype A

4. Elevated ALT

5. Low level of HBV DNA

(Poor data and no encouraging results).

WHO-2013 Recommendations

There should be initiation of ART in all HIV positive individuals without consideration of CD4 levels or WHO clinical stage in the situations mentioned below:

1. HIV positive individuals with active tuberculosis *(low quality evidence, strong recommendation).*

2. Evidence of severe chronic liver disease in HBV/HIV co-infected patients *(low-quality evidence, strong recommendation).*

3. ART to be offered to sero-discordant couples to decrease transmission of HIV to partners who are not infected.*(high-quality evidence, strong recommendation).*

Treatment of HCV in Persons with HCV/HIV Co-infection - EACS 2013

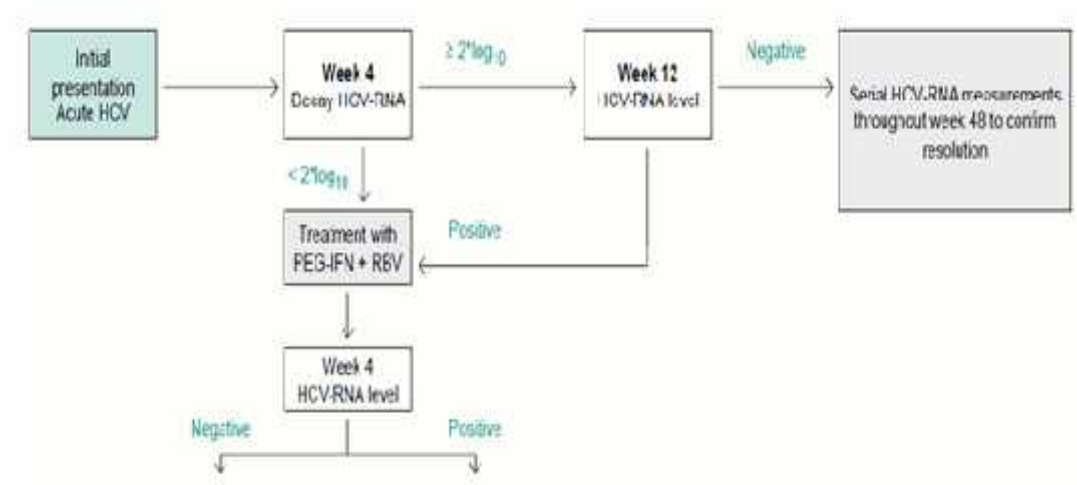
Treatment of ACUTE HCV infection in HIV/HCV co-infected patients

Acute HCV treatment indications is same in both HIV infected and uninfected patients. The initiation of treatment is not done usually till 12 weeks following the infection

by HCV as there might be clearance of HCV infection spontaneously in lone HCV infected individuals by 30-50% and in HIV co-infected patients by 15-20%.

HCV RNA not detectable after 6 months of stopping the treatment also called as [Sustained virologic response (SVR)] is seen in 60-80% of acute HCV patients.145 Ro-bust studies have not taken place on pegylated interferon without ribavirin in acute HCV patients co-infected by HIV.

Algorithm for treatment of Acute HCV in Persons with co-infection of HCV and HIV EACS 2013[50]



Stop after 24 weeks see above text

Treatment of ACUTE HCV infection in HIV/HCV co-infected patients

Combined usage of weight-based ribavirin and pegylated interferon for 48 weeks have been done by several experts, while the use of ribavirin is not advised in acute HCV patients not infected by HIV in whom pegylated interferon usage is advocated for just 24 weeks.

According to the studies which have taken place recently it has been suggested that a quick virological response achieved by patients is defined by viral load of HCV not detectable by 4th week of treatment, treatment reduced to a short course of 24 weeks may be appropriate.

Treatment of CHRONIC HCV infection in HIV/HCV co-infected patients

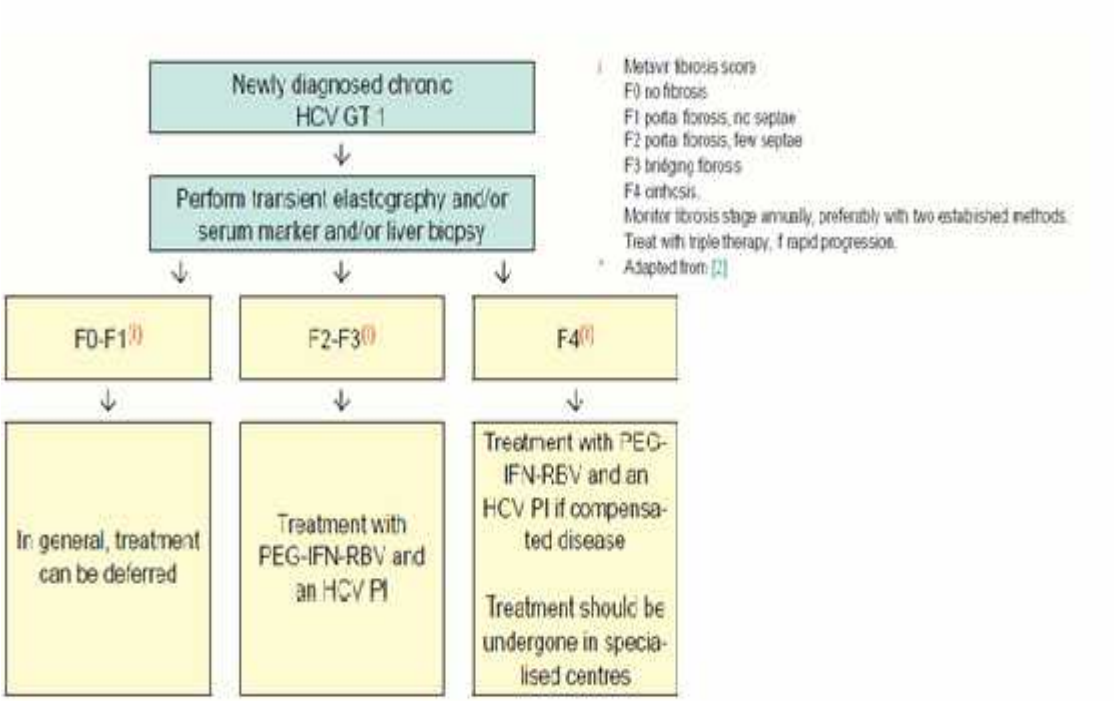
It is more difficult and less algorithmic to treat HIV infected individuals with chronic HCV infection than treating acute HCV patients.

The usual approach is to weigh the benefits of therapy with the pegylated interferon and ribavirin (PEG/R) associated morbidity, while taking into consideration the possibility of every patient responding to the treatment. Given response to treatment of HCV is good in individuals with well controlled HIV, HIV treatment is usually started before starting HCV treatment.

However, in cases where ART related toxicity precludes continuation of ART.

HCV may need to be treated first, allowing for improved tolerability of ART.

Management of Persons with Newly Diagnosed HCV GT 1/ HIV Co-infection- EACS 2013[50]



MATERIALS AND METHODS:

1. SOURCE OF DATA:

The information for the study was collected from HIV positive patients admitted to BLDEU'S Shri B.M Patil Medical college Hospital and Research center, Vijayapur from November 2015 to June 2017.

2. METHOD OF COLLECTION OF DATA:

Information was collected through prepared proforma from each patient. All patients were interviewed as per the prepared proforma and then complete clinical examination was done.

Inclusion Criteria:

- HIV infection diagnosed as per NACO guidelines.
    - **Diagnosed by 3 spot tests:**
        1. **Coombs AIDS test**
        2. **Triline test**
        3. **Qualpro test**

Exclusion Criteria:

- HIV negative patents.
- Patients not willing to take part in the study.

3. TYPE OF STUDY

Cross-sectional study

4. SAMPLE SIZE:

With the incidence of HBV in HIV positive cases 2.25%[3] and at 95% confidence interval and +/-2 margin of error the sample size worked was 211 using

$n = (Z_a^2 * p * q) / d^2$

Z_a = Z value a=99%

p = Incidence rate of HBV

q = 100-p

d = Margin of error [99%=1.46]

Hence to find incidence of HBV and HCV in HIV positive cases 211 cases were included.

Statistical Analysis

1. Diagrams
2. Mean +/- SD
3. Chi-square test (if necessary)
4. Correlation and regression analysis.

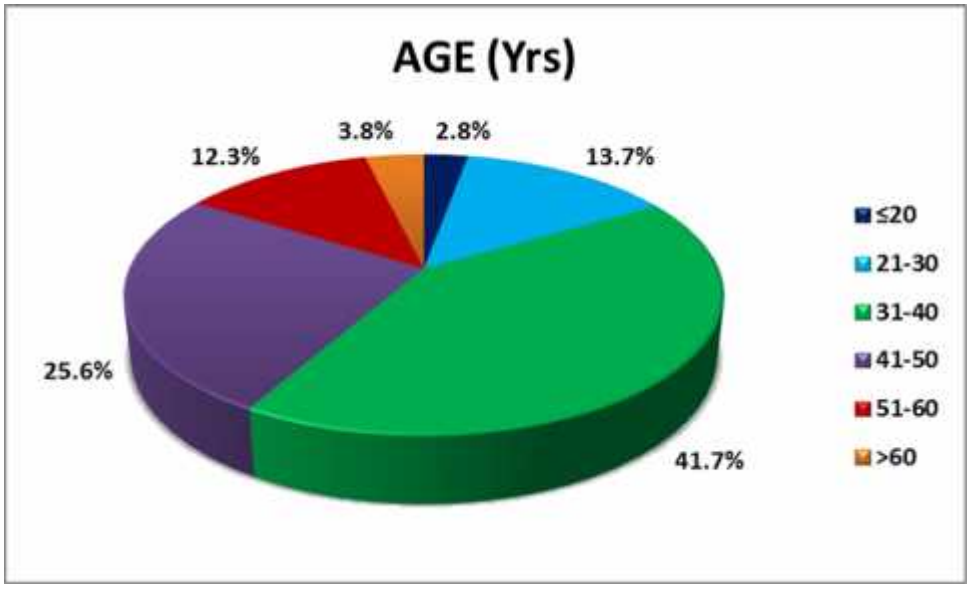
RESULTS:

After detailed history, clinical examination, HIV, HbsAg, Anti HCV antibody testing, the results were obtained and tabulated in following table.

TABLE-1: DISTRIBUTION OF CASES ACCORDING TO AGE

AGE	N	%
≤20	6	2.8
21-30	29	13.7
31-40	88	41.7
41-50	54	25.6
51-60	26	12.3
>60	8	3.8
Total	211	100

FIGURE-1: DISTRIBUTION OF CASES ACCORDING TO AGE

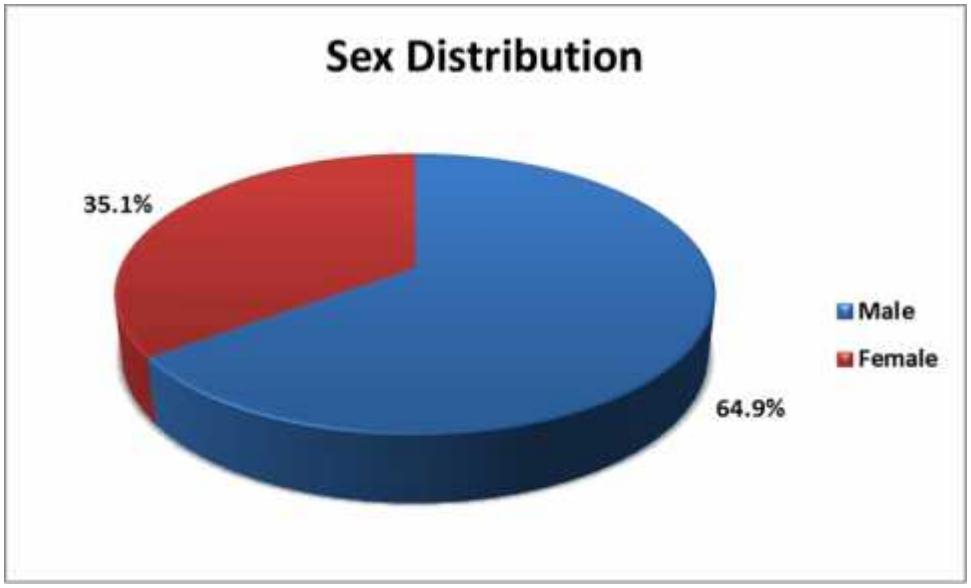


Of the total 211 cases, maximum number of cases were in the age group 31-40 years i.e. 88 (41.7%).

