A STUDY ON PREVALENCE AND CHARACTERISTICS OF HEPATITIS B OR/AND HEPATITIS C CO-INFECTION IN PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL

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THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

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for the award of the degree of

M.D. GENERAL MEDICINE

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THANJAVUR MEDICAL COLLEGE AND HOSPITAL

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(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

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submitted by Dr. MAGHIL BELINTA **of**

Dept. of GENERAL MEDICINE **Thanjavur Medical College, Thanjavur**

was approved by the Ethical Committee.

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DECLARATION

I solemnly declare that this Dissertation titled **"A STUDY ON PREVALENCE AND CHARACTERISTICS OF HEPATITIS B OR/AND HEPATITIS C CO-INFECTION IN PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL"** was done by me in the Department of General Medicine, Thanjavur Medical College, and Hospital , Thanjavur under the Guidance and Supervision of my Chief Prof. Dr. C. Paranthakan M.D., Professor, Department of General Medicine, Thanjavur Medical College, Thanjavur between 2015 and 2018.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University requirements for the award of M.D Degree (GENERAL MEDICINE).

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CERTIFICATE – II

This is to certify that this dissertation work titled "**A STUDY ON PREVALENCE AND CHARACTERISTICS OF HEPATITIS B OR/AND HEPATITIS C CO-INFECTION IN PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL"**of the candidate DR. MAGHIL BELINTA with registration Number 201511212 for the award of M.D in the branch of General Medicine. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2% of plagiarism in the dissertation.

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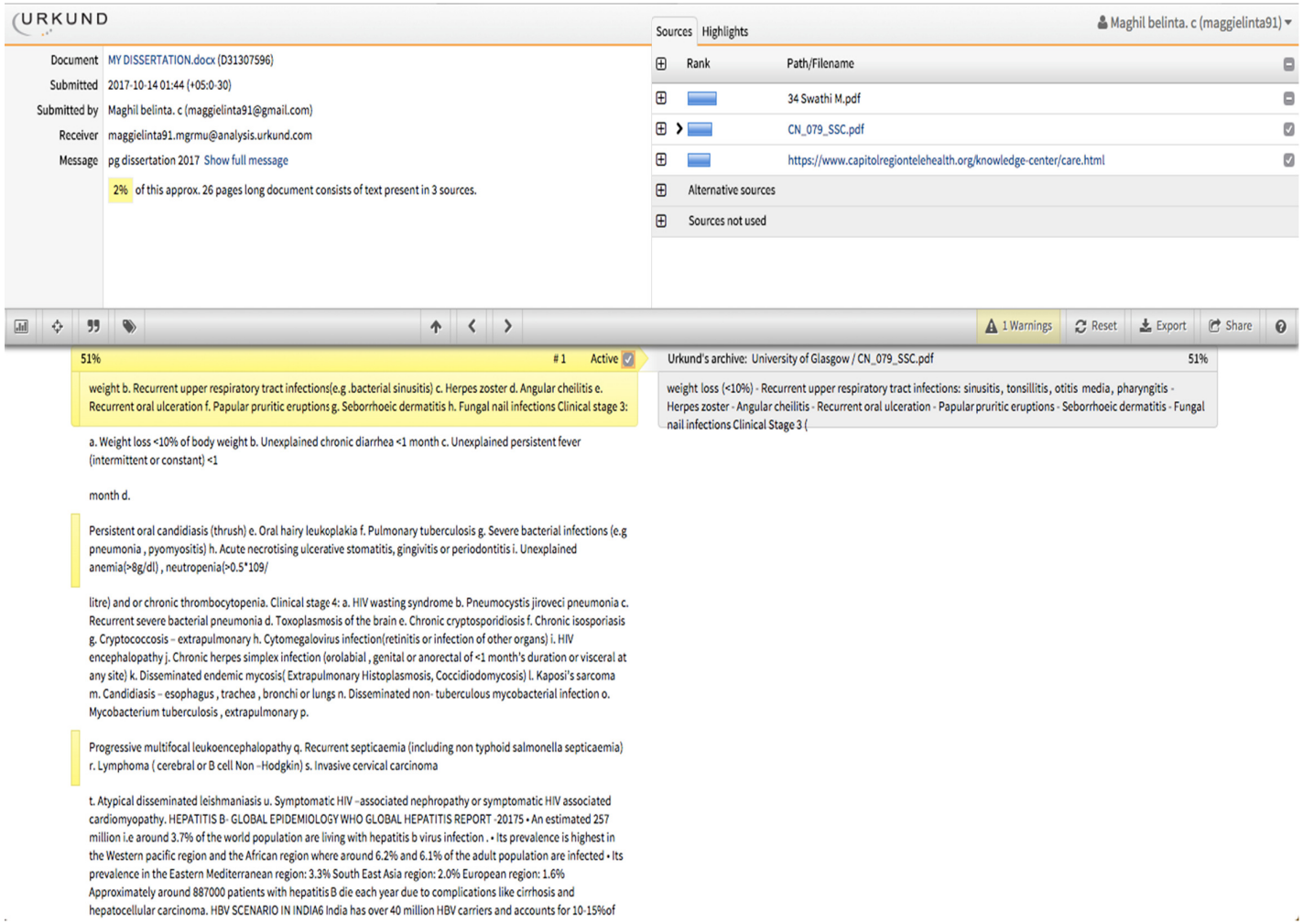
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Progressive multifocal leukoencephalopathy q. Recurrent septicaemia (including non typhoid salmonella septicaemia) r. Lymphoma (cerebral or B cell Non –Hodgkin) s. Invasive cervical carcinoma
t. Atypical disseminated leishmaniasis u. Symptomatic HIV –associated nephropathy or symptomatic HIV associated cardiomyopathy. HEPATITIS B- GLOBAL EPIDEMIOLOGY WHO GLOBAL HEPATITIS REPORT -20175 • An estimated 257 million i.e around 3.7% of the world population are living with hepatitis b virus infection . • Its prevalence is highest in the Western pacific region and the African region where around 6.2% and 6.1% of the adult population are infected • Its prevalence in the Eastern Mediterranean region: 3.3% South East Asia region: 2.0% European region: 1.6% Approximately around 887000 patients with hepatitis B die each year due to complications like cirrhosis and hepatocellular carcinoma. HBV SCENARIO IN INDIA6 India has over 40 million HBV carriers and accounts for 10-15%of
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51%
weight loss (<10%) - Recurrent upper respiratory tract infections: sinusitis, tonsillitis, otitis media, pharyngitis - Herpes zoster - Angular cheilitis - Recurrent oral ulceration - Papular pruritic eruptions - Seborrhoeic dermatitis - Fungal nail infections Clinical Stage 3 (

CONTENTS

INTRODUCTION

HIV

HIV-human immunodeficiency virus infection and AIDS –aquired immunodeficiency disorder is a spectrum of conditions caused by the retro virus called the human immunodeficiency virus. The United States was the first to recognise AIDS in the year 1981 when there emerged an unexplained occurrence of pneumocystis jiroveci pneumonia among the male and female injection drug users, the haemophiliacs and the blood transfusion recipients, female sexual partners of men with AIDS and among infants born to mother with AIDS. In the year 1983 human immunodeficiency virus was isolated from a patient with lymphadenopathy and HIV was clearly demonstrated to be the cause for AIDS by the year 1984.Indias first case of AIDS was reported from Chennai in the year 1986[1].

HBV

The hepatitis B virus is a hepatotropic double stranded DNA virus belonging to the hepadne virus family and causes hepatitis b infection in humans. It was first discovered by DR. Blumberg and his colleagues in the year 1967. A series of research and clinical observations led him to confirm that Austria antigen caused hepatitis B and this won him the Nobel prize in the year 1976.

HCV

The hepatitis c virus is also a hepatotropic but a RNA virus of the flaviviridae family. It causes the hepatitis c infection in human beings. The virus was first discovered by the scientists at CDC, NIH and industry in the year 1989 after nearly 6 years of intensive investigations between 1982 and 1988 in a lab at Chiron corporation. Numerous molecular biological tests were conducted to investigate the viral etiology of this parenterally transmitted NON-A NON-B viral hepatitis.

The hepatitis B and hepatitis C viruses attack the liver and can cause both acute and chronic diseases. These viruses are transmitted through contact with the infected blood and the body fluids in the same way as that of the HIV.

HIV COINFECTION WITH HBV AND /OR HCV

With the advancements in treatment for HIV/AIDS with effective anti retro viral drugs there is a marked decrease in mortality and morbidity due to HIV per se and its associated opportunistic infections . This has led into increased survival of the HIV patients however liver diseases due to co-infection with HBV and HCV is being recognised as a significant problem.

HBV ,HCV and HIV viruses have similar properties such as mode of transmission using a reverse transcriptase enzyme in replication, tendency to

develop chronic infection and an immense capacity of mutation on their genome causing rapid emergence of mutant strains ,some of which are resistant to the widely used anti viral agents.

Co-infection of HIV with HBV and HCV can alter the natural history of these hepatotropic viruses leading to

1. Increased rate of viral replication

2. Decrease in the spontaneous resolution of the infection

3 .Increased reactivation of the latent infection

4. Rapid progression of the disease to chronic hepatitis and cirrhosis of liver

5. Increases the risk of developing hepatocellular carcinoma.

Due to these reasons liver diseases due to HIV, HBV and /OR HCV co-infection may emerge as a great public health problem than before.

Hence knowledge on country by country prevalence of HBV/HCV co-infection with HIV is necessary in order to develop a clear strategy on the prevention and treatment of the above co-infection. Further establishing a reliable estimate of the HIV/HBV or HIV/HCV co-infection burden in the country will guide in provision of appropriate ART regimens that are effective in both the HIV and HBV or HCV co-infected patients and can also prevent the unnecessary development of mutant and drug resistant strains.

AIM OF THE STUDY

1. To estimate the sero prevalence of HBV AND /OR HCV co-infection among the HIV infected individuals.

2. To assess the association of co-infection with liver enzyme levels.

3. To assess the CD4+ T cell levels among the HIV patients co-infected with HBV/HCV.

REVIEW OF LITERATURE

The **UNAIDS** annually provide revised regional and country specific modelled estimate using the best available epidemiological and programmed data to track the HIV epidemic.

GLOBAL SCENARIO OF HIV

The global scenario of HIV as per the UNAIDS -2017 update[2] is as follows

Number of people living with HIV:

Total: 36.7 million

Adults: 34.5 million

Women (15+ years):17.8 million

Children <15 years: 2.1 million

People newly infected with HIV in 2016:

Total: 1.8 million

Adults: 1.7 million

Children (<15 yrs):160000

About 5000 new HIV infections were estimated in a day among adults and children in the year 2016. Among them 64% are in sub-Saharan Africa.