TABLE-2: DISTRIBUTION OF CASES ACCORDING TO SEX

SEX	N	%
Male	137	64.9
Female	74	35.1
Total	211	100

FIGURE-2: DISTRIBUTION OF CASES ACCORDING TO SEX



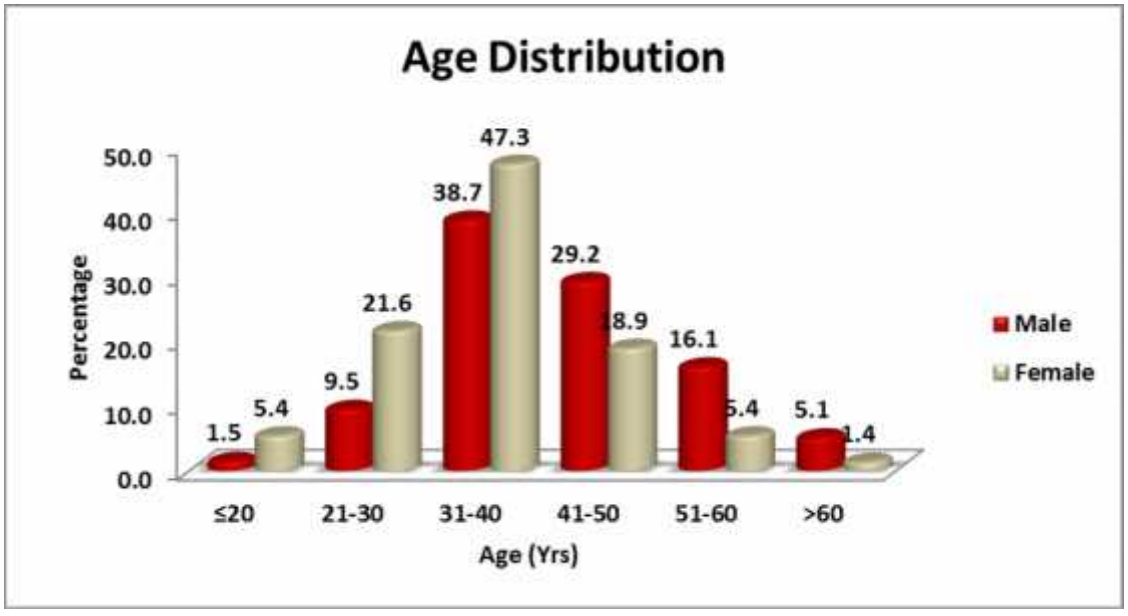
Out of 211 cases, 137 (64.9%) were males and 74 (35.1%) were females.

TABLE-3: ASSOCIATION OF AGE AND SEX

AGE (Yrs)	Male		Female		p value
	N	%	N	%	
≤20	2	1.5	4	5.4	
21-30	13	9.5	16	21.6	
31-40	53	38.7	35	47.3	
41-50	40	29.2	14	18.9	0.005*
51-60	22	16.1	4	5.4	
>60	7	5.1	1	1.4	
Total	137	100.0	74	100.0	

Note: *means significant at 5% level of significance (p<0.05)

FIGURE-3: ASSOCIATION OF AGE AND SEX

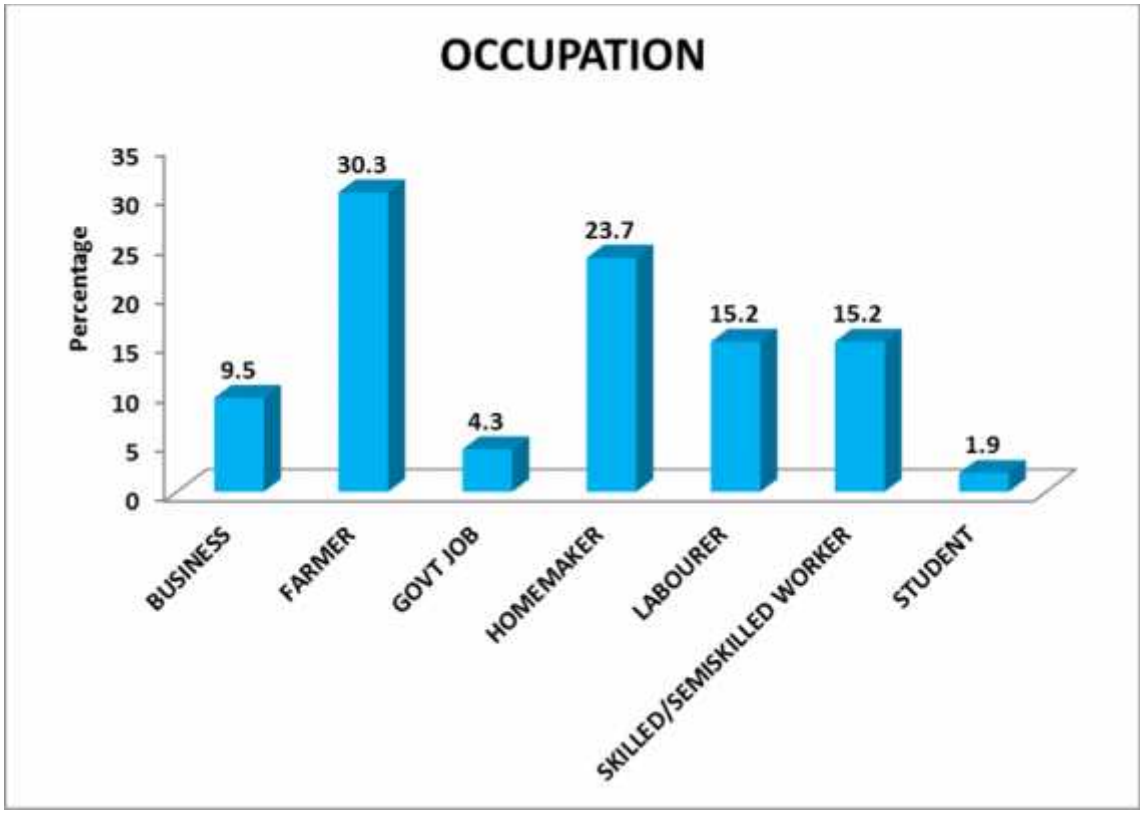


In both sexes maximum numbers of cases were present in 31-40 years age group. It was 53 (38.7%) for males and 35(47.3%) for females.

TABLE-4: DISTRIBUTION OF CASES ACCORDING TO OCCUPATION

OCCUPATION	N	%
BUSINESS	20	9.5
FARMER	64	30.3
GOVT JOB	9	4.3
HOMEMAKER	50	23.7
LABOURER	32	15.2
SKILLED/SEMISKILLED WORKER	32	15.2
STUDENT	4	1.9
Total	211	100

FIGURE-4: DISTRIBUTION OF CASES ACCORDING TO OCCUPATION

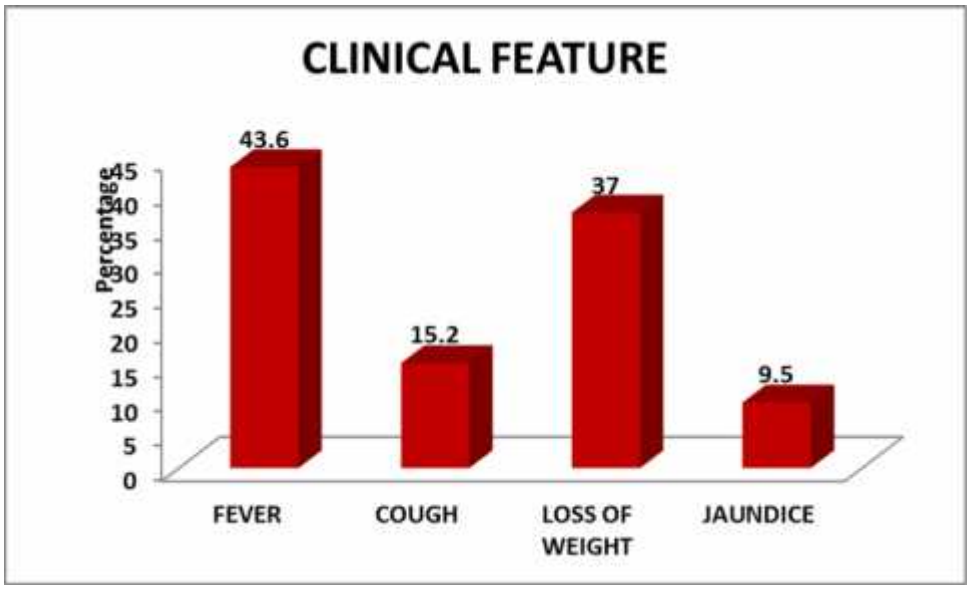


Amongst 211 patients, maximum number of patients were farmers, that is 64 (30.3%).

TABLE-5: DISTRIBUTION OF CASES ACCORDING TO CLINICAL FEATURE

CLINICAL FEATURE	N	%
FEVER	92	43.6
COUGH	32	15.2
LOSS OF WEIGHT	78	37
JAUNDICE	20	9.5

FIGURE-5: DISTRIBUTION OF CASES ACCORDING TO CLINICAL FEATURES

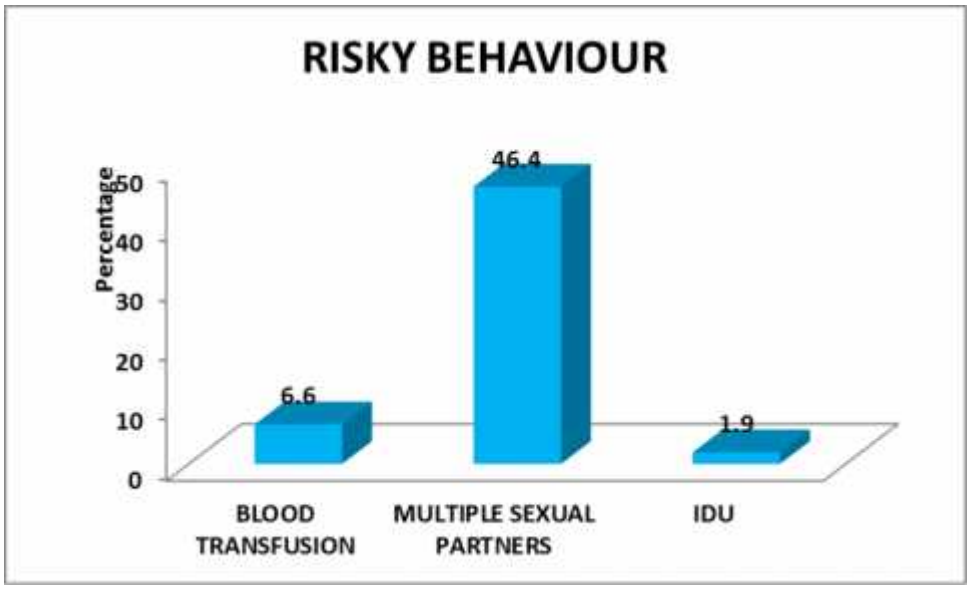


Out of 211 patients, 92 (43.6%) patients had fever, 32 (15.2%) patients had cough, 78 (37%) patients had loss of weight and 20 (9.5%) patients had jaundice.

TABLE-6: DISTRIBUTION OF CASES ACCORDING TO RISKY BEHAVIOUR

RISKY BEHAVIOUR	**N**	**%**
BLOOD TRANSFUSION	14	6.6
MULTIPLE SEXUAL PARTNERS	98	46.4
IDU	4	1.9

FIGURE-6: DISTRIBUTION OF CASES ACCORDING TO RISKY BEHAVIOUR

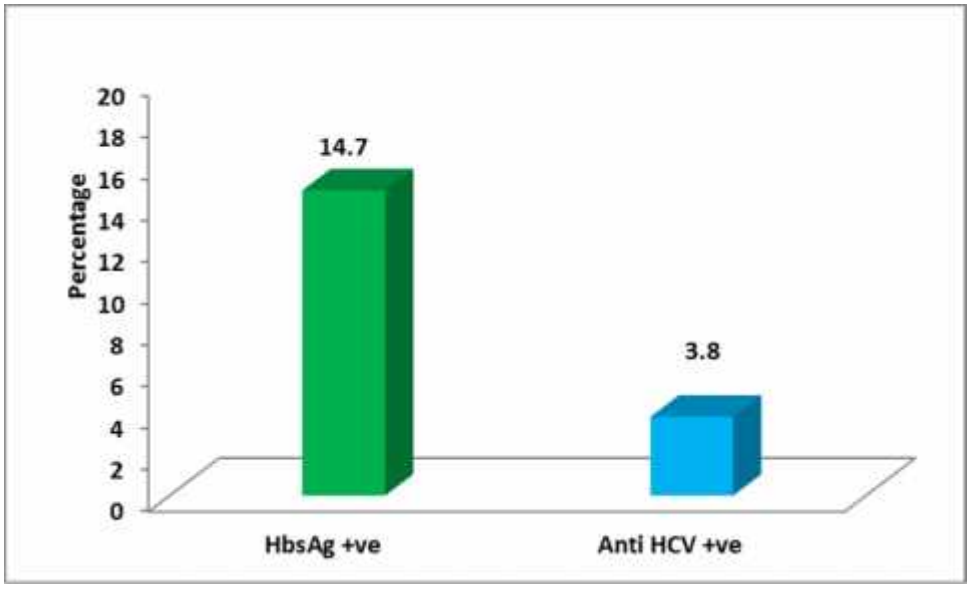


98 (46.4%) patients had multiple sexual partners, 14 (6.6%) of them had blood or blood product transfusion as a risk factor. 4 (1.9%) were intravenous drug users.

TABLE-7: DISTRIBUTION OF CASES ACCORDING TO HbsAg & Anti HCV POSITIVITY

Positivity	N	%
HbsAg +ve	31	14.7
Anti HCV +ve	8	3.8

FIGURE-7: DISTRIBUTION OF CASES ACCORDING TO HbsAg & Anti HCV POSITIVITY



Out of 211 HIV positive patients 31(14.7%) were positive to HbsAg and 8 (3.8%) were positive for Anti HCV.

TABLE-8: DISTRIBUTION OF AGE ACCORDING TO HbsAg POSITIVITY

AGE (Yrs)	HbsAg +ve		HbsAg -ve		p value
	N	%	N	%	
≤20	1	3.2	5	2.8	
21-30	2	6.5	27	15.0	
31-40	11	35.5	77	42.8	
41-50	8	25.8	46	25.6	0.209
51-60	8	25.8	18	10.0	
>60	1	3.2	7	3.9	
Total	31	100.0	180	100.0	
Mean & SD	**43.4**	**10.4**	**40.0**	**10.5**	**0.097**

FIGURE-8(a): DISTRIBUTION OF AGE ACCORDING TO HbsAg POSITIVITY

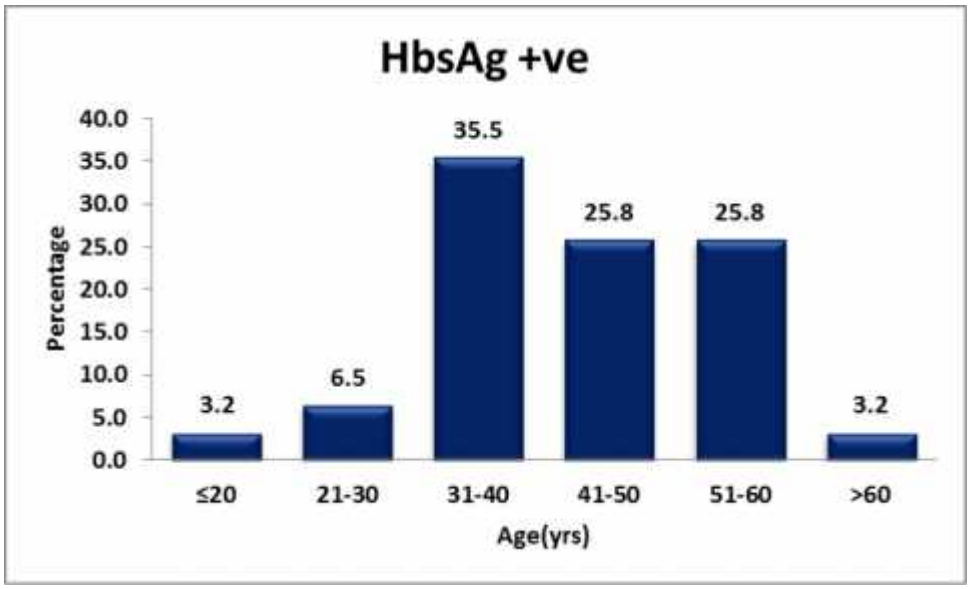
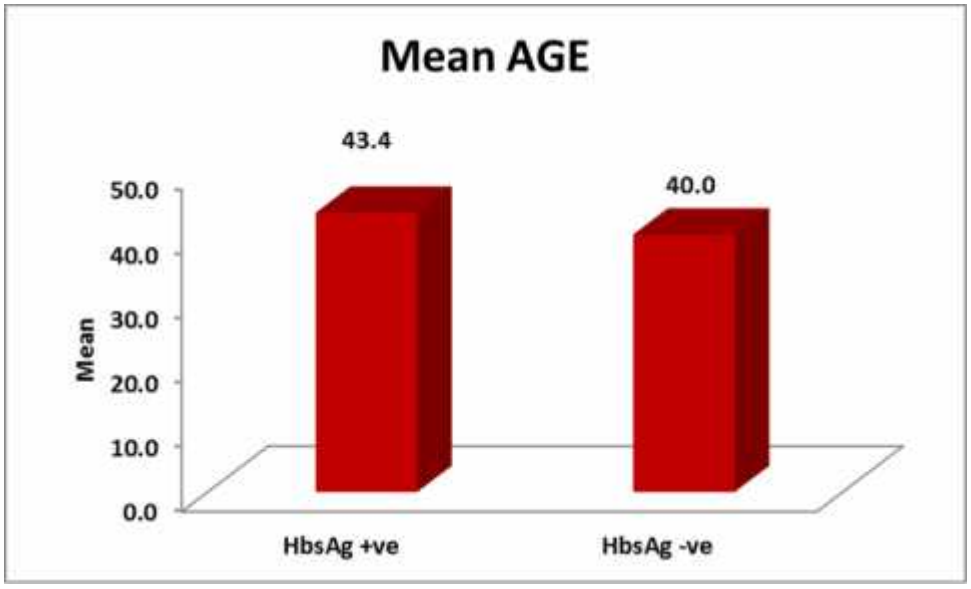


FIGURE-8(b): MEAN AGE ACCORDING TO HbsAg POSITIVITY



Among the HIV positive patients, HbsAg was seen more in age group 31-40 that is 11 (35.5%) patients with mean 43.4 years, showing co-infection is more common in sexually active age group.

TABLE-9: DISTRIBUTION OF AGE ACCORDING TO Anti HCV POSITIVITY

AGE (Yrs)	Anti HCV +ve		Anti HCV -ve		p value
	N	%	N	%	
≤20	0	0.0	6	3.0	
21-30	2	25.0	27	13.3	
31-40	4	50.0	84	41.4	
41-50	1	12.5	53	26.1	0.847
51-60	1	12.5	25	12.3	
>60	0	0.0	8	3.9	
Total	8	100.0	203	100.0	
Mean & SD	**39.0**	**10.0**	**40.5**	**10.6**	**0.685**

FIGURE-9(a): DISTRIBUTION OF AGE ACCORDING TO Anti HCV POSITIVITY

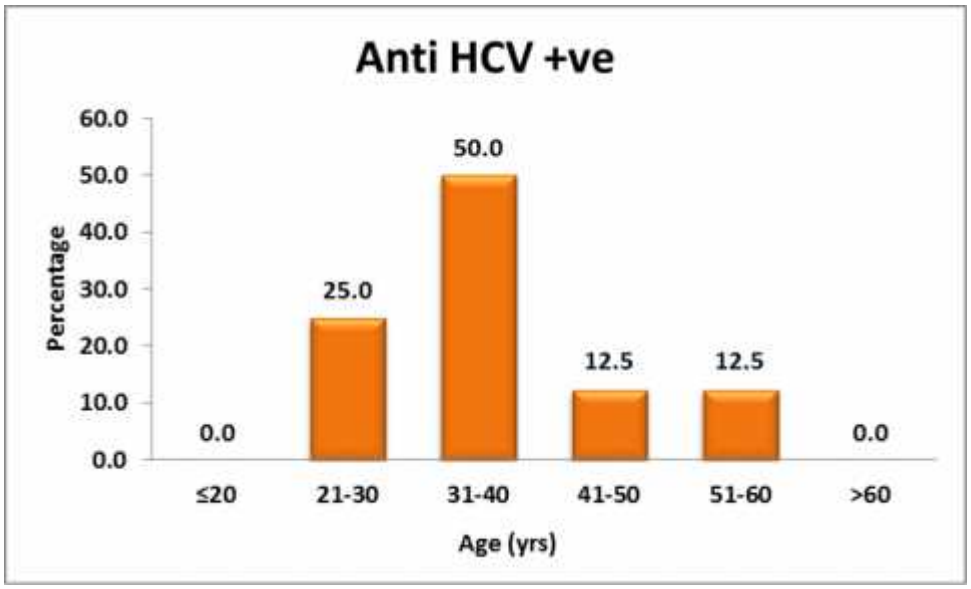
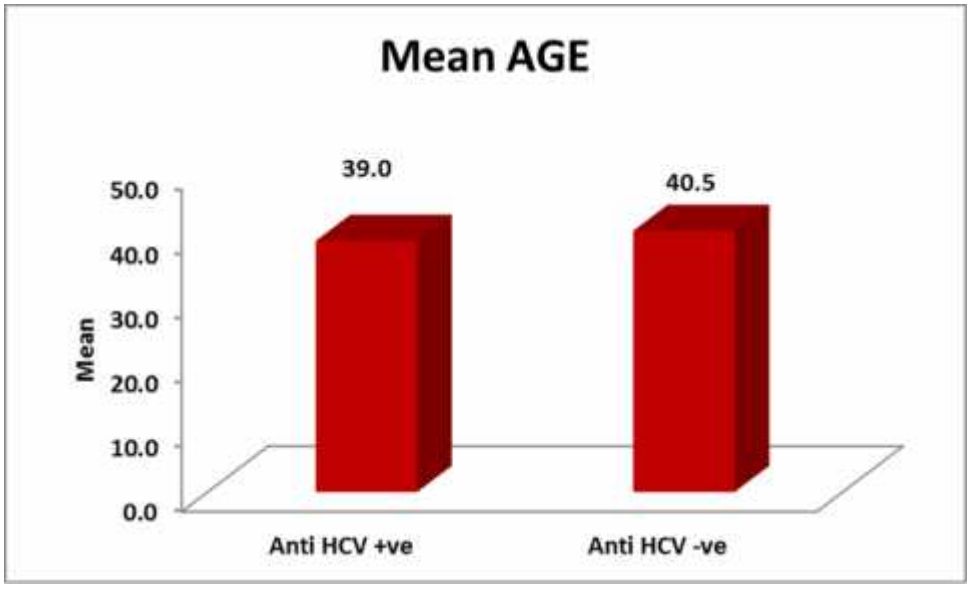


FIGURE-9(b): MEAN AGE ACCORDING TO Anti HCV POSITIVITY

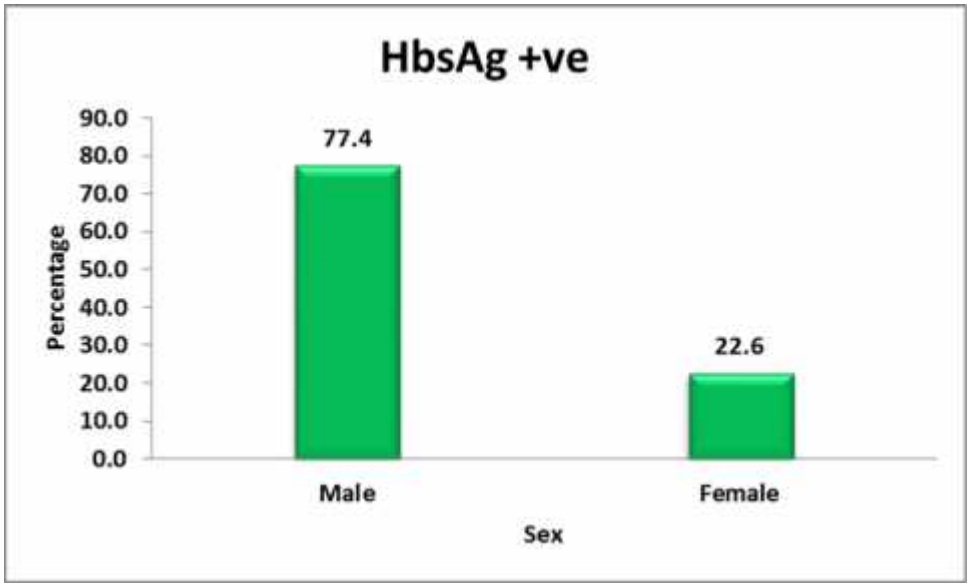


Among the HIV positive patients Anti HCV was seen more in age group 31-40 that is 4 (50%) patients with mean 39 years, showing co-infection is more common in sexually active age group.

TABLE-10: DISTRIBUTION OF SEX ACCORDING TO HbsAg POSITIVITY

SEX	HbsAg +ve		HbsAg -ve		p value
	N	%	N	%	
Male	24	77.4	113	62.8	0.115
Female	7	22.6	67	37.2	
Total	31	100.0	180	100.0	

FIGURE-10: DISTRIBUTION OF SEX ACCORDING TO HbsAg POSITIVITY

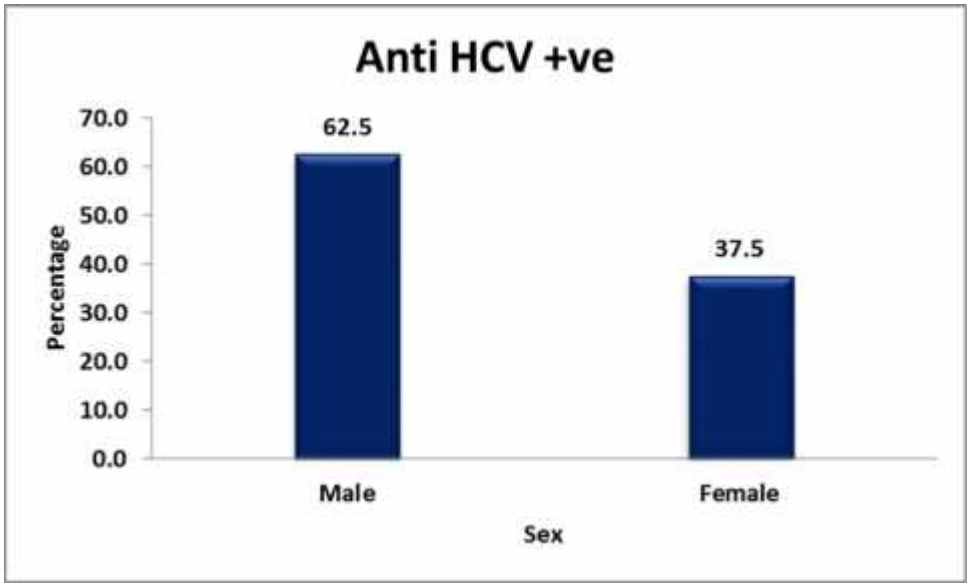


Out of 31 HbsAg positive HIV patients 24 (77.4%) were male and 7 (22.6%) were female.