AIDS related death in 2016:

Total: 1.0 million

Adults: 890000

Children (< 15 yrs):120000

INDIAN SCENARIO OF HIV

According to the UNAIDS- 2017 UPDATE[2]

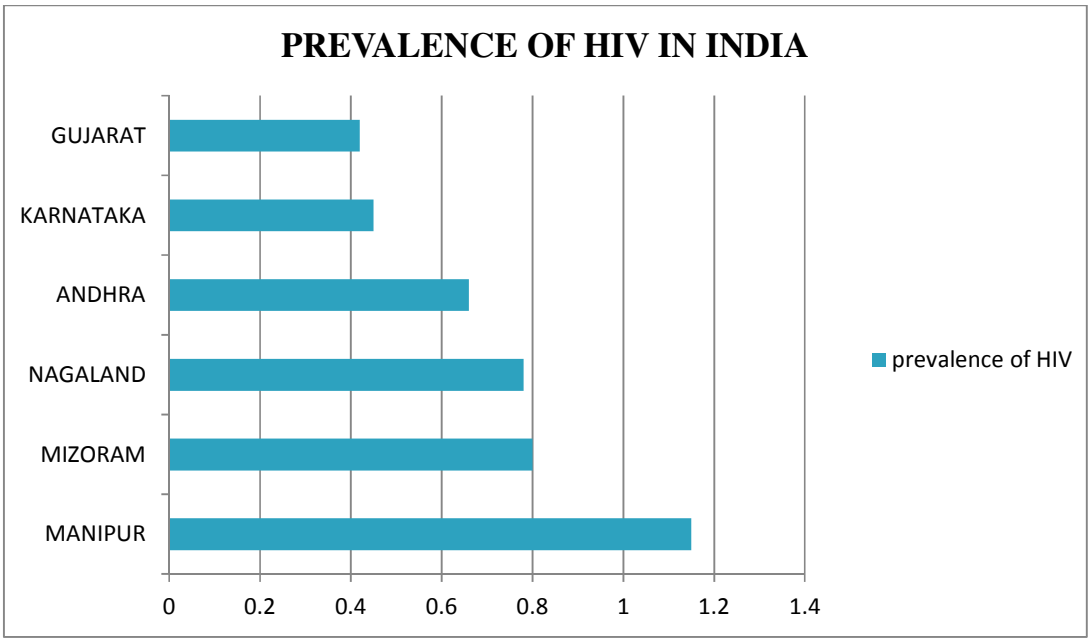
	2005	**2010**	**2016**
TOTAL NO OF PEOPLE LIVING WITH HIV	23 lakhs	22 lakhs	21 lakhs
NO OF NEW HIV INFECTION	150000	100000	80000
NO OF AIDS RELATED DEATH	150000	120000	62000

HIV prevalence in India among the adults is around 0.3%.

Among them around 50% of the adults and 33% of children are on antiretro viral treatment.

By prevalence the worst affected states in India are in the following order as per the UNAIDS –national AIDS organisation [2]

FIG: 1 PREVALENCE OF HIV AMONG VARIOUS STATES IN INDIA



Maharashtra, Chandigarh, Tripura, Tamil Nadu > 0.26%

SCENARIO IN TAMIL NADU

According to recently released, India HIV Estimation 2015 report, the prevalence of HIV infection in Tamil Nadu is greater than the national prevalence **(0.26%)**.The total number of People living with HIV in Tamil Nadu is 1.43lakhs.

HUMAN IMMUNODEFICIENCY VIRUS

The origin of HIV is not known. It is said that HIV was first introduced into the humans from another primate in sub-Saharan Africa. HIV belongs to the retroviridae family and the genus lentivirus . There are 2 sub types of HIV viruses namely HIV-1 and HIV-2.Among them HIV-1 virus has global prevalence with high virulence and high infectivity. The HIV-2 virus is prevalent among the West African region and it has low virulence and infectivity. These cytopathic viruses predominantly infect the CD4 T –helper cells of the immune system there by compromises the immune system and make the individual susceptible to many opportunistic infections.

MORPHOLOGY OF HIV[1]

Through the electron microscope the HIV is visualised as a spherical shape enveloped virus of about 90-120 nm in size. The basic structure consists of an outer envelope, HIV matrix proteins and the viral core. The outer envelope is made up of a lipid bilayer in which the envelope proteins are embedded. The various envelope proteins include

1. **Glycoprotein 120(gp120)**: It is involved in attachment of the virion to the host cell

2. **Glycoprotein 41(gp41):** It is involved in the cell fusion process

The core virus particle is composed of the ribonucleoproteins. The virion has two single stranded RNA surrounded by the capsule protein P24.. The HIV matrix proteins are found between the HIV envelope and the viral core.

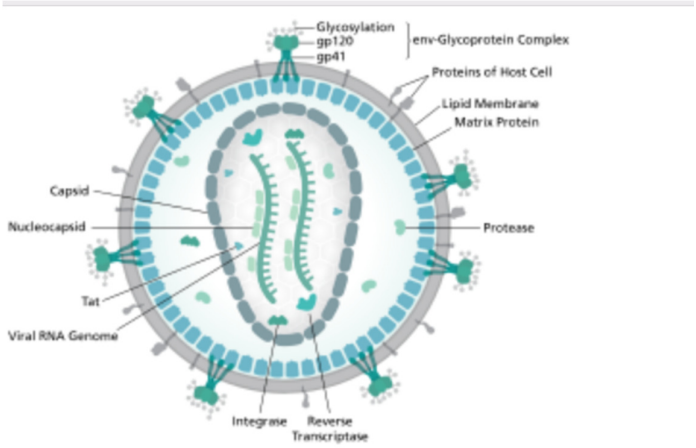


FIG:2 STRUCTURE OF HIV

HIV GENOME[1]

The genome of HIV-1 has the following genes

Gag-encodes the proteins that form the core of the virion

Pol- it encodes the viral enzymes necessary for replication, integrase, reverse transcriptase and protease

Env-encodes glycoprotein

The other six genes include tat,rev,nef,vpr,vpu which codes for the proteins taking part in the regulation of the gene expression. However HIV-2 lacks the vpu gene but has vpx gene which is not present in HIV -1.

LIFE CYCLE OF HIV[1]

The steps of the viral replication is as follows

Step1. Binding of the gp120 protein to the host cell surface of the cd4 molecule

⇩

Step2. A conformational change takes place which facilitates the binding of the virion to the co-receptors CCR5 and CXCR4.

⇩

Step 3 . This is followed by the un-coating of the virion and conversion of RNA into C-DNA – complementary DNA Using the reverse transcriptase enzyme.

⇩

Step 4.Using the viral specific integrase enzyme the C-DNA eventually gets incorporated into the host cell chromosome.

⇩

Step 5.The integrated DNA is transcribed into mRNA which further aids in synthesis of viral proteins.

⇩

Step 6. The synthesised viral RNA and the viral proteins are packed and released by cell budding process

The viruses are released following the destruction of the host cell which can be a cd4 cell, dendritic cell or a macrophage there by finally compromising the immune system of the body.

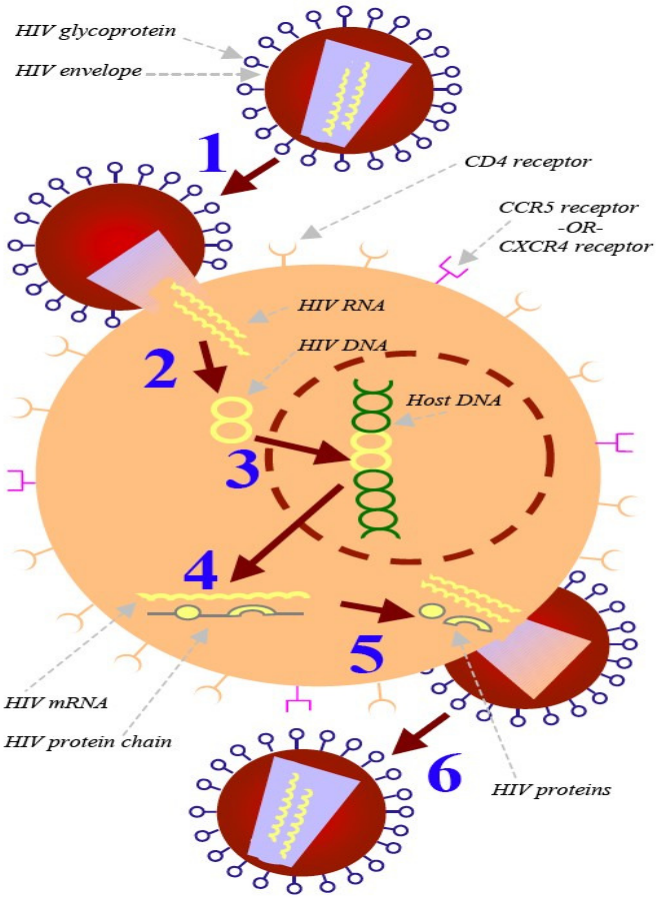


FIG: 3 PATHOGENESIS OF VIRAL REPLICATION IN HIV

CLASSIFICATION OF HIV INFECTION (WHO CLINICAL STAGING SYSTEM)

Clinical stage 1:

a. Asymptomatic

b. Persistent Generalized Lymphadenopathy

Clinical stage 2:

a. Weight loss <10% of body weight

b. Recurrent upper respiratory tract infections(e.g .bacterial sinusitis)
c. Herpes zoster
d. Angular cheilitis
e. Recurrent oral ulceration
f. Papular pruritic eruptions
g. Seborrhoeic dermatitis
h. Fungal nail infections

Clinical stage 3:

a. Weight loss >10% of body weight
b. Unexplained chronic diarrhea >1 month
c. Unexplained persistent fever (intermittent or constant) >1 month
d. Persistent oral candidiasis (thrush)
e. Oral hairy leukoplakia
f. Pulmonary tuberculosis
g. Severe bacterial infections (e.g pneumonia , pyomyositis)
h. Acute necrotising ulcerative stomatitis, gingivitis or periodontitis
i. Unexplained anemia(<8g/dl) , neutropenia(<$0.5*10^9$/litre) and or chronic thrombocytopenia.

Clinical stage 4:

a. HIV wasting syndrome
b. Pneumocystis jiroveci pneumonia

c. Recurrent severe bacterial pneumonia

d. Toxoplasmosis of the brain

e. Chronic cryptosporidiosis

f. Chronic isosporiasis

g. Cryptococcosis – extrapulmonary

h. Cytomegalovirus infection(retinitis or infection of other organs)

i. HIV encephalopathy

j. Chronic herpes simplex infection (orolabial , genital or anorectal of >1 month's duration or visceral at any site)

k. Disseminated endemic mycosis(Extrapulmonary Histoplasmosis, Coccidiodomycosis)

l. Kaposi's sarcoma

m. Candidiasis – esophagus , trachea , bronchi or lungs

n. Disseminated non- tuberculous mycobacterial infection

o. Mycobacterium tuberculosis , extrapulmonary

p. Progressive multifocal leukoencephalopathy

q. Recurrent septicaemia (including non typhoid salmonella septicaemia)

r. Lymphoma (cerebral or B cell Non –Hodgkin)

s. Invasive cervical carcinoma

t. Atypical disseminated leishmaniasis

u. Symptomatic HIV –associated nephropathy or symptomatic HIV associated cardiomyopathy.

HEPATITIS B- GLOBAL EPIDEMIOLOGY

WHO GLOBAL HEPATITIS REPORT -2017[5]

- An estimated **257 million** i.e around **3.7%** of the world population are living with hepatitis b virus infection .
- Its prevalence is **highest** in the Western pacific region and the African region where around **6.2%** and **6.1%** of the adult population are infected
- Its prevalence in the

  Eastern Mediterranean region**: 3.3%**

  South East Asia region: **2.0%**

  European region: **1.6%**

Approximately around 887000 patients with hepatitis B die each year due to complications like cirrhosis and hepatocellular carcinoma.