TABLE-11: DISTRIBUTION OF SEX ACCORDING TO Anti HCV POSITIVITY

SEX	Anti HCV +ve		Anti HCV -ve		p value
	N	%	N	%	
Male	5	62.5	132	65.0	0.883
Female	3	37.5	71	35.0	
Total	8	100.0	203	100.0	

FIGURE-11: DISTRIBUTION OF SEX ACCORDING TO Anti HCV POSITIVITY



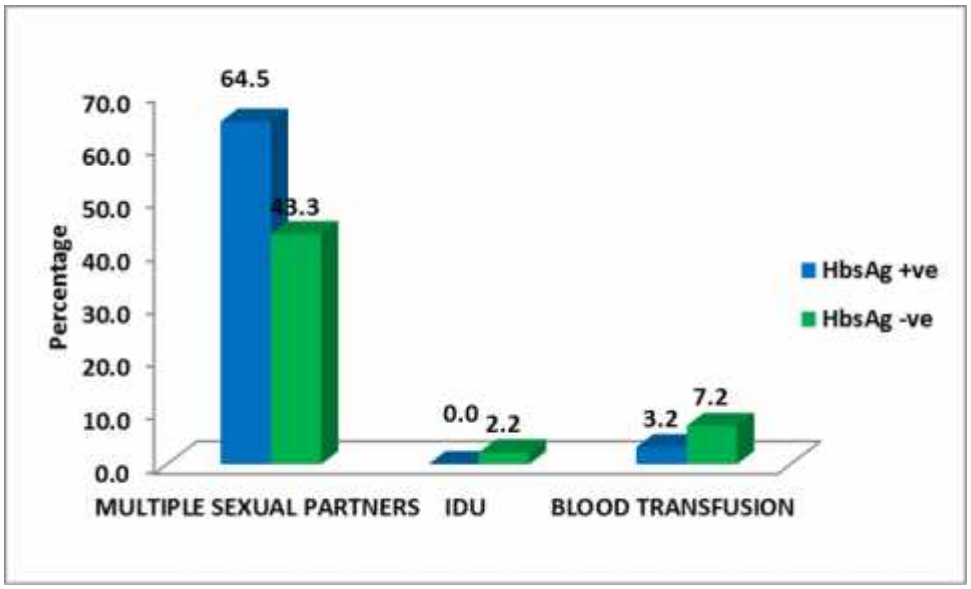
5 (62.5%) males and 3 (37.5%) females were Anti-HCV positive HIV patients.

TABLE-12: HbsAg POSITIVITY ACCORDING TO RISKY BEHAVIOUR

Variables	HbsAg +ve		HbsAg -ve		p value
	N	%	N	%	
MULTIPLE SEXUAL PARTNERS	20	64.5	78	43.3	0.029*
IDU	0	0.0	4	2.2	0.402
BLOOD TRANSFUSION	1	3.2	13	7.2	0.409

Note: *means significant at 5% level of significance ($p<0.05$)

FIGURE-12: HbsAg POSITIVITY ACCORDING TO RISKY BEHAVIOUR



20 (64.5%) of the 31 Hbs Ag positive patients had multiple sexual partners, 1 (3.2%) Hbs Ag positive patient had transfusion of blood and blood products as risk factor.

TABLE-13: HbsAg POSITIVITY ACCORDING TO PARAMETERS

PARAMETERS	HbsAg +ve		HbsAg -ve		p value
	Mean	SD	Mean	SD	
CD4 COUNT	154.3	40.5	314.8	129.8	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

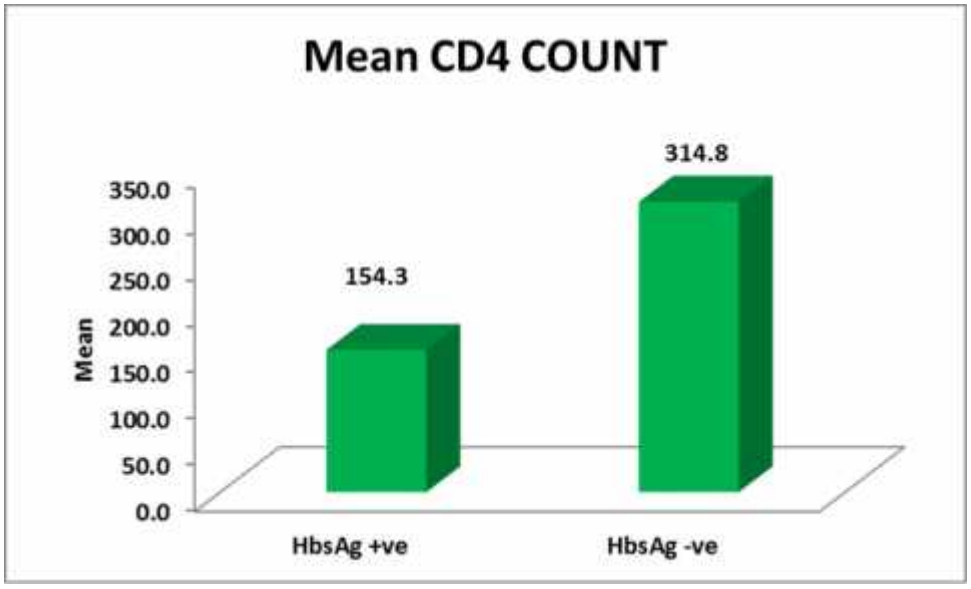
PARAMETERS	HbsAg +ve		HbsAg -ve		p value
	Mean	SD	Mean	SD	
S.BIL	1.0	1.2	0.9	1.1	0.456

Note: *means significant at 5% level of significance (p<0.05)

PARAMETERS	HbsAg +ve		HbsAg -ve		p value
	Mean	SD	Mean	SD	
SGOT	229.4	354.2	46.3	56.5	<0.001*
SGPT	168.4	180.4	37.5	56.8	<0.001*
ALP	182.4	69.1	134.9	104.1	0.015*

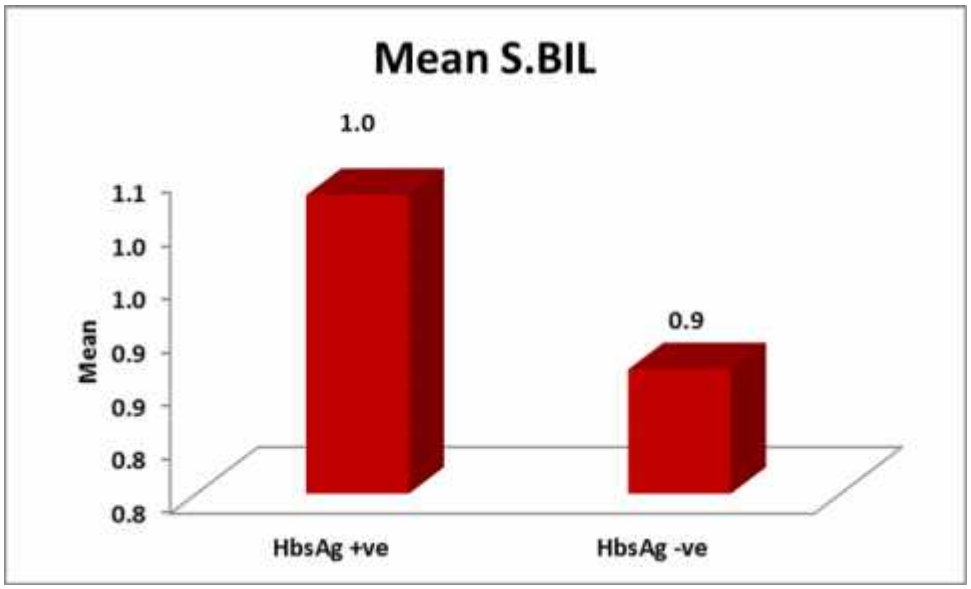
Note: *means significant at 5% level of significance (p<0.05)

FIGURE-13(a): HbsAg POSITIVITY ACCORDING TO PARAMETERS



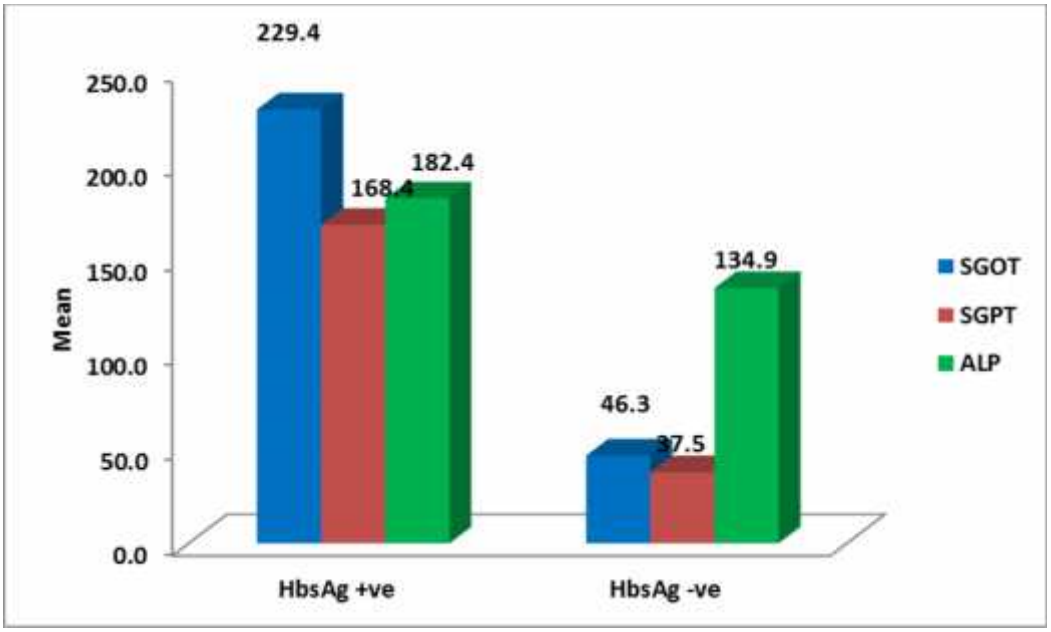
Mean CD4 count was 154.3 among Hbs Ag positive HIV patients which is significantly less as compared to mean CD4 count of 314.8 among Hbs Ag negative HIV patients and it is statistically significant.

FIGURE-13(b):



Mean Serum bilirubin level is 1mg/dl among Hbs Ag positive HIV patients which is not significantly increased as compared to mean serum bilirubin level of 0.9 among Hbs Ag negative patients and it is not statistically significant.

FIGURE-13(c):

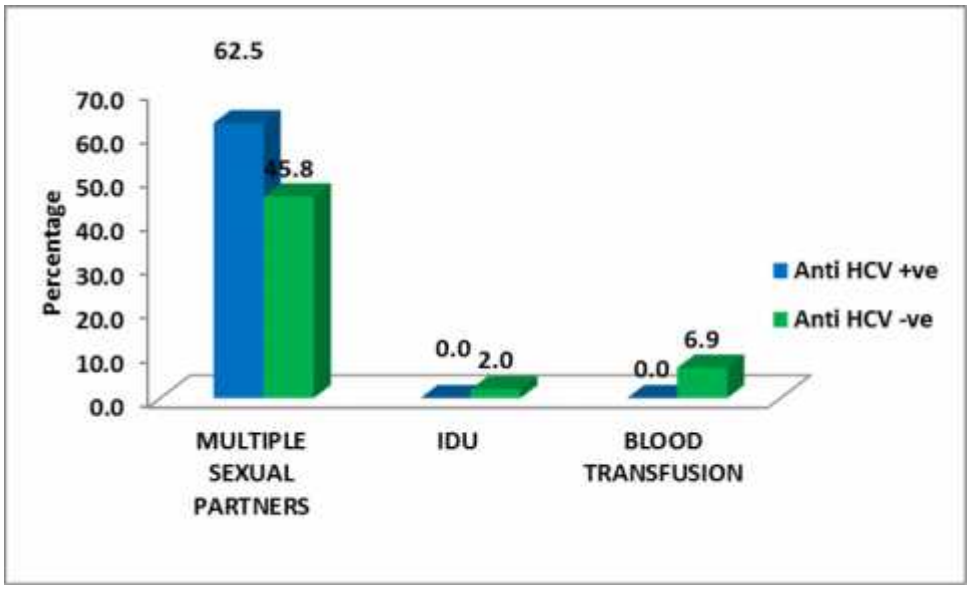


Mean SGOT is 229.4, mean SGPT is 168.4 and mean ALP is 182.4 in Hbs Ag positive HIV patients which is significantly increased as compared to that of Hbs Ag negative HIV positive patients and it is statistically significant.

TABLE-14: Anti HCV POSITIVITY ACCORDING TO RISKY BEHAVIOUR

Variables	Anti HCV +ve		Anti HCV -ve		p value
	N	%	N	%	
MULTIPLE SEXUAL PARTNERS	5	62.5	93	45.8	0.353
IDU	0	0.0	4	2.0	0.689
BLOOD TRANSFUSION	0	0.0	14	6.9	0.442

FIGURE-14: Anti HCV POSITIVITY ACCORDING TO RISKY BEHAVIOUR



Among the 8 Anti HCV positive patients, 5 (62.5%) had multiple sexual partners.