HBV SCENARIO IN INDIA[6]

India has over **40 million** HBV carriers and accounts for **10-15%** of the entire pool of HBV carriers of the world. The overall HBsAg positivity ranges between

2-4.7%. The point prevalence of hepatitis B among the nontribal and the tribal population was **3.07%** and **11.85%** respectively. The overall prevalence of hepatitis B was **3.70%** (corresponding to a chronic carrier rate of 2.96%)

The hyper endemic foci of HBV infection in India are reported among the Muslim tribes of Andra Pradesh where the point prevalence is found to be **21.2%**

Higher prevalence of HBV infection has also been reported among the HIV positive intra venous drug users in Manipur in north east India

The prevalence of HBsAg positivity among the pregnant woman in India is around

0.9 to 6.3%.

NATURAL HISTORY OF HBV[6]

The incubation period of HBV ranges from 30 days to 180 days with a mean of 60 days to 90 days.

Fulminant hepatitis is seen in only **0.1% to 1%** of the cases.

Progression to chronicity is as high as **95%** in neonates and is only **1-5%** in case of adults

Unlike HIV which is a cytopathic virus **HBV is not a cytopathic virus** and the liver injury in this case is due to the host mediated immune response.

Depending upon the host immune response and the viral replication chronic hepatitis B can be classified into five phases as follows:

1 .IMMUNE TOLERANT PHASE:

In this phase there is high viral replication due to the absent immune activity by the host against the virus. This is due to the development of immune tolerance by the virus by various mechanisms.

Serology- HBe antigen (HBeAg) is detected in the serum. High levels of viremia

LFT- The hepatic transaminase enzymes are usually normal or slightly elevated

2. IMMUNE CLEARANCE PHASE:

In this phase there is increased immune activity against the virus hence there is sero conversion.

Serology: HBeAg may be lost and Anti HBe developes. Lower levels of viremia is seen

LFT- Increased and fluctuating liver enzymes

Histology: Necro inflammatory activity with progressive liver injury

3.INACTIVE RESIDUAL PHASE:

Serology- loss of HBeAg, lower serum viral DNA levels < 2000 IU/ml

LFT- Normalisation of liver enzymes

Histology - Absent necro inflammatory activity.

Spontaneous loss of HBsAg may occur and this is seen upto **1-3%** per year. Following loss of HBsAg there is only a low risk for progression of liver disease or progression to hepatocellularcarcinoma. Persistent activity may be seen in 10-30% of the patients despite HBeAg conversion. Seroconversion to HBeAg positive status may be seen in approximately **4-20%** of the patients. Hence lifelong follow up is necessary even in case of carriers.

4. HBeAg NEGATIVE HEPATITIS:

This phase of hepatitis is seen immediately following HBeAg loss or after years of inactive carrier state. It is characterised by ongoing viral replication and fluctuating liver enzymes and HBV DNA levels. The absence of HBeAg in this type of hepatitis is due to presence of virions with precore or promoter mutations. There is a high risk for progressive liver fibrosis, cirrhosis and HCC

5. HBsAg NEGATIVE PHASE:

In this phase there is loss of HBsAg and HBV DNA in the serum with the appearance of anti HBc with or without anti Hbs. Around 0.5% of the patients with chronic hepatitis can lose HBsAg per year

Most of the patients of chronic hepatitis B do not have history of prior acute hepatitis or jaundice in the past. However they may have intermittent episodes of fever myalgia , malaise, nausea due to the necroinflammatory activity in the liver during the process of immune clearance of the virus. Rest of the patients are diagnosed only after the development of decompensated liver disease or liver cirrhosis like ascitis, hepatic encephalopathy, upper gastro intestinal bleeding or persistent jaundice.

HEPATITIS C-GLOBAL EPIDEMIOLOGY[5]

WHO GLOBAL HEPATITIS REPORT -2017

- WHO estimates that 71million persons were living with HCV infection in the world, accounting for **1%** of the population.
- In 2015 there were **1.75 million** new HCV infections (globally, 2.37 new HCV infections per 100000 people).
- HCV infection is unevenly distributed in the world. The European and Eastern Mediterranean regions are more affected with a prevalence of

2.3% and **1.5%** respectively. Around **2.3 million** persons living with HIV also had HCV infection.

- Approximately **3.99 lakh** people die each year from hepatitis c, mostly from cirrhosis liver and hepato cellular carcinoma.

WHO estimates that in 2015, viral hepatitis was responsible for 1.34 million deaths. This number was comparable with the number of deaths from tuberculosis, but higher than the number of deaths from HIV. Left untreated, HBV and HCV infection can lead to cirrhosis (720 000 deaths) and hepatocellular carcinoma (470 000 deaths). These long-term complications are life-threatening and accounted for **96%** of the deaths due to viral hepatitis.

HCV IN INDIA[6]

In India the prevalence of hepatitis c is around **1%** of the general population of which 80% of the patients have detectable HCV RNA.

HCV-VIROLOGY[6]

HCV is a single stranded positive sense enveloped RNA virus measuring around 40nm-60nm in size. It belongs to the genus *Hepacivirus* in the *Flaviviridae* family. There are 6 genotypes 1to 6 .The most common genotype is India is genotype 3 followed by the genotype 1. Genotype 1, 2, 3 are more common in North America and Europe. In Africa the most common genotype

is genotype 4. Genotype 5 is seen mostly in South Africa and South East Asia while genotype 6 is seen mostly in South East Asia.

NATURAL HISTORY OF HEPATITIS C [6]

HCV infected individuals have an incubation period which ranges from15 days to 160 days with a mean of around 50 days.

The patient is usually asymptomatic in the acute phase of the illness .During this phase of illness the disease is usually unapparent with self limiting elevation of hepatic transaminases. However the patient can have nonspecific symptoms like nausea, malaise, myalgia etc which is seen in around 20-30% of the patients.

In patients who develop symptomatic acute infection with jaundice, there is a higher chance of spontaneous clearance. Spontaneous loss of HCV RNA can occur in these patients within 3 to 4 months.

Progression to chronicity occurs in around **70-85%** of the patients. Among them

20-30% at the infected individuals may develop cirhosis over a time period of 20 years

Once cirrhosis develops decompensated liver disease can occur at the rate of **3%** per year and hepatocellular carcinoma can develop at the rate of **1-4%** per year.

Liver damage in hepatitis C infection is immune mediated similar to that in hepatitis B infection. However in case of immunocompromised states like co infection with HIV or in the organ transplant recipients the patients may develop a syndrome called

' fibrosing cholestasis hepatitis'. This is due the direct cytopathic action of the HCV and is characterised by rapidly progressive disease with the development of jaundice, coagulopathy and hepatic encephalopathy.

MODE OF TRANSMISSION[1]

HIV, HBV AND HCV have **common mode of transmission like**

1. Materno-foetal transmission

2 .Sexual contact with an infected person

3**.** Sharing of contaminated needles, syringes or other injection drug equipment

4. Needle stick or other sharp instrument injuries.

5. Transfusion of blood and blood products

6. Organ recipients

PERSONS AT RISK WHO NEED TO BE SCREENED FOR HBV/HCV/HIV[1]

1. Infants born to the infected mothers

2. Sexual partners of the infected patients

3. Persons who have multiple sex partners

4. Persons with sexually transmitted diseases

5. Men who have sex with men

6. Household contacts of the infected persons

7. Health care and public safety workers who are exposed to blood and blood products

8. Patients on hemodialysis

9. Residents and staff of facilities for developmentally disabled persons

10. Persons who travel to regions with intermediate or high rates of the infection

11. Recipients of clotting factor concentrates before 1987

12. Recipients of unscreened blood or donated organs

13. HIV patients for HBV and HCV infections.

14. Pregnant women

15. Inmates of correctional facilities.

16. Persons who require immunosuppressive or cytotoxic drug therapy (including anti-tumour necrosis factor alpha therapy for rheumatologic and inflammatory bowel disorders.

INTRODUCTION ON HCV AND/OR HBV CO-INFECTION WITH HIV

The incidence of traditional HIV related opportunistic infections has declined with successful and effective anti retro viral therapy. Due to these reasons HBV and HCV related liver diseases are emerging as leading cause of morbidity and mortality in HIV infected individuals. Co-infection of HIV and/or HCV is common due to the similar modes of transmission of these diseases. HIV has impact on almost all phases of the natural history of hepatitis b and hepatitis c infection. This leads to

1. Persistence of infection[10, 11]

2. Higher level of serum HBV DNA[12, 13].

3. Lower rates of hepatitis B e antigen loss[15 ,16].

4. Increased incidence of liver cirhosis and other liver related mortality[12, 18]

5. Increased risk of hepatocellular carcinoma at lower cd4 T cell counts[19].

More over it has been found that there is a greater incidence of lamivudine resistant HBV in the patients who are positive for HIV. This has been explained due to the emergence of mutant strain of HBV and HCV when co-infected with HIV.

Hence wide research is needed in order to study the mechanism of co-infection, various characteristic pattern and presentation of co-infection and the nature of progression of liver disease among these patients. All these research will help out in arriving at optimal treatment plans and effective way of management of the diseases. Finally, it is also important that these researches are important to delineate the different patterns of resistance that emerge in this population and to understand the ways to minimize the development of antiviral resistance from long-term anti-HBV or HCV therapy

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HIV/HBV CO-INFECTION GLOBAL ESTIMATE[16]

About 1% of the persons living with HBV infection (2.7 million people)are also infected with HIV. Conversely, the global prevalence of HBV infection in HIV infected individuals is **7.4%.**Tenofovir, which is included in the treatment combinations recommended in first intention against HIV infection, is also active against HBV.

HIV/HCV CO-INFECTION –GLOBAL ESTIMATE[17]

HIV and HCV infections have overlapping mode of transmission and affected population. Globally there are 37 million people infected with HIV and around 115 million people with HCV infection. WHO sponsored a study on this in collaboration with the London school of Hygiene and Tropical medicine and the University of Bristol and published online in the Lancet Infectious disease on December 4 2016.The study shows that HCV infection is found with maximum prevalence among the people who inject drugs which was around 80% .Hence there is a need to plan routine tests to diagnose HCV infection in HIV programmes worldwide especially among the high risk groups.

As estimated around 2.3 million people living with HIV are co-infected with HCV globally, of these more than half or 1.3million are people who inject drugs. The study also concluded that HIV infected patients are on an average 6 times more likely than the HIV uninfected people to have HCV infection, pointing to a need to improve integrated HIV/HCV status.

PREVALENCE OF CO-INFECTION- VARIATION WITH ENDEMICITY AND MODE OF TRANSMISSION

1. The prevalence of co-infection of HBV and /or HCV among the patients with HIV varies globally depending upon the **endemicity** of the disease based on low risk, intermediate risk or high risk distribution of the disease. Thus the prevalence of co-infection in high endemicity countries is greater upto **20%,**

however on the low endemicity countries like the Western Europe and united state is estimated to be around **5-7%.**

Review Of Journal

Lukman femi owolabi et al[18] has described in their study on 'the prevalence and burden of HIV and hepatitis B co-infection' with data from seventeen states in **Nigeria** that the overall prevalence of HIV/HBV co-infection was found to be 17% in adults

Naval Chandra et al [19]conducted a similar study in around 120 HIV patients in **South India** and estimated the prevalence of HBV and HCV to be 15% and 8% respectively.