TABLE-15: Anti HCV POSITIVITY ACCORDING TO PARAMETERS

PARAMETERS	Anti HCV +ve		Anti HCV -ve		p value
	Mean	SD	Mean	SD	
CD4 COUNT	135.5	18.8	297.3	132.4	0.001*

Note: *means significant at 5% level of significance (p<0.05)

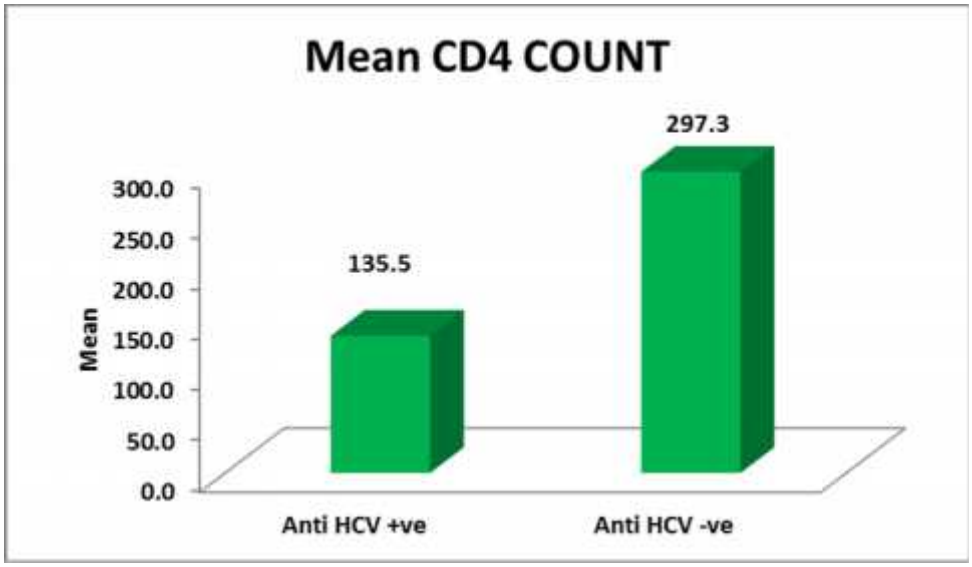
PARAMETERS	Anti HCV +ve		Anti HCV -ve		p value
	Mean	SD	Mean	SD	
S.BIL	2.4	3.3	0.8	0.9	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

PARAMETERS	Anti HCV +ve		Anti HCV -ve		p value
	Mean	SD	Mean	SD	
SGOT	467.6	635.3	57.7	73.8	<0.001*
SGPT	239.8	301.3	49.5	73.6	<0.001*
ALP	278.3	225.6	136.5	89.9	<0.001*

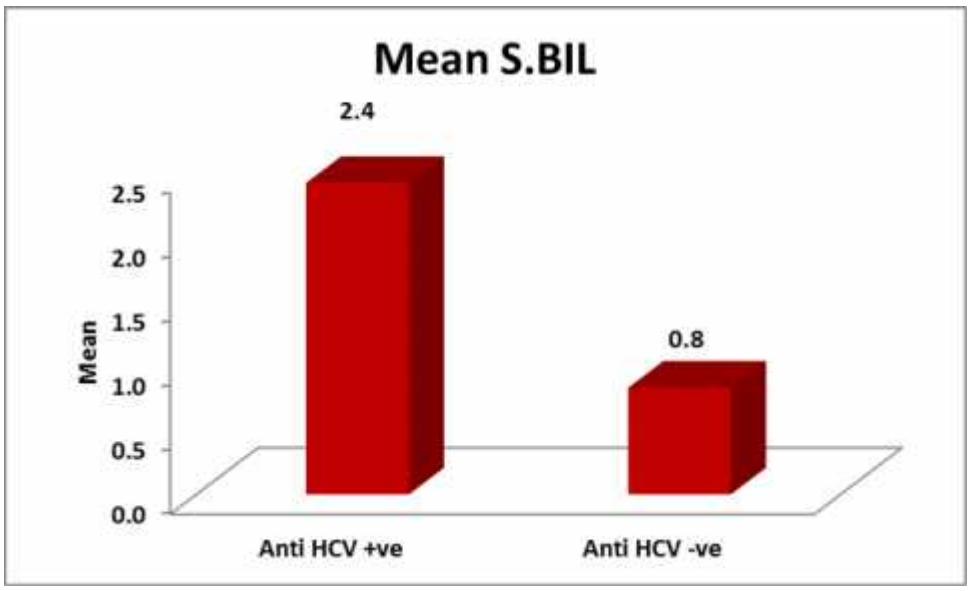
Note: *means significant at 5% level of significance (p<0.05)

FIGURE-15(a): Anti HCV POSITIVITY ACCORDING TO PARAMETERS



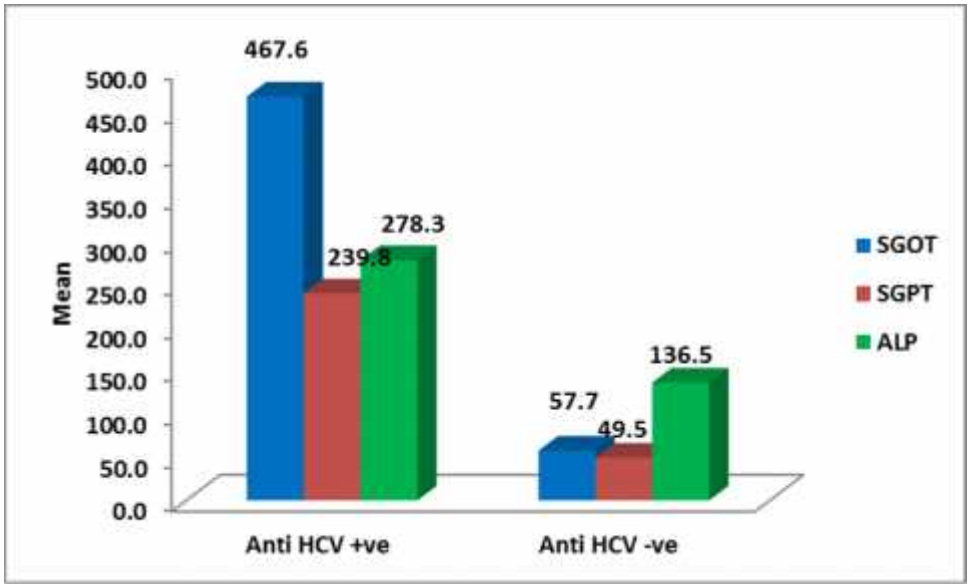
Mean CD4 count was 135.5 in Anti HCV positive HIV patients which is significantly less than that of Anti HCV negative HIV positive patients and it is statistically significant.

FIGURE-15(b):



Mean Serum bilirubin among Anti HCV positive HIV patients is 2.4 which is only 0.8 in Anti HCV negative patients. It is statistically significant.

FIGURE-15(c):

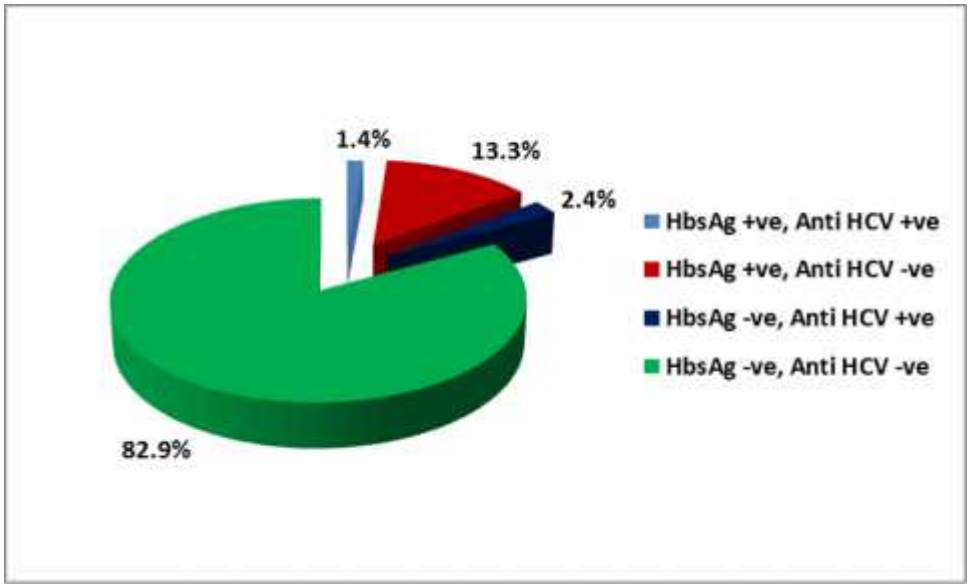


Mean SGOT is 467.6, mean SGPT is 239.8 amd mean ALP is 278.3 in HIV and HCV co-infected patients which is significantly increased as compared to that of Anti HCV negative HIV positive patients and it is statistically significant.

Table-16: Relationship between HbsAg and Anti HCV positivity

Positivity	N	%
HbsAg +ve, Anti HCV +ve	3	1.4
HbsAg +ve, Anti HCV -ve	28	13.3
HbsAg -ve, Anti HCV +ve	5	2.4
HbsAg -ve, Anti HCV -ve	175	82.9

Figure-16: Relationship between HbsAg and Anti HCV positivity



Out of 211 HIV positive patients, 28 (13.3%) are Hbs Ag positive, 5 (2.4%) are Anti HCV positive and 3 (1.4%) are both Hbs Ag and Anti HCV positive.

DISCUSSION:

The survival among HIV infected individuals is increasing and they are surviving up to a longer age, due to the advent of HAART, but they are suffering more due to liver diseases caused by hepatitis B and Hepatitis C virus. Co-infection is common because of common mode of transmission and similar risky behavior.

Many studies have been done in the western countries regarding the co-positivity of HIV with HbsAg and HCV but there are very few studies done in India.

Considering the above this study was done at BLDEU's Shri B M Patil medical college hospital and research centre, Vijayapur. This study comprises, confirmed HIV positive individuals who were subjected to HbsAg and Anti HCV antibody testing.

Various studies denotes the HbsAg and anti HCV antibody co-positivity from 5-30 % for HbsAg and 0-20% for Anti HCV.

Table 17: Comparison between other studies and our study

Author	**Sample size**	**HBV**	**HCV**	**HBV+HCV**
Indian studies				
Chandra N, Shantharam V	60	13.2%	8.3%	1 case
Thankiwale SS et al	130	30.4%	7.27%	-
Dhanvijay et al	175	28.0%	-	-
Agarval K et al	100	13%	-	-
Ramanamma et al	140	14.3%	3%	
Sud A et al	57	-	5.3%	-
Shazia M Ehsaan et al	200	3.5%	8%	-
Our study	50	6%	2%	-
Foreign studies				
Salmon-Ceron D and others	822	8%	29%	4%
Somnuek Sungkanuparph MD, et al.	529	8.7%	7.8%	-
Anna Kalinowska-Nowak.et al.	133	5%	9%	66%
Lincoln D, Petoumenos K, Dore GJ	2086	6.3%	9%	-

Agbaji o et al	1044	14.8%	7.6%	-
Saillor f et al	1935	6.9%	42.5%	-
Seme K et al	136	-	16.9%	-
Orkanga et al	232	9%	23%	-
Mustapha SK et al	200	26.5%	-	-
Dimitrakopoulas et al	181	67.5%	13.8%	-
Our study	211	14.7%	3.8%	1.4%

Study conducted by Saroj Hooja et al[51] found that CD4 counts were significantly lower in the HIV/HBV co-infected group as compared to HIV alone.

Naval Chandra et al[52] also showed that CD4 counts were significantly lower in HIV patients co-infected with HBV or HCV. The same is seen in our present study.

Study conducted by Naval Chandra et al[52] found that there was marked elevation of liver enzymes in HIV patients co-infected with HBV or HCV as compared to HIV alone. Similar results were found in our present study.

HBV has considerable potential to activate HIV replication directly. In addition, chronic and persistent activation of the immune system by an ongoing immune response (e.g., an infection with a hepatotropic virus) increases the expression of HIV and may therefore accelerate immunodeficiency and the course of HIV infection.

It is evident from various Indian studies that co-positivity of HbsAg in HIV positive individuals is low if compared to foreign studies.

It is evident from literature and which is also present in our study the maximum number of co-positive patients are in 30-50 age group. Agarval K, Sarin SK of Moulana Azad Medical College found maximum number of cases to be in 30-50 years age group.

In comparison to western studies co-positivity is lower in India. In HIV Atlanta cohort study it was found that Anti HCV was prevalent at rate of 32% among HIV positive individuals.

In western countries among co-infected individuals, the risk behavior was different from the Indian population. There HIV spread is more common with Intra venous drug abuse; this mode of transmission is negligible in India except in metro cities and north eastern states which show higher Anti HCV prevalence than our studies.

Since there is a changing trend of HIV transmission with increase in intravenous drug abuse the co-infection with Hepatitis B and Hepatitis C may increase.