Prakash khunte et al[20] conducted a study on the prevalence of HBV /HCV co-infection among patients infected with HIV among the Tribals from central India at the govt hospital at Chattisgarh and found the prevalence of HIV/HBV to be 6%, HIV/HCV to be 2% and the prevalence of HIV/HCV/HBV was found to be 1%.

2. The prevalence also varies with **the route of transmission of the disease**. It is known that in countries which are endemic to HBV infection perinatal transmission is the major route of transmission. Similarly the prevalence of HCV co-infection among HIV positive individuals were found to be high among the intravenous drug abusers in Manipur.

NATURAL HISTORY OF HBV CO-INFECTION IN HIV PATIENTS

1. HIV adversely affects all phases of the natural history of adult-acquired hepatitis B and hepatitis C. Thus the HIV-infected individuals are up to six-fold more likely to develop chronic hepatitis B than are HIV-negative individuals on co infection with either HBV OR HCV
2. **Bodsworth et al**. retrospectively studied 77 men who acquired HBV infection. Of them 31 were positive for HIV prior to HBV infection and .10 Of the HIV-infected men, almost 23% of the patients developed chronic hepatitis B compared to 4% of the HIV-uninfected men. Further, the meanCD4_
3. T cell counts were lower in the HIV-infected men who developed chronic hepatitis B compared to the HIV-infected men who did not become chronically infected.
4. HIV infection decreases the rate of clearance of the hepatitis B e antigen (HBeAg) up to five-fold and increases the level of HBV replication .This is manifested by higher HBVDNA levels in the serum.
5. HIV-infected individuals who acquire protective antibody to hepatitis B surface antigen (anti-HBs) may lose the anti-HBs antibody. This can result in the reactivation of HBV (reverse seroconversion).

6. HIV accelerates the progression of HBV-related liver disease. Cirrhosis is more common in HIV-HBV co-infection. and this may be related to lower CD4_T cell counts.
7. **Thio et al[46]** in his study found the liver-related mortality in an analysis of 5293 HIV positive men of whom 326 were positive for hepatitis B surface antigen (HBsAg).
8. The HIV-HBV co-infected men are over 17 times more likely to die of liver related causes when compared to those mono infected with HBV.
9. There is an evidence that lower CD4_ T cell counts are associated with increased risk for Hepatocellular carcinoma among the HIV/HBV co-infected individuals, but however it is not known whether HIV in general increases the risk.
10. In HIV/HBV co-infection, flares of elevated transaminases may result from immune reconstitution, adverse reactions to antiretroviral agents, discontinuation of agents with anti-HBV activity, and emergence of resistance.

IMPACT OF HIV INFECTION ON HCV DISEASE PROGRESSION

1. Several studies have demonstrated that patients co-infected with HCV and HIV have more rapid fibrosis progression than monoinfected patients, even after taking into account age, sex and alcohol consumption[21].

2. People with HCV/HIV co-infection may have quantitative and/or qualitative deficiency in their immune responses to HCV. HIV accelerates the course of HCV-associated liver disease, particularly in patients who are more severely immune deficient, by increasing the HCV viraemia level from two- to eightfold, resulting in a significant decrease in spontaneous recovery from acute hepatitis [22]
3. The risk of mother-to-child and sexual transmission (from averages of 6% to 20% and from 0% to 3%, respectively); and rates of liver fibrosis (two- to fivefold), cirrhosis, decompensation, hepatocellular carcinoma (HCC) and liver-related mortality [23]
4. Liver disease is the leading cause of morbidity and mortality in HCV/HIV-co-infected patients, despite the suggestion that HAART, especially protease inhibitors, may decrease the severity of liver disease and the related mortality[30].
5. Comorbidities with hepatic consequences (drug hepatotoxicity, HBV, steatosis, alcohol or drug abuse) are frequent in co-infected patients and may increase the rate of complications associated with HCV-related liver disease.
6. Patients with CD4 <200 cells/mm3 are those most likely to progress to severe liver disease [21, 53, 24]. For example, HIV-infected patients with CD4 <200 cells/mm3 who drink more than 50 g of alcohol daily have a median expected time to cirrhosis of 16 years, versus 36 years for HIV-infected

patients with CD4 >200 cells/mm3 who drink 50 g or less of alcohol daily[24].

7. Spontaneous clearance of HCV is significantly lower in HIV-infected patients than in immunocompetent patients with acute hepatitis. As HCV ribonucleic acid (RNA) might become temporarily undetectable during the acute phase of HCV infection, clearance must be confirmed with a sensitive qualitative HCV RNA assay on at least two occasions six months apart [25,26].
8. In profoundly immunosuppressed patients, HCV serology has occasionally been found to be falsely negative despite HCV chronic infection. Such false negatives have become very rare due to the high sensitivity of third-generation serology[25, 26].

IMPACT OF HCV INFECTION ON HIV DISEASE PROGRESSION

1. HCV has little or no effect on the response to ART or on the immunological, virological or HIV-related clinical disease progression. Although HCV antibodies per se do not influence progression, infection with certain multiple genotypes might do so[27].

2. Extended follow-up in various studies indicate that patients on HAART do not have any major differences in HIV-related mortality from HCV/HIV-co-infected patients or those infected with HIV alone, particularly if ART is given[29]. There is, however, an increased risk for liver disease-related

morbidity and mortality in hepatitis co-infected HIV, as well as more hepatotoxicity under ART regimens[28].

CO-INFECTION AND CD4 TCELL COUNT

The CD4 count gives an estimation of the immune status of the patient. Studies have shown that the CD4 T cells were lower among the HIV patients co-infected with HBV/HCV indicating the immune suppressed state of co-infected conditions. The mechanism proposed was that HIV/HCV infections can promote HIV replication in the CD4 T cells thereby increasing their destruction eventually leading to lower CD4 counts.

1. Alo M et al.[31] in their study conducted at sokoto state of Nigeria found that Out of the 88 HIV positive subjects with concomitant HBV infection, 56 had a CD4 + T cell count of ≤350cells/μL, while 32 subjects had a CD4 + T cell count of ≥350cells/μL. This amounts to a ratio of 7:4, hence most of them with concomitant HIV/HBV infection had CD4 cells below the baseline count of 350cells/μL . This may be an indication that HBV infection aggravates the propensity of the pathogenesis of AIDS in HIV infected persons as CD4 count is directly proportional to the level of immunosuppression.

2. This is comparable to the study by **Mayaphi and colleagues**[32] which observed that an increased HBV prevalence in HIV patients with CD4 count of ≤100cells/μL had a major risk factor of increased HBV replication.

HIV AND LIVER INJURY

Abnormalities of LFT ARE common in HIV/AIDS patients in developed countries. Studies show that these abnormalities may be due to

1. Direct inflammation induced by the HIV virus on the liver cell.

2. Due to gall bladder disease and infection with bacterial, viral or other opportunistic agents

3. HIV can also infect the hepatic or kupffer cells that may further contribute to the development of liver fibrosis and raised liver enzyme levels

It is therefore important to characterise the nature of this abnormality and to institute appropriate management. However, more studies are required in this field of HIV related liver disease.

HIV patients without HCV/Hepatitis B Virus (HBV) and without primary immunodeficiency are independently associated with mild to moderate elevations in both Aspartate transaminase (AST) and Alanine transaminase (ALT). Elevated AST and ALT levels are one of the clinical manifestations, but is infrequently reported in the literature and elevated levels may be an initial manifestation of primary HIV infection and is more common than expected.

Primary HIV-1 infection will serve as one of the differential diagnosis to be considered in young men presenting with unexplained, new-onset liver function impairment [32]

There is an association between HIV viral load and aminotransferases as markers of hepatic damage leading to improved recognition, diagnosis and potential therapy of hepatic damage in HIV infected patients [33]

Liver disease in HIV infected individuals encompasses the spectrum from abnormal LFTs, liver decompensation, with and without evidence of cirrhosis on biopsy, to Non-Alcoholic Liver Disease (NALD) and in its more severe form, Non-Alcoholic Steatohepatitis (NASH) and hepatocellular cancer (HCC).

HIV is a cytopathic virus and can directly infect multiple cells in the liver, leading to enhanced intrahepatic apoptosis, activation and fibrosis. HIV can also alter gastro-intestinal tract permeability, leading to increased levels of circulating lipopolysaccharide that may have an impact on liver function[34]

HBV AND LIVER INJURY

HBV is not directly cytopathic to liver cells as in case of the HIV virus. However hepatic necrosis is mediated by Th1 lymphocyte induced cytotoxic T lymphocytes (CTL). Therefore any process that affects quantity and quality

of immune response will have a bearing on the outcome of liver damage in HBV infection

HIV WITH HCV AND/OR HBV CO-INFECTION AND LIVER INJURY

A more robust immune restoration was observed among HBV/HCV co-infected subjects who developed liver enzyme elevation after antiretroviral (ARV) initiation compared with other groups. This finding suggests that ART-related liver enzyme elevation may be related in part to immune reconstitution, as measured by changes in CD4 T-cell counts [35]

Although some studies explain Increased levels of ALT and AST associated with HBV/HIV co-infection status, the refined explanation is that , LFT abnormalities and fibrosis scores were only significantly higher in co-infected patients in the immune clearance and Hepatitis B surface antigen (HbsAg) negative chronic hepatitis phases

Thus in order to develop elevated liver enzymes in case of co-infection there should be efficient functioning of the immune system [36]

Hence, liver function test can be an important predictor for HBV/HIV co-infection and so screening for HBV/HCV coinfection in HIV-positive patients is necessary [37]

.

MATERIALS AND METHODS

PLACE OF STUDY:

Thanjavur Medical College and hospital Thanjavur-613004

TYPE OF STUDY: Cross sectional study

DURATION OF STUDY: March 2017- August 20017

COLLABERATING DEPARTMENTS:

The ART centre, Dept of Biochemistry,Dept. of Microbiology.

STUDY POPULATION:

It included 159 HIV positive adult patients who were on anti retro viral treatment and were managed in the ART outpatient clinic. HIV positive cases include the patients who were positive for HIV antibody by ELISA which was further confirmed by the western blot method .

INCLUSION CRITERIA:

1. All HIV positive patients on ART
2. Patients above 12 years of age
3. Both male and female patients were included in the study

EXCLUTION CRITERIA:

1. Patients below 12 years of age

2. Patients with obesity, Diabetes mellitus, systemic hypertension/metabolic syndrome.

3. Patients on chronic hepatotoxic drug intake other than ART.

4. Refusal to give consent.

DATA COLLECTION:

1.Demographic data:

Name, age, sex, occupation, education, residential address were collected

2.History:

Detailed history was collected from patients by subjecting them to a questionnaire to assess the **mode of transmissi**on like history regarding sexual exposures, use of intravenous drugs, sharing of needles, blood transfusion, organ transplantation/IUI etc.