As literature shows that co-infectivity with Hepatitis B and Hepatitis C virus with HIV causes increased damage to Liver resulting in increased mortality and morbidity, it is advisable to test every HIV positive individual with HbsAg and Anti HCV, and if not exposed to infection vaccination should be given to such individuals.

CONCLUSIONS:

The present study concluded that

1. Co-positivity with HBV in HIV positive patients was found to be 14.7%

2. Co-positivity with HCV was found to be 3.8%

3. Age group 31-40 years showed highest co-positivity for HBV

4. Age group 31-40 years showed highest co-positivity for HCV

5. Study showed Male preponderance.

6. Study also shows that highest co-positivity is seen in patients whose CD 4 count was <200.

7. Liver enzymes are markedly elevated in co-infected cases.

8. Study of viral DNA levels could not be done because of non-availability of facilities in our set up; this could have been more sensitive in finding the exact co-positivity of HbsAg and Anti HCV with HIV infection.

SUMMARY:

The main aim of my dissertation was to study Hepatitis B and C Virus co-infection in HIV patients. 211 HIV infected individuals were taken up for the study and were subjected to HbsAg and Anti HCV antibodies, liver enzymes and CD4 count at BLDEU's Shri B M Patil medical college hospital and research centre, Vijayapur for a period of 2 years.

Following observation made:

1. Common age group for HIV positive patients was 31-40 years (41.7%).
2. Males out number females with ratio of 13:7 [64.9% : 35.1%].
3. Multiple sexual partner behavior as a risk factor for HIV accounted for about 46.4%.
4. HbsAg positive in HIV positive individuals were recorded in 31 patients (14.7%).
5. Anti HCV antibodies status in HIV positive individuals were recorded in 8 patients (3.8%).
6. The age group of 31-40 years was the commonest period for HBV and HCV co-infection in HIV individuals.
7. Mean CD4 counts were 154.3 and 135.5 for HbsAg and Anti HCV positive HIV patients respectively.
8. Mean SGOT and SGPT levels were 229.4 and 168.4 respectively in HbsAg positive HIV patients.
9. Mean SGOT and SGPT levels were 467.6 and 239.8 respectively in Anti HCV positive HIV patients.
10. Mean Serum bilirubin was 1 and 2.4 in HbsAg positive and Anti HCV positive HIV patients respectively.

This study shows that co-positivity of HBV and HCV is less prevalent than that in western countries, probably due to different risk behaviors and different traditional and cultural practices.

LIMITATIONS OF THE STUDY:

1. We could not measure the viral load

2. Follow up of the patients could not be done for the disease progression.

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ANNEXURES

B.L.D.E.UNIVERSITY'S

SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103

INSTITUTIONAL ETHICAL COMMITTEE

NO/58/2015
20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 17-11-2015 *at* 03pm *scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.*

Title "A Clinical study of co-infection of hepatitis B & hepatitis C virus in HIV infected patients in correlation with CD_4 count & liver enzymes"

Name of P.G. Student : Dr Shashank S. Gowda
Dept of Medicine

Name of Guide/Co-investigator : Dr Sanjeev kumar N. Bentoor
professor

DR.TEJASWINI VALLABHA
CHAIRMAN

CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:
1)Copy of Synopsis/Research Project
2)Copy of informed consent form.
3)Any other relevant documents.

INFORMED CONSENT FORM

BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR- 586103

TITLE OF THE PROJECT STUDY OF CO-INFECTION OF HEPATITIS B AND HEPATITIS C IN HIV PATIENTS

PRINCIPAL INVESTIGATOR - Dr. SHASHANK S GOWDA

9945844657

P.G.GUIDE NAME - Dr. SANJEEV KUMAR N. BENTOOR

PROFESSOR OF MEDICINE

08352-, Ext-2148

All aspects of this consent form are explained to the patient in the language understood by him/her.

I) **INFORMED PART**

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study

3) RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation in this study will help to patient's survival and better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime. Dr. SHASHANK S GOWDA is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. SHASHANK S GOWDA may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Dr. SHASHANK S GOWDA Date

(Investigator)

II) **STUDY SUBJECT CONSENT STATEMENT:**

I confirm that Dr. SHASHANK S GOWDA has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____

Witness to signature Date

PROFORMA:

Name:	I.P. No.:
Age:	Unit:
Sex:	Case No.
Religion:	
Income:	DOA:
Education:	DOD:

I. Presenting complaints:

Fever:

Cough:

Weight loss and others:

II. History of presenting illness:

1. Fever:

Duration: Hours /days / Months

Onset: Acute / Sub acute/ Gradual

Any recording of temperature

2. Loss of Appetite: Days / Months / Years

Loss of Weight: Days / Months / Years

About _____%

Previous weights recorded and date (if any)

Weakness

Malaise

Headache

Vomiting

Nausea

Yellowish discoloration of eyes

Swelling of neck

Distension of abdomen

II. Past History

a. Tuberculosis: Yes- No Pulmonary Extra Pulmonary

b. Diabetes Mellitus - Present / Absent Duration:

c. Hypertension - Present / Absent Duration,

d. Allergic - Present / Absent Duration,

e. Any other.

III. Treatment history:

a. For Tuberculosis

Duration:

Regular / Irregular:

Side Effects: Yes / No

b. For Diabetes Mellitus

Drugs:

Regular / Irregular:

c. For hypertension

Drugs:

Regular / Irregular

d. BCG taken - Yes / No

e. Use of corticosteroids - Yes / No

Previous Radiological examination

Previous Laboratory Examination

IV. Family History

Parents - Alive / Dead

Brother - Alive / Dead

Sisters - Alive / Dead

Children - Alive'/ Dead

Spouse - Alive / Dead

History of Tuberculosis: Yes / No

Who is suffering:

Whether on Treatment

H/o Contact

History of HIV Infection:

Who is suffering

H/O Contact

V. Personal history:

Diet: Vegetarian / Mixed

Appetite: Normal / Reduced / Increased

Bladder: Regular / Altered

Bowel: Regular / Altered

Sleep: Normal / Disturbed/ Insomnia

Habits:

H/O Smoking: Yes / No

Duration: Days / Months / Years

Quantity: _____ / day

Cigarettes / Beedi

H/O Tobacco Chewing: Present / Absent

Duration: Days / Months / Years

H/O Alcohol: Yes / No

Duration: Days / Months / Years

Quantity: _____ / day

Country Liquor / Brandy / Whisky / Cocktail

Exposure to risk of STD: Yes / No

Direct Exposure / Indirect Exposure

Heterosexual / Homosexual

Marital Status: Married / Single/ Polygamy / Polyandry

Menstrual History:

Age of Menarche

Present Menstrual cycles

Age of Menopause

Use of OCPs

VI. General Physical Examination

Built: Well / Moderate / Poor

Nutrition: Well / Average / Poor

Pallor / Icterus / Cyanosis / Clubbing / Koilnychia / Oedema

Dysponea - Yes / No

Lymph Nodes:

Site Consistency

No:

Matted - Yes / No

Size

Sinus discharge:

Oral Candidiasis: Yes / No

VII. Vital data:

Pulse:

BP:

Temperature:

Respiratory Rate:

Weight: On admission: On discharge:

JVP:

VIII. Systemic examination:

1. Respiratory system:

a. Inspection:

Type of Chest -

Position of Trachea:

Apical Impulse.

Drooping of shoulder - Yes / No Side - Rt / Lt

Intercostal in drawing: Yes / No

Type of Respiration

Movements:

Expansion:

Accessory muscles of respiratory - Acting - Yes / No

Local bulging - Yes / No

Prominent Veins - Yes / No

b. Palpation:

Position of trachea:

Position of apex beat:

Movement: Right hemithorax Left hemithorax

Inspiration (Cm)

Expiration (Cm)

Expansion (Cm)

Total expansion of chest (Cm)

Antero-posterior diameter (Cm)

Transverse diameter (Cm)

Ratio (AP:Transverse)

Vocal fremitus

Tenderness:

Distended veins - Direction of flow

Any bulging:

c. Percussion:

Direct Percussion:

Indirect Percussion:

Liver dullness:

Tidal Percussion:

Cardiac dullness:

Shifting dullness:

d. Auscultation:

Breath sounds:

Type - Vesicular / Broncho-vesicular / Bronchial

Intensity - Normal / Increased / Decreased

Bronchial area

Added sounds:

Rales Area Type

Ronchi Area Type

Pleural rub Yes / No

Vocal resonance - Normal / Increased / Decreased

Bronchophony / Aegophony / Whispering Pectoroliquoy / Succussion splash

2. Cardiovascular system:

Inspection:

Palpation:

Percussion:

Auscultation:

3. Per-abdomen

Inspection:

Palpation:

Percussion:

Auscultation:

Genitalia:

4. Central nervous system:

Mental status examination:

Cranial Nerves:

Motor System:

Sensory System:

Co-ordination:

Signs of Meningeal irritation - Yes / No

Signs of Cerebellar disease - Yes / No

Locomotor System:

Optic fundus examination:

IX Investigations

1. ELISA test for HIV-I and II

2. HbsAg by ELISA

3. Anti HCV Antibodies by ELISA

4. Urine Examination for bile salts and bile pigments.

5. LFT in HBV and HCV positive cases.

6. Ultrasound examination of abdomen.

X. Final Diagnosis:

XI. Treatment Given:

XII. Advice on Discharge:

XIII. Follow up: Cured / Improved / No change / Aggravated /Expired.

XIV. Conclusion

Signature of guide: Signature of student

KEY TO MASTER CHART

Sl. No. - Serial Number

PRE/ART NO- Pre-antiretroviral therapy number

IP NO. - Inpatient Patient Number

M - Male

F - Female

IDU - Intravenous drug abusers

HbsAg - Hepatitis B surface antigen

Anti HCV - Hepatitis C antibodies

S.Bil- Serum Bilirubin

SGOT- Serum Glutamic Oxaloacetic Transaminase

SGPT- Serum Glutamic Pyruvic Transaminase

ALP- Alkaline Phosphatase.

MASTER CHART

SL/NO	PATIENTS NAME	ART/PRE ART NO	IPNO	AGE	SEX	OCCUPATION	BLOOD TRANSFUSION	MULTIPLE SEXUAL PARTNERS	IDU	FEVER	COUGH	LOSS OF WEIGHT	JAUNDICE	HbsAg	Anti HCV	CD4 COUNT	S.BIL	SGOT	SGPT	ALP
1	PANDU	4234/15	39236/15	43	M	CARPENTER	-	+	-	-	-	+	-	-	-	720	1	375	435	117
2	SAMBAJI	3728/15	37213/15	36	M	LABOURER	-	+	-	+	-	-	+	-	-	320	5.5	195	124	155
3	MAHANANDA	483/14	38827/15	30	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	303	0.6	40	116	47
4	RAVINDRA	682/14	41684/15	38	M	COBBLER	-	+	-	+	+	+	-	-	-	676	0.4	28	88	59
5	SUKADEV	2367/15	159/16	40	M	SHOPKEEPER	+	-	-	+	+	+	-	-	-	410	0.6	17	25	175
6	BASAPPA	6887/15	510/16	65	M	FARMER	-	+	-	-	-	-	-	-	-	295	0.5	24	22	101
7	SHANKREPPA	799/16	1886/16	39	M	LABOURER	-	+	-	+	-	-	-	-	-	430	0.5	14	25	41
8	SHIVANAND	1674/15	1267/16	38	M	DRIVER	-	+	-	-	-	-	-	+	-	74	0.5	119	111	50
9	SHOBHA	6554/15	1576/16	35	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	398	0.4	22	26	202
10	BASAPPA	162/15	1699/16	52	M	FARMER	-	-	-	-	-	+	-	+	-	205	0.5	128	216	274
11	ALLABAKASH	2235/15	1239/16	40	M	WELDER	-	+	-	-	+	+	-	-	-	620	0.5	27	48	151
12	SHARANAPPA	157/16	1266/16	42	M	DRIVER	-	-	-	-	-	-	-	-	-	43	0.6	23	54	149
13	BASAPPA	524/15	1168/16	60	M	LABOURER	-	+	-	-	-	-	-	-	-	304	1	28	13	90
14	VIJAYKUMAR	2034/14	844/16	48	M	CLERK	-	-	-	+	-	-	-	+	-	178	0.4	168	371	137
15	KRISHNA	1543/14	751/16	43	M	POLICE	-	+	-	-	-	-	-	+	-	120	0.4	119	182	251
16	SHOBHA	456/15	887/16	35	F	TAILOR	+	-	-	-	-	+	+	-	-	344	2.1	13	16	70
17	RAGHAVENDRA	4677/14	654/16	32	M	LABOURER	-	-	-	-	-	-	-	-	-	604	0.5	16	53	52
18	GURAPPA	2423/15	720/16	38	M	FARMER	+	-	-	-	-	-	-	-	-	404	0.9	20	45	169
19	BOURAMMA	1876/15	153/16	40	F	HOMEMAKER	-	-	-	-	-	+	-	-	-	360	0.3	33	41	118
20	KUMAR	3078/14	2437/16	45	M	FARMER	-	+	-	+	-	-	-	+	-	280	0.5	19	42	82
21	GOPAL	1542/15	4401/16	52	M	CARPENTER	-	-	-	+	-	-	-	+	-	128	0.6	300	350	100
22	ADEWWA	148/16	3997/17	22	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	382	0.5	16	14	76
23	BAGAMMA	189/16	3938/17	49	F	FARMER	-	+	-	-	-	-	+	-	-	720	2	375	435	117
24	MALEPPA	1635/15	3220/17	60	M	FARMER	-	-	-	-	-	-	-	-	-	128	0.6	42	25	119
25	SHANKARGOUDA	1980/15	3641/17	42	M	BARBER	-	-	-	-	-	-	-	-	-	138	0.5	19	82	157
26	VISHWANATH	2867/16	1534/17	36	M	FARMER	-	+	-	+	+	+	-	-	-	600	0.5	124	83	338
27	PANDIT	2314/15	4284/17	30	M	LABOURER	-	+	-	+	-	+	-	-	-	659	0.4	48	21	112
28	BASAPPA	3463/14	4035/17	28	M	FARMER	-	-	-	-	-	+	-	-	-	500	1	34	30	88
29	MAHADEVA	3378/15	3453/17	47	M	FARMER	-	+	-	-	-	-	+	-	-	128	2	40	19	56
30	AKSHATA	243/16	2560/17	18	F	STUDENT	-	-	+	-	-	+	-	-	-	296	0.8	34	15	175
31	SHIVANAND	876/14	1112/17	32	M	FARMER	+	-	-	-	-	-	-	-	-	400	0.7	33	17	206
32	BHIMAVVA	986/15	2652/17	35	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	300	0.5	17	20	64
33	GOURAMMA	1985/16	1293/17	32	F	HOMEMAKER	-	-	-	+	+	-	-	-	-	140	0.6	68	20	116
34	SHRISHAIL	3674/14	1699/17	58	M	TEACHER	-	-	-	-	-	-	-	-	-	600	0.5	16	53	52
35	PANDU	541/16	25355/16	45	M	LABOURER	-	+	-	-	-	+	-	-	-	128	0.5	14	15	155
36	BHIMARAYA	3277/14	20555/16	20	M	FARMER	-	+	-	-	-	+	-	-	-	247	0.5	18	16	83
37	LAXMI	285/15	18407/16	32	F	HOMEMAKER	-	+	-	+	-	-	-	-	-	500	0.4	28	13	164
38	SHANTAPPA	2563/15	18107/16	55	M	LABOURER	-	-	-	+	+	+	-	-	-	130	0.5	63	38	94
39	SUNIL	1982/14	17817/16	22	M	FARMER	-	+	-	+	+	-	-	-	-	296	0.5	17	20	180
40	VIDYA	743/14	17723/16	45	F	TAILOR	-	-	-	-	+	+	-	-	-	329	0.5	43	48	151
41	GIRIJA	129/14	960/17	38	F	HOMEMAKER	-	+	-	+	-	-	-	-	-	347	0.6	27	25	104