Further **treatment history** such as details about the date of start of ART, type of ART regimen, duration of treatment, h/o recent vaccination for hepatitis B were obtained.,

In order to assess the **risk of hepatic injury** h/o alcohol intake with duration, h/o blood transfusion and h/o any other chronic drug intake were also assessed. The possible mode of transmission among these patients were classified as

1. Heterosexual
2. Homosexual
3. IUD/ Needle stick injury
4. IUI/Organ transplantation
5. Parent to child transmission.
6. Unknown.

3.Clinical Examination:

- The patients were meticulously examined for height and weight to assess the BMI, skin and mucosal lesions and any opportunistic infections.
- Based on the clinical examination the patients were grouped into the different **WHO clinical stages** such as 1, 2, 3,and 4. The Criteria for this classification is as discussed in the page 14
- **BMI**: Body mass index was calculated from the patients Height and Weight using the formula

BMI=WEIGHT in Kg/HEIGHT in meter^2

BMI was further classified as follows

<19.5 –under weight

19.5-25-normal weight

25-30-over weight

>30-0besity

4. Cd4 Count Assay:

The standard method for counting the CD4 T cells is by using a **Flow cytometer**. Computer can calculate the number Of CD4 T cells by analysing the size of the cells and type of the antibodies these cells have been tagged with. The overall process is called **Fluorescence Activated Cell Sorting (FACS) .**

5. Cllection Of Blood Sample:

1**.** Around 10 ml of venous blood sample was collected using plain and EDTA vaccutainer tubes (5ml in each tube) for the determination of **Liver fuction tests** , HBsAg, Anti HCV and CD4 Tcell count respectively.

2. The blood sample in the plain tube was centrifuged at 3000 rpm for 5 min to separate the serum which was used for the determination of liver enzymes , bilirubin levels and Total protein .

3. The remaining serum was kept in deep refrigerator (-40 degree celcius) for detection of **HBsAg** and **Anti-HCV** by **ELISA.**

4**.**The sample in the EDTA tube was used for **CD4 Tcell** level determinate

DEFINITIONS.

(i) HBV/HIV coinfection was defined by a positive HBV surface antigen (HBsAg) in HIV positive cases.

(ii) HCV/HIV coinfection was defined by a positive HCV antibody in HIV positive cases.

(iii) Prevalence of HBV coinfection, HCV coinfection, and HBV/HCV coinfection was calculated for those with recorded test results for HBsAg and HCV antibody.

ETHICAL CLEARANCE:

The study was conducted after obtaining ethical clearance certicate from the ethical committee of Thanjavur medical college .

DATA ANALYSIS

The data was entered and analysed using Graph Pad Prism version 5 software .

The Mann Whitney U test and Unpaired T test were used to calculate the statistical significance. P value of< 0.05 was considered as statistically significant.

OBSERVATION

TABLE 1: DISTRIBUTION OF AGE IN YEARS IN THE STUDY POPULATION

S. No	Age (in years)	Number (n)	Frequency (%)
1	18 – 30	20	12.5
2	30 to 50	106	66.7
3	>50	33	20.8

The total No of HIV positive cases in the study population is 159.

The mean age is 42.4 years with the standard deviation of 10.6.

The minimum age is 16 years and the maximum age is 70 years.

Around 66.7% of the study population is between the age group of 30-50 years of age.

TABLE 2: DISTRIBUTION OF GENDER IN THE STUDY POPULATION

S. No	Age (in years)	Male		Female	
		(n)	(%)	(n)	(%)
1	Overall (n=159)	85	53.5	74	46.5
2	16 – 30 (n=20)	8	9.4	12	16.2
3	>30 to 50 (n=106)	55	64.7	51	68.9
4	>50 (n=33)	22	25.9	11	14.9

Total no of HIV positive cases: 159

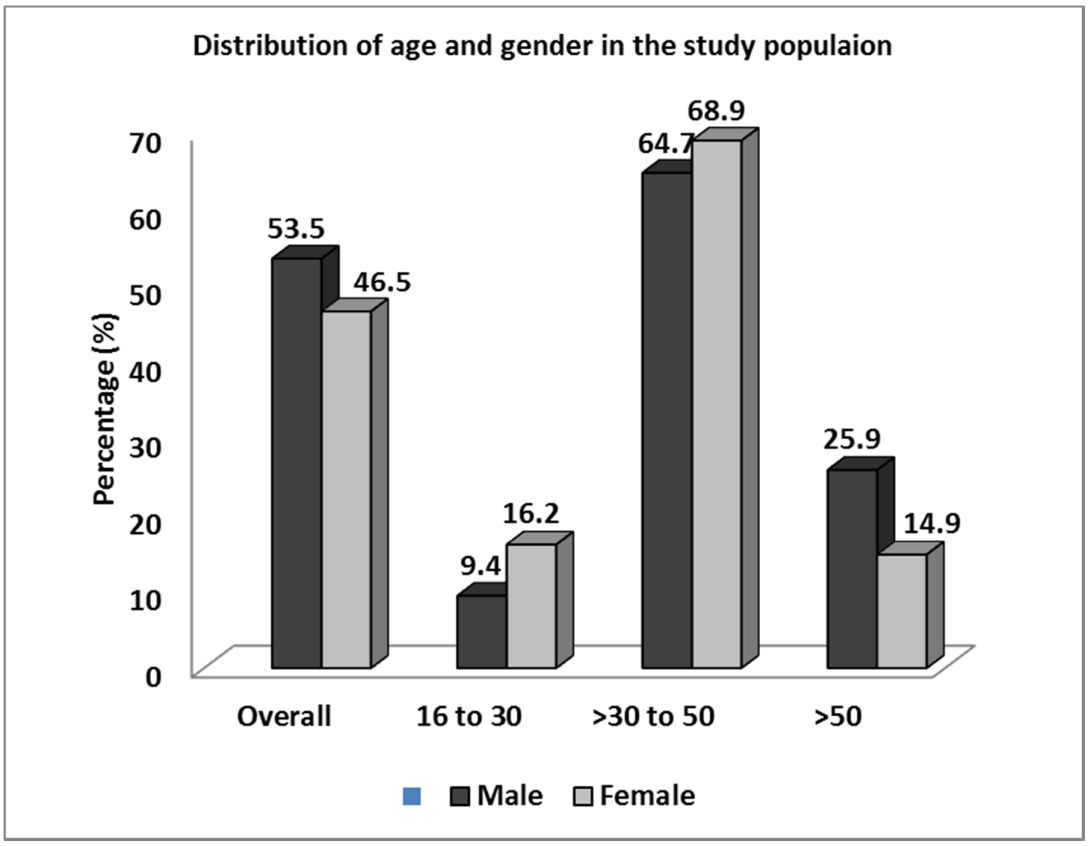
Total no of males: 85(53.5%)

Total no of female: 74(46.5%)

Male to female ratio:

Majority of the study population lies in the age group of 30-50 yrs of age in both males(64.7%) and females(68.9%)

FIGURE 4: DISTRIBUTION OF AGE AND GENDER IN THE STUDY POPULATION



The figure shows that,

Among the total study population males **(53.5%)** > females **(46.5%)**

Age wise distribution shows that majority of the HIV Positive cases were distributed among the age group of 30-50 yrs of age.

TABLE 3: DISTRIBUTION OF MODE OF TRANSMISSION OF HIV IN THE STUDY POPULATION

S. No	Mode of transmission of HIV	Overall N (%)	Male (n=85)		Female (n=74)	
			(n)	(%)	(n)	(%)
1	Heterosexual	153 (96.2)	82	96.5	71	96
2	Homosexual	2 (1.3)	2	2.4	0	0
3	IUD/Needle stick injury	0(0)	0	0	0	0
4	Organ transplantation or intrauterine insemination	1(0.6)	0	0	1	1.3
5	Parent to child	3 (1.9)	1	1.2	2	2.7

The Commonest mode of transmission is **Heterosexual** mode of transmission

The mode of transmission in the increasing order of frequency is

- Heterosexual- 96.2%
- Parent to child-1.9%
- Homosexual-1.3%
- IUI-0.6%

FIG: 5 DISTRIBUTION OF MODE OF TRANSMISSION OF HIV IN THE STUDY POPULATION

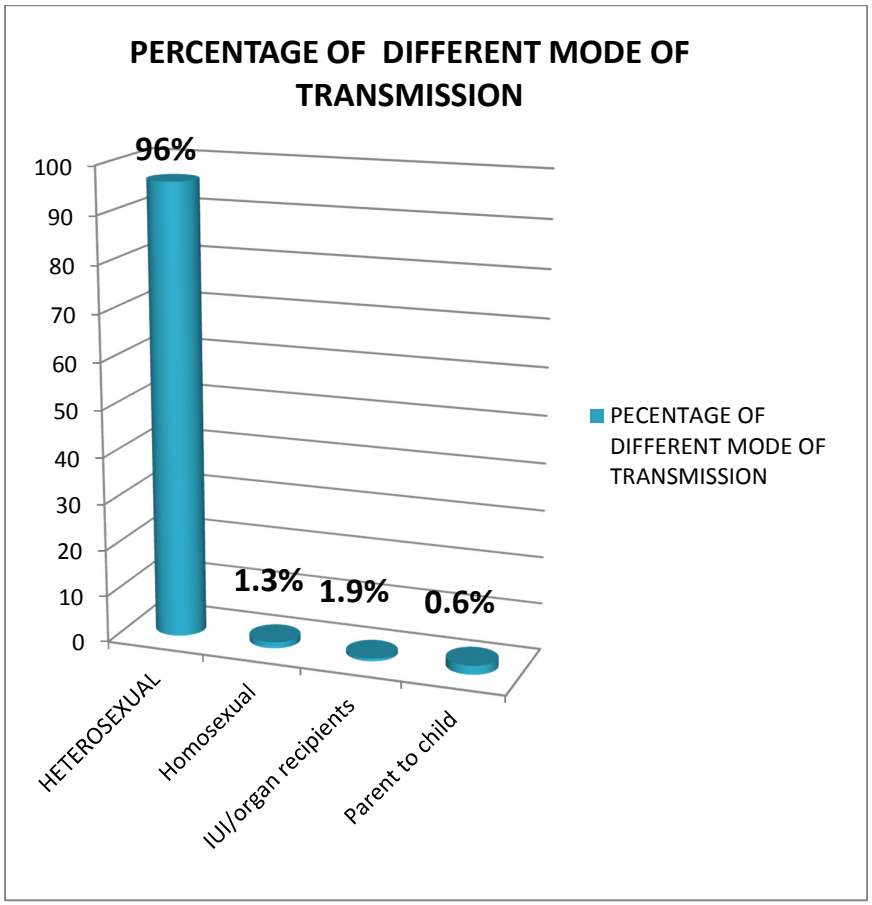


TABLE 4: DISTRIBUTION OF TYPE OF ART REGIMEN IN THE STUDY POPULATION.