42	MARUTI	3874/15	43170/16	36	M	LABOURER	-	+	-	+	+	+	-	-	-	418	0.4	30	39	181
43	JAYASHREE	946/14	149/17	45	F	HOMEMAKER	-	-	-	+	-	+	+	-	-	300	5.6	195	124	155
44	JANABAI	178/15	4164/17	36	F	HOMEMAKER	-	+	-	+	-	+	-	-	-	130	0.4	40	45	100
45	SURYAKANTH	199/14	27988/16	44	M	DRIVER	-	-	-	-	-	-	-	-	-	100	1	375	400	100
46	BHARATI	1829/15	28306/16	35	F	HOMEMAKER	-	-	-	-	-	+	-	-	-	289	0.5	18	9	60
47	SANGAMESH	1452/15	28054/16	61	M	DRIVER	-	-	-	-	-	-	-	-	-	140	0.5	25	50	149
48	SUSHILABAI	1390/14	27135/16	30	F	HOMEMAKER	-	-	-	+	-	-	+	-	-	690	2	33	10	71
49	RANJANA	705/14	27255/16	25	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	600	1	33	10	71
50	BHARATI	1892/15	16581/16	31	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	184	0.7	50	55	150
51	BASAPPA	1382/13	17004/16	52	M	BARBER	-	-	-	-	-	-	-	-	-	70	0.7	25	50	140
52	KASIBAI	1765/15	22388/16	35	F	FARMER	-	-	-	+	-	+	-	-	-	216	0.6	253	17	175
53	KISHAN	1696/15	23711/15	45	M	LABOURER	-	+	-	+	+	+	-	-	-	220	0.4	20	9	62
54	MARUTI	1567/16	26238/16	66	M	FARMER	-	+	-	+	-	-	-	-	-	120	0.5	53	16	52
55	SUKHDEV	1420/15	159/16	40	M	DRIVER	-	+	-	+	+	+	-	-	-	159	0.6	25	17	175
56	MALASHREE	1620/16	1392/17	26	F	CLERK	-	+	-	+	-	-	-	-	-	153	0.7	14	12	111
57	MALLIAPPA	1380/15	15942/16	65	M	FARMER	-	-	-	-	-	-	-	-	-	120	1	30	40	90
58	SHRISHAIL	1391/14	16393/16	35	M	FARMER	-	+	-	-	-	-	-	-	-	192	0.9	39	37	87
59	LAXMI	1874/15	16259/16	29	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	154	1.2	40	20	107
60	KAMALABAI	1598/14	16592/16	55	F	FARMER	-	-	-	+	-	-	-	-	-	292	0.9	39	37	87
61	PARVATI	1900/14	17134/16	25	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	200	0.7	32	28	152
62	RENUKA	301/15	2788/16	30	F	HOMEMAKER	-	+	-	-	-	+	-	-	-	250	0.5	16	18	74
63	BASAVARAJ	4531/16	139/17	30	M	LAWYER	-	+	-	-	-	+	-	-	-	270	0.4	23	12	110
64	SIKANDAR	1252/14	3003/16	55	M	SHOPKEEPER	-	+	-	-	-	+	-	-	-	142	1.5	40	20	69
65	SHIVSHARAN	786/14	3399/16	55	M	MECHANIC	-	+	-	+	+	+	-	-	-	200	1.5	32	26	104
66	BHIMARAYA	3523/14	2374/16	35	M	FARMER	-	+	-	+	+	+	-	-	-	312	0.8	62	29	86
67	LALITA	2311/15	3369/16	40	F	HOMEMAKER	-	+	-	+	-	+	-	-	-	194	0.5	54	25	100
68	ANNAVEERAYYA	643/15	13522/16	29	M	FARMER	-	+	-	-	-	-	-	-	-	174	0.5	36	21	82
69	PARVATI	1592/16	4958/17	29	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	180	0.6	32	27	92
70	RASHMI	324/14	14112/16	18	F	LABOURER	-	+	-	-	-	-	-	+	-	120	0.4	130	135	255
71	SHAILA	1521/15	10876/16	35	F	HOMEMAKER	-	+	-	+	-	-	-	-	-	225	0.5	25	14	50
72	SURESH	873/16	1077/17	45	M	FARMER	-	+	-	-	-	-	-	+	-	103	0.7	150	145	280
73	LAXMIBAI	1500/15	11152/16	47	F	HOMEMAKER	-	-	-	-	-	+	-	+	-	120	0.4	81	74	203
74	MAREPPA	987/14	10967/16	35	M	FARMER	+	+	-	-	-	+	-	+	-	207	0.5	127	110	280
75	KASTURI	385/15	10355/16	35	F	FARMER	-	+	-	-	-	+	-	+	-	150	0.4	72	86	196
76	BOURAMMA	824/14	9000/16	28	F	HOMEMAKER	-	+	-	-	-	+	-	+	-	173	0.5	97	98	160
77	BAPURAYA	1823/14	8858/16	34	M	TEACHER	-	+	-	+	+	+	-	+	-	97	0.7	147	118	246
78	BHEEMARAYA	762/15	7208/16	50	M	BARBER	-	+	-	+	-	+	-	-	-	306	0.5	18	15	69
79	SHRISHAIL	871/14	7343/16	55	M	FARMER	-	+	-	+	-	+	-	+	-	189	0.4	133	112	246
80	SURESH	1833/15	6779/16	50	M	CARPENTER	-	+	-	-	-	-	-	-	-	258	0.9	46	32	166
81	BASAVARAJ	388/14	6399/16	33	M	FARMER	-	+	-	-	-	+	+	+	-	154	4.3	500	434	157
82	RAVIKANT	1722/15	359/16	36	M	SHOPKEEPER	-	-	-	-	-	+	-	-	-	236	0.5	28	14	80
83	GOUDAPPA GOUDA	365/14	1270/16	49	M	COOLIE	-	-	-	-	-	-	+	-	-	211	2.1	92	38	211
84	BASAVARAJ	308/14	1340/16	38	M	BARBER	-	-	-	-	-	+	-	-	-	309	0.5	20	10	77
85	SHARANAPPA	267/16	1460/16	48	M	FARMER	-	-	-	-	-	+	-	-	-	464	0.5	109	61	500
86	PANDURANGA	2465/16	1504/16	45	M	FARMER	-	-	-	-	-	-	-	-	-	281	0.5	32	22	62

87	MAHADEVI	1856/15	1566/16	32	F	HOMEMAKER	-	-	-	-	-	+	-	-	-	214	0.5	30	22	61
88	SURESH	453/15	1987/16	52	M	SHOPKEEPER	-	-	-	-	-	-	-	+	-	186	0.6	229	174	84
89	SHRISHAIL	2891/14	2197/16	32	M	FARMER	-	-	-	-	-	-	+	-	-	176	5.5	187	90	108
90	MAHADEVI	869/15	2228/16	30	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	278	0.5	37	13	45
91	SAIBANNA	769/13	2544/16	28	M	FARMER	-	-	-	-	-	-	-	-	-	414	0.6	13	10	41
92	SHANTABAI	419/13	3480/16	45	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	327	1.2	24	10	98
93	SURESH	917/15	4142/16	52	M	FARMER	-	+	-	+	-	-	-	+	-	175	0.6	51	37	127
94	CHANNABASU	4892/15	4258/16	38	M	COOLIE	-	-	-	-	+	+	-	-	-	482	0.5	83	45	439
95	KASTURI	43/14	4304/16	34	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	178	0.4	22	36	96
96	INDRABAI	672/13	4664/16	45	F	FARMER	+	-	-	-	-	-	-	-	-	524	0.5	45	26	494
97	BUVANESHWARI	7189/14	4870/16	42	F	COOLIE	-	-	-	-	+	+	-	-	-	368	0.5	28	26	49
98	SHIVAJI	3123/15	5254/16	45	M	DRIVER	-	+	-	+	-	-	-	-	-	225	0.4	27	14	130
99	SHARANAPPA	6255/14	5349/16	60	M	VENDOR	-	-	-	+	-	+	-	-	-	196	0.5	43	39	99
100	HANAMAVVA	3981/14	6916/16	50	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	504	0.5	18	20	63
101	BABU	1092/14	6928/16	43	M	SHOPKEEPER	-	-	-	-	+	+	-	-	-	216	0.5	29	39	111
102	MAHADEVI	3144/15	7749/16	40	F	FARMER	-	-	-	-	-	-	-	-	-	414	0.4	12	10	98
103	BANEPPA	14/13	7220/16	40	M	CLERK	+	-	-	-	-	-	+	-	-	278	3	48	15	154
104	BASAVARAJ	3267/14	7958/16	35	M	FARMER	-	-	-	+	-	-	-	-	-	177	1.2	42	20	91
105	MADIVALAPPA	211/13	8010/16	45	M	DRIVER	+	+	-	-	-	-	-	-	-	438	0.6	44	59	471
106	BASAPPA	3761/14	8366/16	45	M	FARMER	-	-	-	-	-	-	-	-	-	216	0.5	20	17	109
107	BALACHANDRA	4724/13	8855/16	54	M	FARMER	-	-	-	+	-	-	+	-	-	376	5.8	37	18	85
108	MADIVALAPPA	7152/14	9204/16	58	M	LABOURER	-	-	-	-	-	+	-	-	-	266	0.6	177	33	82
109	SIDDAPPA	4519/13	9276/16	40	M	COOLIE	-	-	-	-	-	-	-	-	-	512	0.8	22	13	82
110	JAYASHREE	1713/13	9333/16	55	F	HOMEMAKER	-	-	-	-	-	+	-	-	-	226	0.5	15	10	110
111	JAKAVVA	298/13	9489/16	48	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	421	0.4	24	20	319
112	HANAMANT	3811/14	9885/16	35	M	C0BBLER	-	-	-	+	+	-	+	-	-	412	3	168	37	78
113	SAIBANNA	4379/13	10001/16	28	M	BARBER	-	-	-	-	-	-	-	-	-	474	0.4	59	52	500
114	MALLIKARJUN	6519/14	10364/16	35	M	FARMER	-	-	-	+	+	-	-	-	-	286	0.5	86	92	24
115	ANNAPURNA	3881/13	10729/16	25	F	HOMEMAKER	-	-	-	+	+	-	-	-	-	477	0.6	29	14	49
116	SUKHDEV	9712/14	11014/16	40	M	FARMER	-	+	-	+	-	-	-	-	-	296	0.5	18	19	107
117	DURGAPPA	8913/13	11180/16	45	M	DRIVER	-	+	-	+	+	-	-	-	-	242	0.5	22	26	137
118	RENUKA	3178/13	11307/16	35	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	207	0.9	42	16	37
119	MAHAVEER	3904/14	11474/16	32	M	LABOURER	-	-	-	-	+	+	-	-	-	512	0.5	25	15	91
120	SHRIMANTH	4511/13	12044/16	45	M	FARMER	-	-	-	+	-	-	-	-	-	326	0.5	26	24	78
121	KALYANSINGH	3901/14	12080/16	70	M	SHOPKEEPER	-	+	-	+	-	-	+	-	-	412	3.1	56	50	109
122	LAXMIBAI	563/14	11152/16	47	F	HOMEMAKER	-	-	-	-	+	+	-	+	-	115	1.2	124	120	264
123	RAYAGOND	413/15	12580/16	42	M	LABOURER	-	-	-	+	-	-	-	-	-	394	0.9	26	24	64
124	SHIVAPPA	3412/15	12654/16	45	M	FARMER	-	+	-	+	-	-	-	-	-	428	1	28	26	54
125	VIJAYALAXMI	1542/15	12788/16	28	F	CLERK	-	-	-	+	-	-	-	-	-	365	0.4	23	16	75
126	KALAVATHI	671/14	13000/16	32	F	HOMEMAKER	-	-	-	+	+	-	-	-	-	506	1.2	27	23	70
127	TIPANNA	639/15	13721/16	39	M	DRIVER	-	+	-	+	-	-	-	-	-	413	0.4	44	26	68
128	SIDDANNA	213/13	13914/16	48	M	FARMER	-	-	-	+	-	-	-	-	-	263	0.5	19	11	157
129	BOURAMMA	1462/14	14106/16	33	F	FARMER	-	-	-	+	+	-	-	-	-	426	0.4	20	17	88
130	AHAMED	451/13	14551/16	35	M	DRIVER	-	+	-	+	-	-	-	-	-	380	0.4	76	71	81
131	KHEMU	3812/14	14815/16	52	M	COOLIE	+	-	-	-	-	-	-	-	-	382	0.5	36	18	202

132	B KRISHNA	4138/13	15011/16	50	M	DRIVER	-	+	-	+	-	-	-	+	+	140	0.6	222	182	106
133	RAJSHEKHAR	1752/14	16271/16	45	M	BARBER	-	+	-	-	+	-	-	-	-	456	0.7	28	25	56
134	RAMESH	1873/15	18513/16	25	M	STUDENT	-	-	+	-	-	-	-	-	-	324	0.5	24	20	144
135	SHEKAPPA	814/14	18597/16	35	M	DRIVER	-	+	-	-	+	-	-	-	-	308	0.5	25	12	337
136	SHRISHAIL	1652/15	19392/16	60	M	FARMER	-	+	-	+	-	-	+	+	-	184	2.5	100	32	226
137	MAHESH PATIL	1332/15	19397/16	42	M	LABOURER	-	+	-	-	+	-	-	-	-	324	0.6	42	27	302
138	ANASUYA	1786/13	19409/16	38	F	FARMER	-	-	-	-	-	-	-	-	-	282	0.5	32	13	61
139	SHANMUKH	451/14	19737/16	32	M	FARMER	-	-	-	+	-	+	-	-	-	341	0.5	44	20	86
140	SHRISHAIL	816/13	19930/16	47	M	DRIVER	-	+	-	+	-	-	-	-	-	344	0.6	34	26	60
141	IRAMMA	731/14	20167/16	60	F	HOMEMAKER	-	-	-	-	+	-	-	-	-	376	0.8	28	24	84
142	MUDAKAPPA	2419/15	21426/16	45	M	FARMER	-	-	-	+	-	+	-	+	-	128	0.4	86	81	194
143	BHAGANNA	879/15	21460/16	55	M	FARMER	-	-	-	-	-	-	-	+	-	184	0.5	48	17	138
144	PANDIT	3613/15	21924/16	30	M	DRIVER	-	+	-	+	-	+	-	+	-	168	0.4	60	67	168
145	SHANTABAI	2881/13	22276/16	38	F	FARMER	+	-	-	-	-	+	-	-	-	314	2	40	21	150
146	SANGAPPA	155/15	23376/16	35	M	DRIVER	-	+	-	+	-	-	-	-	-	320	0.5	32	37	179
147	PARASHURAM	3551/14	23469/16	32	M	FARMER	-	-	-	+	-	+	-	-	-	292	0.6	18	12	340
148	DODDAWWA	658/15	24680/16	32	F	HOMEMAKER	+	-	-	-	-	+	-	-	-	431	0.5	18	20	92
149	BASAMMA	451/15	25340/16	35	F	HOMEMAKER	-	-	-	+	-	-	+	+	-	140	4.6	455	291	291
150	BASAVARAJ	367/16	26881/16	38	M	FARMER	-	-	-	+	-	-	+	-	-	208	2.6	38	10	104
151	NINGAPPA	512/15	27259/16	30	M	LABOURER	-	-	-	-	-	-	-	-	+	112	0.7	50	46	233
152	LAXMIBAI	1423/13	27590/16	45	F	FARMER	-	-	-	+	+	-	-	-	-	267	0.5	62	34	165
153	MADIVALAPPA	3511/13	28504/16	48	M	DRIVER	-	+	-	-	-	+	-	-	-	240	0.4	74	157	89
154	ATULRAY	2144/14	28866/16	42	M	SHOPKEEPER	-	+	-	+	-	-	-	-	-	318	0.7	21	16	76
155	SUNANDA	599/14	29490/16	35	F	HOMEMAKER	-	+	-	-	+	+	-	-	+	144	0.6	153	130	326
156	RENUKA	1553/16	29872/16	36	F	HOMEMAKER	-	+	-	+	-	+	-	-	-	312	0.4	24	16	116
157	SIDDAMMA	611/15	30702/16	28	F	FARMER	-	+	-	-	-	+	-	-	-	311	0.5	25	11	111
158	BASANNA	544/13	31349/16	52	M	FARMER	-	+	-	+	-	+	-	-	-	276	0.6	15	23	89
159	RAMESH	1889/14	31571/16	35	M	COOLIE	-	+	-	-	-	-	-	-	-	303	0.4	21	23	79
160	RUKMINI	341/14	32404/16	31	F	HOMEMAKER	-	-	-	+	-	-	-	-	-	390	0.5	25	75	99
161	MALLIKARJUN	3991/14	32949/16	35	M	DRIVER	-	+	-	+	-	-	-	+	-	170	0.7	57	95	164
162	BIBI FATIMA	726/15	33109/16	45	F	HOMEMAKER	-	+	-	-	-	-	-	-	-	220	0.5	41	13	110
163	NINGAMMA	561/13	33945/16	45	F	FARMER	-	-	-	+	-	-	-	-	-	292	0.5	17	11	99
164	YASHAVANTH	2881/14	33952/16	50	M	SHOPKEEPER	+	+	-	-	-	-	-	-	-	344	0.5	18	11	32
165	DASHARATH	3671/13	36052/16	60	M	COOLIE	-	+	-	+	-	+	-	-	-	256	0.5	37	26	25
166	SANTOSH	615/15	36264/16	26	M	DRIVER	-	+	-	+	+	-	-	-	+	170	0.6	148	154	164
167	NABISAB	2715/15	36439/16	40	M	CARPENTER	-	-	-	-	-	-	-	-	-	372	0.5	37	23	258
168	SOMAPPA	2466/15	37011/16	40	M	PAINTER	-	+	-	+	-	+	+	-	+	140	10.1	108	27	371
169	MAHAVEER	3712/14	37538/16	37	M	FARMER	+	+	-	-	-	-	-	-	-	204	0.6	19	11	99
170	HAJILAL	1561/14	37548/16	35	M	MECHANIC	-	+	-	+	-	-	-	+	-	154	0.5	61	27	236
171	ABDUL	569/14	38475/16	37	M	BUTCHER	+	+	-	-	-	-	-	-	-	336	0.4	47	81	107
172	SUNANDA	3553/13	38484/16	45	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	285	0.5	21	17	105
173	SARASWATHI	466/15	39634/16	35	F	FARMER	-	-	-	+	-	-	+	-	+	140	2.5	77	28	780
174	YOUNUS	884/15	40281/16	40	M	MECHANIC	-	+	-	-	-	-	+	+	-	170	2.4	180	46	198
175	BORAMMA	719/14	40697/16	36	F	FARMER	-	+	-	-	-	-	-	-	-	190	0.4	81	42	134
176	AJEET	431/16	41220/16	41	M	PAINTER	-	+	-	-	-	-	-	-	-	260	0.8	59	16	286

177	SHUBHASH	1677/15	513/17	40	M	SHOPKEEPER	-	-	-	-	-	-	-	-	-	304	0.5	33	80	244
178	AMEENA	499/14	1036/17	35	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	274	0.5	25	18	68
179	SHOBHA	789/13	2819/17	30	F	HOMEMAKER	-	+	-	+	-	+	-	-	-	275	0.4	43	9	59
180	HOLIBASAPPA	1886/14	2842/17	43	M	FARMER	-	-	-	-	-	-	-	-	-	352	0.4	37	23	325
181	DURGANATH	712/13	3776/17	40	M	SHOPKEEPER	-	+	-	-	-	-	-	-	-	242	0.4	27	16	93
182	SIDDU	251/15	5191/17	40	M	FARMER	-	-	-	-	-	+	-	+	-	170	0.8	52	61	99
183	SHARANAMMA	614/13	6007/17	40	F	HOMEMAKER	-	+	-	-	-	-	-	-	-	224	0.8	66	20	76
184	SURYAKANTH	412/15	6727/17	55	M	COOLIE	-	+	-	-	-	-	-	-	-	340	1	43	46	140
185	SUNIL	3421/13	6754/17	40	M	DRIVER	-	+	-	-	-	+	-	-	-	295	0.6	30	34	95
186	KASHINATH	2333/15	8134/17	46	M	SHOPKEEPER	-	+	-	-	-	-	-	-	-	284	0.8	25	27	72
187	REVANNASIDDAPPA	3155/14	8237/17	50	M	LABOURER	-	+	-	-	-	-	-	-	-	278	0.4	31	13	103
188	KASHIRAYA	2886/15	8935/17	70	M	FARMER	-	+	-	-	-	+	-	-	-	394	0.5	26	24	92
189	SANJUKUMAR	591/14	9487/17	35	M	DRIVER	-	+	-	-	-	+	-	-	-	326	0.6	20	13	112
190	ABDUL	3411/12	9542/17	38	M	MECHANIC	-	+	-	-	-	-	-	-	-	277	0.5	19	18	66
191	SOMU	477/15	9561/17	50	M	FARMER	-	-	-	+	-	-	-	-	-	308	0.4	29	17	159
192	SUSHILABAI	782/14	9677/17	34	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	372	0.4	42	42	74
193	MALLAPPA	479/16	10922/17	65	M	FARMER	-	+	-	-	-	-	-	+	-	164	0.6	112	54	195
194	YANKAPPA	388/15	10833/17	34	M	DRIVER	-	+	-	-	-	-	-	-	-	412	0.9	25	12	395
195	MALAKAPPA	588/15	10922/17	48	M	FARMER	-	+	-	-	-	-	-	-	-	278	0.5	48	25	164
196	MALLAPPA	633/14	11192/17	30	M	LABOURER	-	-	-	-	-	+	-	-	-	360	0.4	40	73	181
197	ASHOK	1558/15	12033/17	35	M	CLERK	-	-	-	-	-	+	-	-	-	292	0.8	22	23	123
198	SUNDRAWWA	550/14	12110/17	65	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	228	0.4	10	14	106
199	SHARADA	699/13	12146/17	40	F	HOMEMAKER	-	+	-	-	-	+	-	+	+	114	0.8	1566	905	105
200	SAMEER	377/15	12460/17	19	M	STUDENT	-	-	+	-	-	-	-	-	-	458	0.4	32	41	131
201	SONABAI	1813/13	12655/17	60	F	HOMEMAKER	-	-	-	+	-	+	-	-	-	344	0.6	26	28	94
202	SARASWATHI	695/15	13115/17	35	F	FARMER	-	-	-	+	-	+	-	-	-	414	0.9	32	17	172
203	TIMMAPPA	3669/15	13204/17	45	M	COOLIE	-	+	-	-	-	-	-	-	-	326	0.5	16	10	288
204	SHARANAGOUDA	2771/13	13484/17	35	M	SHOPKEEPER	-	+	-	+	-	-	-	-	-	402	0.4	19	15	252
205	NARASAWWA	1880/15	13681/17	40	F	FARMER	-	-	-	-	-	-	-	-	-	354	0.5	18	14	78
206	RENUKA	677/14	13685/17	18	F	STUDENT	-	-	+	+	-	+	-	-	-	374	0.5	18	15	62
207	BHARATHI	1889/16	13802/17	28	F	HOMEMAKER	-	+	-	-	-	-	-	-	-	288	0.5	34	22	255
208	ROOPA	590/16	14478/17	20	F	HOMEMAKER	-	-	-	-	-	-	-	-	-	320	0.5	20	16	70
209	SIDDALINGAPPA	1993/14	14484/17	56	M	FARMER	-	-	-	-	-	-	+	+	+	124	3.5	1417	446	141
210	MAHADEV	775/15	14495/17	30	M	SHOPKEEPER	-	-	-	-	-	-	-	-	-	236	0.7	25	22	86
211	DAYANAND	644/14	14640/17	35	M	DRIVER	-	+	-	+	-	+	-	-	-	238	0.4	32	17	92