S. No	Type of ART regimen	Number (n)	Frequency (%)
1	ZLN	69	43.4
2	TLE	72	45.3
3	TLN	9	5.7
4	ZLE	6	3.8
5	Others (TL/AR, TL/L/R)	2	1.2
6	No regimen	1	0.6

The distribution according to the type of ART regimen in the increasing order of frequency is as follows

TLE- 72(45.3%)

ZLN-69(43.4%)

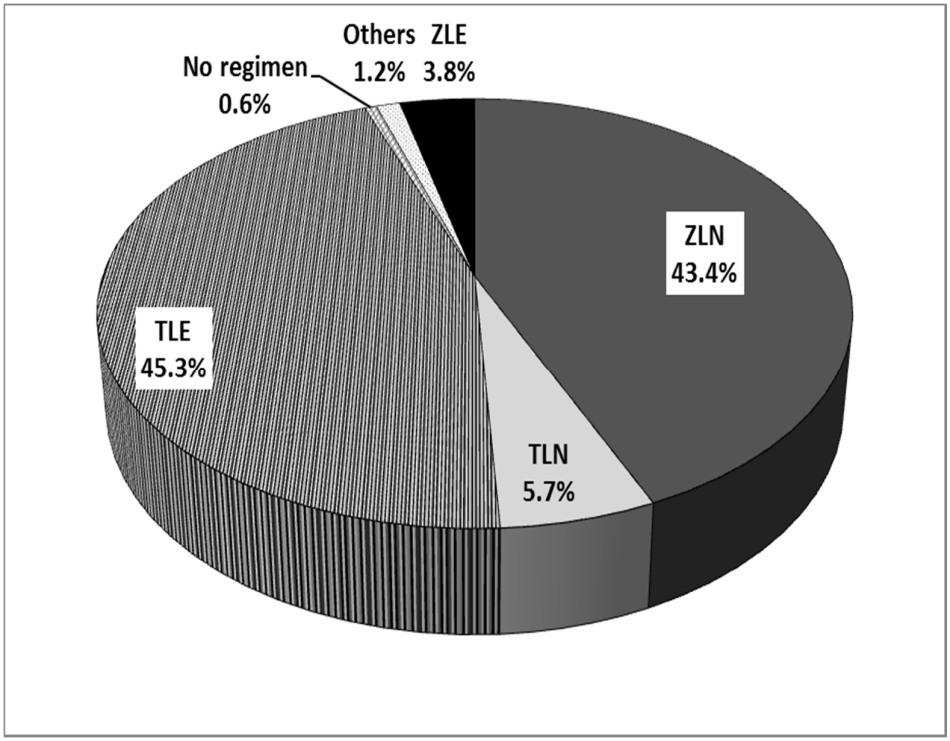
TLN- 9(5.7%)

ZLE- 6(3.8%)

OTHERS- 2(1.2%)

NO REGIMEN-1(0.6%)

FIG: 6 DISTRIBUTION OF TYPE OF ART REGIMEN GIVEN FOR THE STUDY POPULATION



The distribution of different types of ART regimen among HIV patients

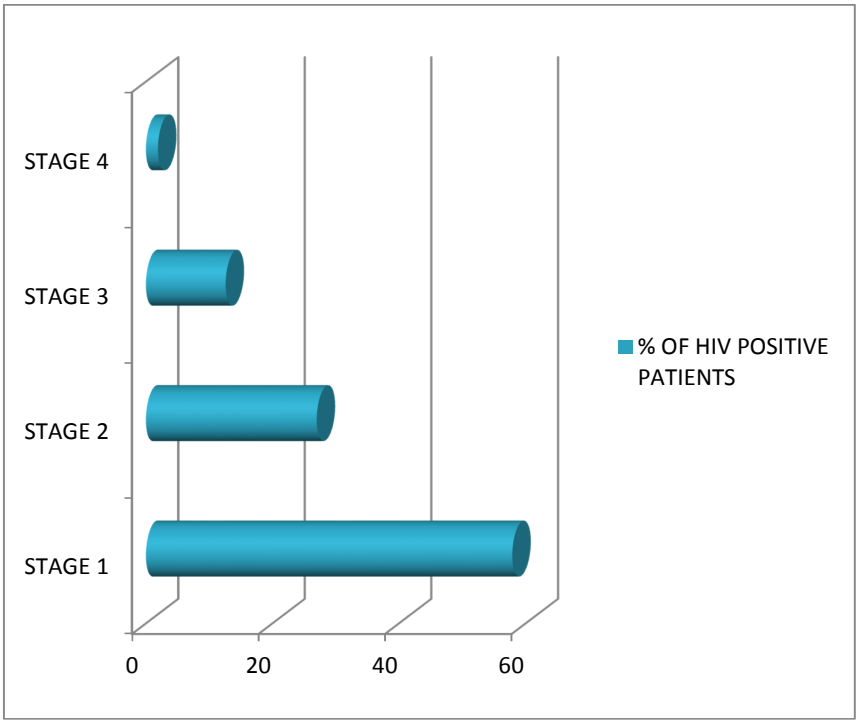
- TLE-72(45.3%)
- TLN-9(5.7%)
- ZLE-6(3.8%)
- OTHERS-2(1.2%)
- NO REGIMEN-1(0.6%)

TABLE 5: DISTRIBUTION OF HIV POSITIVE PATIENTS BASED ON THE WHO CLINICAL STAGING

S. No	Type of clinical staging	Number (n)	Frequency (%)
1	Stage 1	92	57.9
2	Stage 2	43	27
3	Stage 3	20	12.6
4	Stage 4	3	1.9
5	Not determined	1	0.6

Among the HIV positive patients **ALMOST 57.9 %** OF the patients were in the stage 1 of the WHO clinical staging, and only 1.9% of the patients were in stage 4.

FIG:7 DISTRIBUTION OF HIV POSITIVE PATIENTS AS PER THE WHO CLINICAL STAGING



Maximum no of HIV patients on ART were in stage 1 of WHO CLINICAL STAGE 1.

TABLE:6 FREQUENCY DISTRIBUTION OF THE PATIENTS IN THE STUDY POPULATION BASED ON BMI

S. No	Range of the BMI (Kg/m^2)	Number (n)	Frequency (%)
1	<19.5	56	35.2
2	19.51 to 25	100	62.9
3	25.01 to 30	3	1.9
4	30.01 to 35	0	0
5	>35	0	0

- Underweight- 56 **(35.2%)**
- Normal weight- 100**(62.9)**
- Overweight-3 **(1.9%)**
- Obesity-0 **(0%)**

FIG: 8 FREQUENCY DISTRIBUTION OF HIV PATIENTS IN THE STUDY BASED ON BMI.

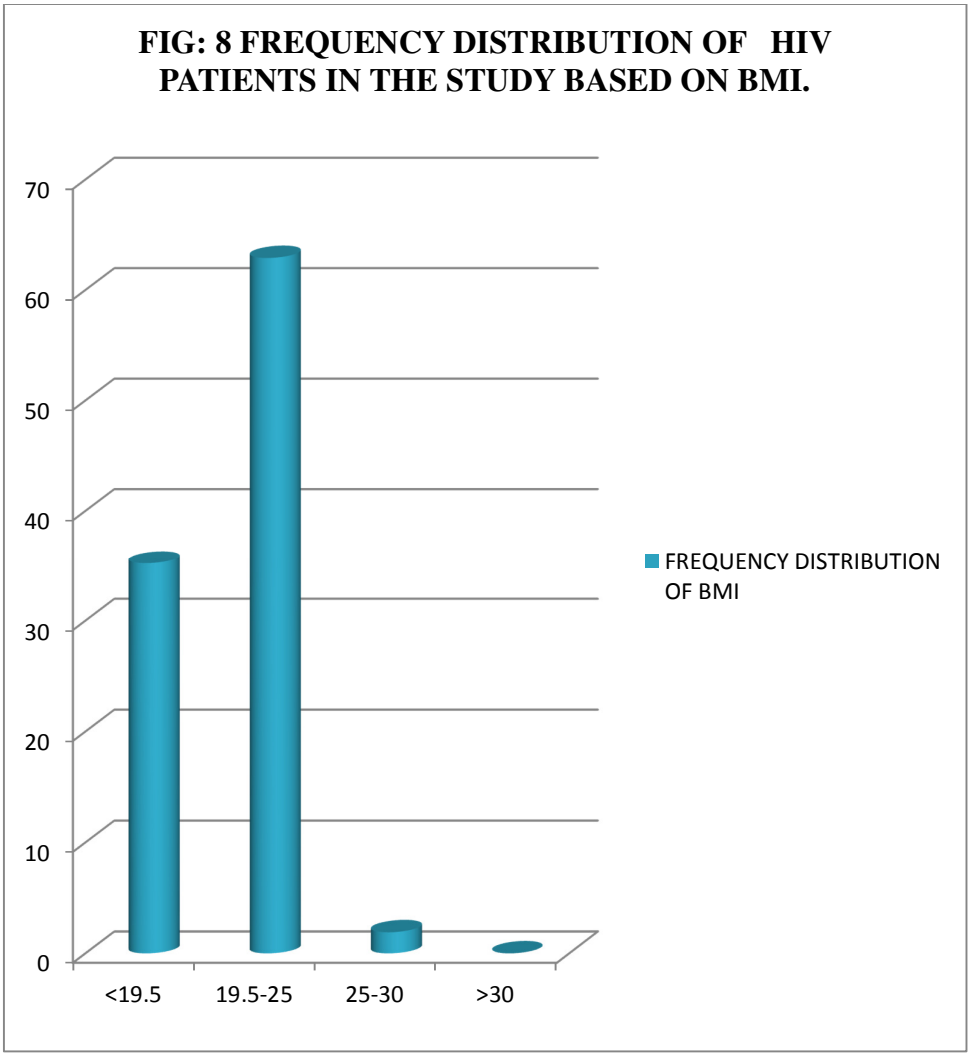


TABLE 7: PREVALENCE OF HEPATITIS B AND/OR C INFECTION AMONG DIFFERENT GROUPS OF THE STUDY POPULATION.

S. No	Type of infection	Overall (n=159)		Male (n=85)		Female (n=74)		Alcoholic males (n=49)		Non alcoholic males (n=36)	
		n	%	n	%	n	%	N	%	N	%
1	HIV alone	149	93.7	80	94.1	64	86.4	45	91.8	35	97.2
2	HIV with co-infection (HIV with Hep B or HIV with Hep C or both)	10	6.28	5	5.88	5	6.75	4	8.16	1	2.77
3	Hep B alone	8	5.03	3	3.52	5	6.75	3	6.12	0	0
4	Hep C alone	2	1.25	2	2.35	0	0	1	2.04	1	2.77

Prevalence of Hepatitis B among the HIV patients is **5.03%**

Prevalence of Hepatitis C among the HIV positive individuals is **1.25%**

Prevalence of HIV positive patients co-infected with HBV OR HCV is **6.28%**

FIG: 9 PREVALENCE OF CO-INFECTION AND MONOINFECTION AMONG THE STUDY POPULATION

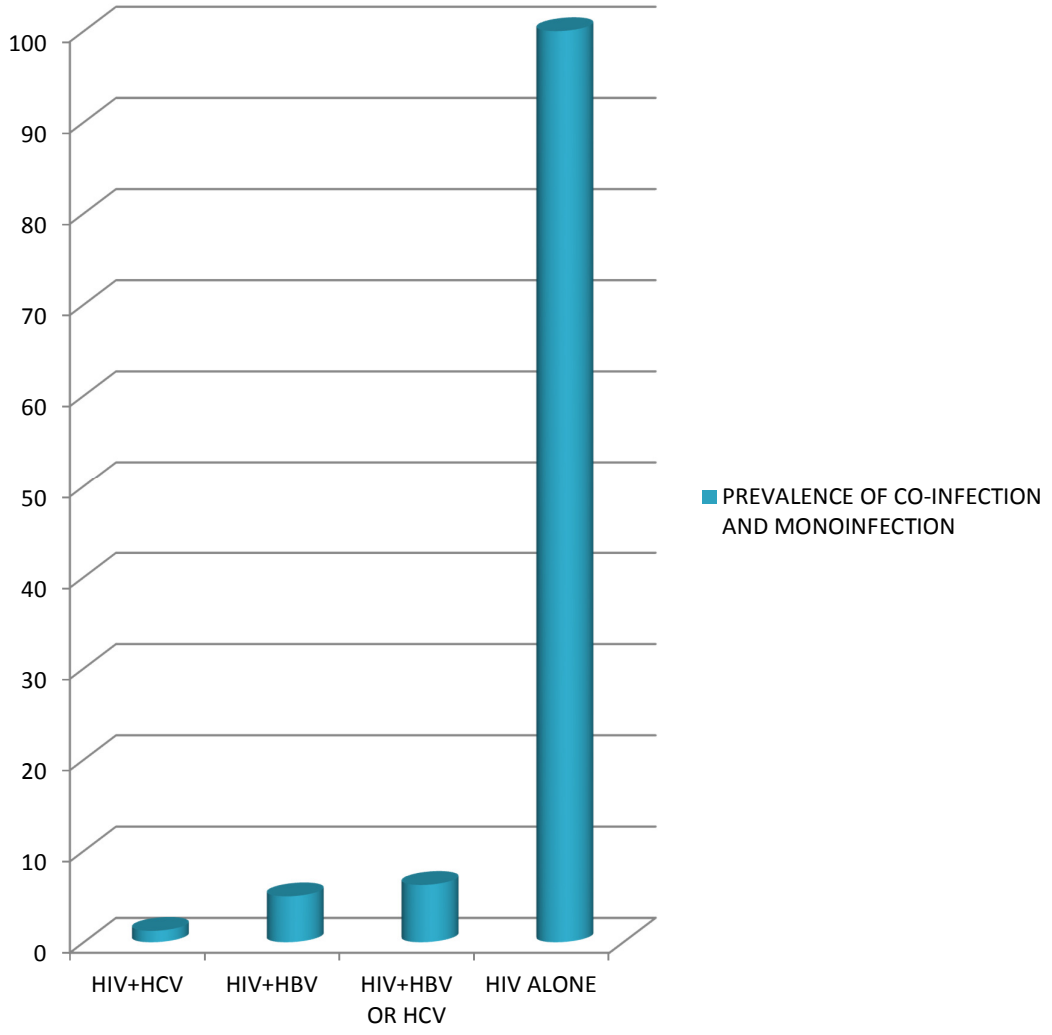


TABLE: 8 FREQUENCY DISTRIBUTION OF PATIENTS WITH BLOOD TRANSFUSION BETWEEN MONO INFECTED AND CO-INFECTED PATIENTS IN THE STUDY POPULATION

S. No	Nature of blood transfusion	Overall (n=159)		Infected with HIV alone (n=149)		HIV with co-infection (n=10)	
		(n)	(%)	(n)	(%)	(n)	(%)
1	Yes	10	6.28	10	6.71	0	0
2	No	148	93.08	138	92.61	10	100
3	Not known	1	0.64	1	0.67	0	0

Overall around 10 (**6.71%**) of the HIV positive patients in the study population have history of blood tansfusion in the past.

However, the HIV positive patients who were coinfected with either HBV or HCV did not have any history of blood transfusion in the past.

TABLE:10 DISTRIBUTION OF LEVEL OF CD4 COUNT AMONG THE PATIENTS IN THE STUDY POPULATION

S. No	CD4 count (per mm^3)	Overall (n=159)		Infected with HIV alone (n=149)		HIV with co-infection (n=10)	
		(n)	(%)	(n)	(%)	(n)	(%)
1	<200	17	10.7	15	10.1	2	20
2	201 to 500	55	34.6	47	31.5	8	80
3	>500	87	54.7	87	58.4	0	0

In patients monoinfected with HIV

- **58%** othe patients have CD4 Tcell counts >500
- **31.5%** of the patients have CD4 counts between 201-500

However among the Coinfected patiens

- **80%** of the patients have their CD4 Tcell count between 201-500
- **20%** of them have CD4 counts between <200
- **None of the patients** with co infection had a CD4 count >500 in our study**.**

TABLE :10 MEAN CD4 COUNT IN RELATION TO GENDER

AMONG THE MONOINFECTED AND COINFETED PATIENTS

S.No	CD4 count – Type of Parameter	Patient with only HIV infection (n=149)		Patient with HIV and co-infection (n=10)		P value	Statistical test used
		Mean	SD	Mean	SD		
1	Male Gender	527.5	308	298	133	0.08 (NS)	Mann Whitney U test
2	Female Gender	659.1	311.9	265	113.5	<0.0001*	Mann Whitney U test

* Indicates p value <0.0001 (Extremely significant)

The mean CD4 counts of the male and female monoinfected patients were **527.5** and **656.1** respectively .This was higher than the mean CD4 counts of the male and female co-infected patients which were **298** and **265** respectively

The difference in CD4 count was statistically significant among the females but not among the males.

Similarly, the overall mean CD4 count was higher among the monoinfected individuals but the difference was not statistically significant.

FIG: 10 DIFFERENCE IN THE MEAN CD4 COUNTS BETWEEN MALE AND FEMALE PATIENTS OF THE MONOINFECTED AND CO-INFECTED HIV PATIENTS.

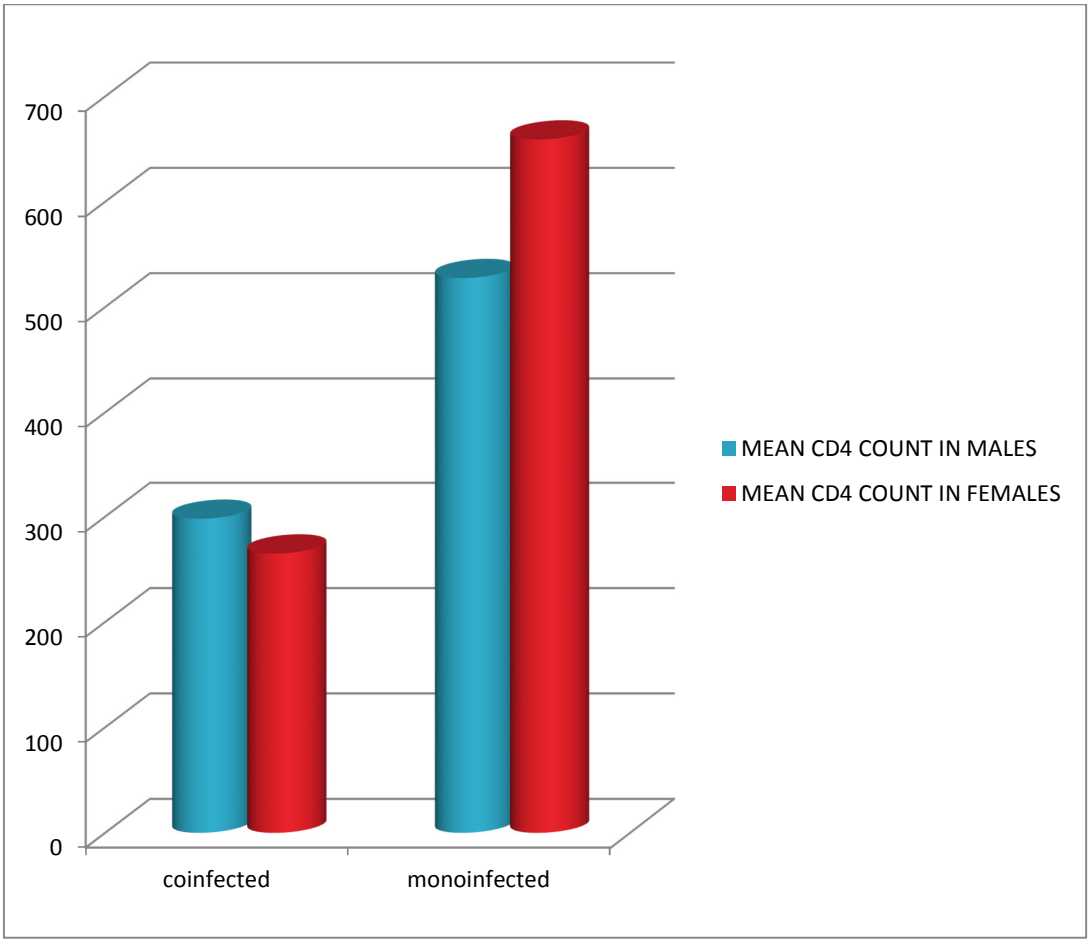


TABLE: 11 COMPARISON OF THE MEAN BMI BETWEEN THE PATIENT WITH HIV MONO INFECTION AND HIV CO INFECTION IN THE STUDY POPULATION

S. No	Name of the parameter	Patient with only HIV infection (n=149)		Patient with HIV and co-infection (n=10)		P value	Statistical test used
		Mean	SD	Mean	SD		
1	BMI (Kg/m^2)	21.03	3.3	18.1	2.53	0.0007*	Unpaired T test

* Indicates p value <0.01 (Highly significant)

The mean BMI in HIV monoinfected -**18.1**-underweight

The mean BMI in co-infected HIV patients-**21.3**-normal weight

There is statistically significant difference in BMI among the HIV monoinfected and the co-infected individuals.

FIG: 11 COMPARISON OF THE MEAN BMI BETWEEN THE CO-INFECTED AND MONO INFECTED PATIENTS

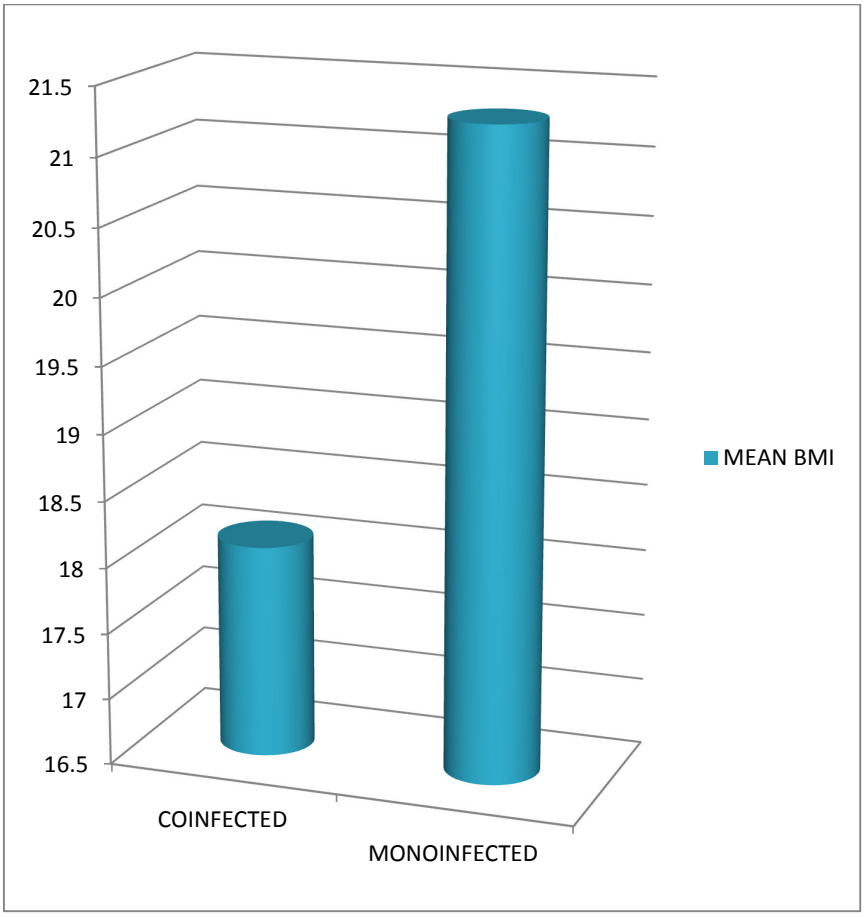


TABLE: 12 COMPARISON OF DIFFERENT LIVER ENZYME LEVELS BETWEEN THE MONOINFECTED AND COINFECTED PATIENTS

S. No	Name of the liver function test	Patient with only HIV infection (n=149)		Patient with HIV and co-infection (n=10)		P value	Statistical test used
		Mean	SD	Mean	SD		
1	SGOT (IU/mm^3)	28.37	16.1	88.4	111.1	<0.0001**	Mann Whitney U test
2	SGPT (IU/mm^3)	29.5	17.4	83.1	62.3	<0.0001**	Mann Whitney U test
3	ALP (IU/mm^3)	69.1	27.6	84.4	24.8	0.04*	Mann Whitney U test

* indicates p value <0.05 (significant) and ** indicates p<0.0001 (extremely significant)

1.The mean SGOT,SGPT, and ALP were relatively higher among the co-infected patients when compared with that of the monoinfected patients and this was statistically significant .

FIG: 12 COMPARISON OF THE MEAN SGOT,SGPT AND ALP BETWEEN THE COINFECTED AND MONOINFECTED STUDY POPULATION

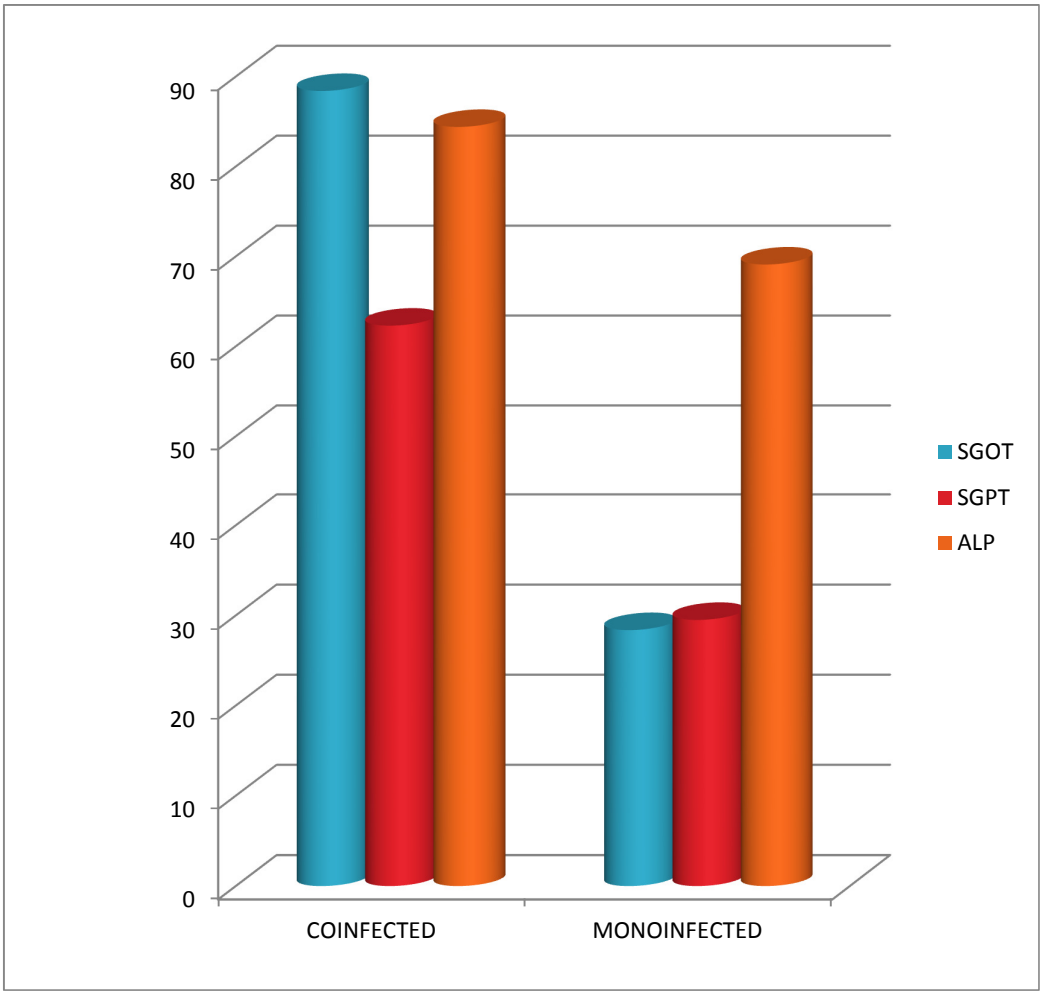


TABLE : 13 COMPARISION OF PROPORTION OF PATIENTS WITH ABNORMAL LIVER ENZYMES BETWEEN THE MONOINFECTED AND CO-INFECTED PATIENTS IN THE STUDY POPULATION

S. No	Name of the liver function test	Overall (n=159)		Infected with HIV alone (n=149)		HIV with co-infection (n=10)	
		(n)	(%)	(n)	(%)	(n)	(%)
1	SGOT (IU/mm^3)	32	20.1	24	16.1	8	80
2	SGPT (IU/mm^3)	33	20.7	23	15.4	10	100
3	ALP (IU/mm^3)	10	6.2	7	4.7	3	30

SGOT- was abnormally elevated in **80%** of the co-infected patients but only in **16.1%** of the mono infected patients

SGPT- was abnormal in **100%** of the co-infected patients and only in **15.4%** of the mono infected patients

ALP-was abnormal in **30%** of the co-infected patients but only in **4.7%** of the mono infected patients

FIG: 13 PROPORTION OF PATIENTS WITH ELEVATED LIVER ENZYMES AMONG THE MOINFECTED AND COINFECTED CATEGORY.

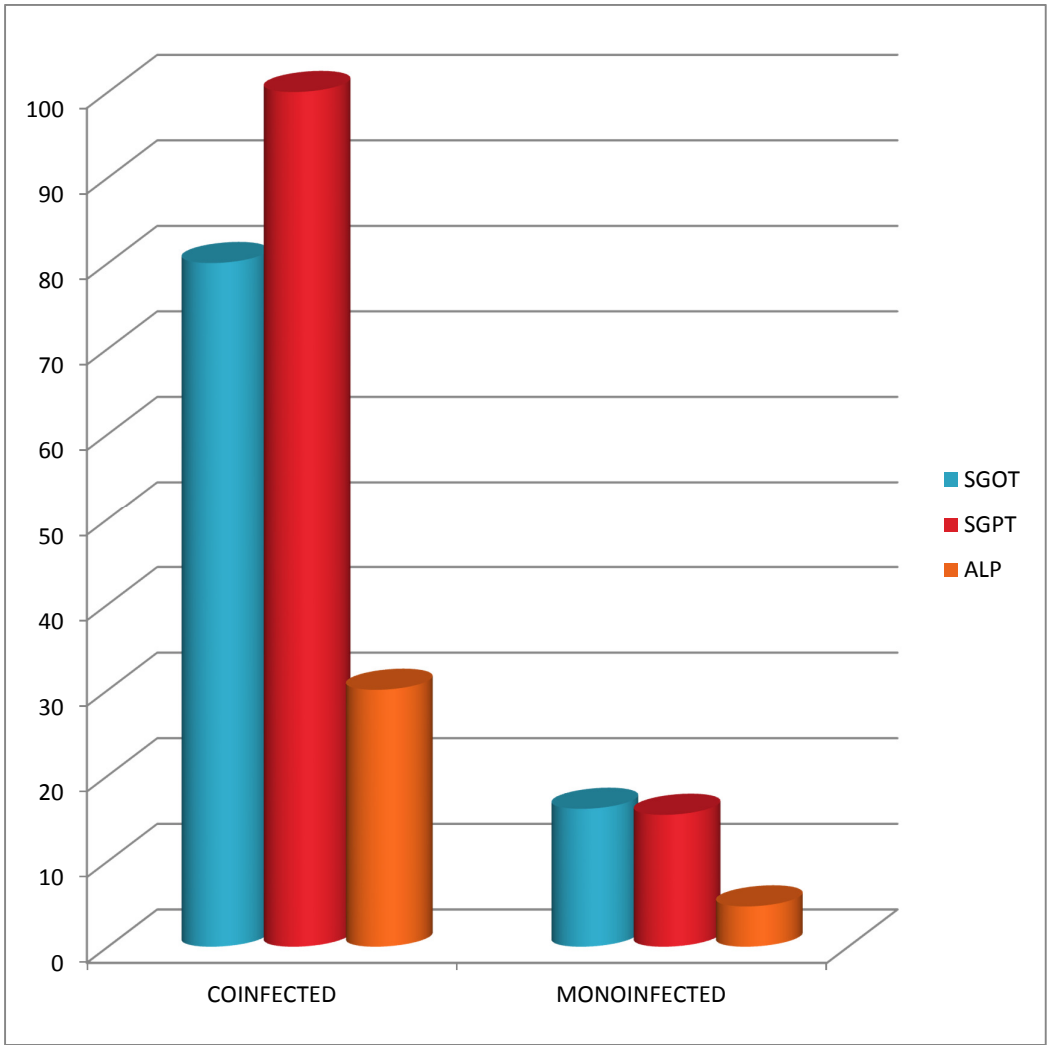


TABLE: 14 COMPARISON OF LIVER ENZYMES BETWEEN THE MONOINFECTED AND CO-INFECTED PATIENTS WITH RESPECT TO NO HISTORY OF ALCOHOL INTAKE.

S. No	Name of the liver function test	Patient with only HIV infection (n=100)		Patient with HIV and co-infection (n=6)		P value	Statistical test used
		Mean	SD	Mean	SD		
1	SGOT (IU/mm^3)	28.05	17.9	116.7	139	0.0002*	Mann Whitney U test
2	SGPT (IU/mm^3)	30	19.9	97.17	78.6	<0.0001**	Mann Whitney U test
3	ALP (IU/mm^3)	67.5	24.5	99.3	18.1	0.002*	Mann Whitney U test

** indicates p<0.0001 (extremely significant) and * indicates p<0.01and considered highly significant

There is a statistically significant difference in the mean level of SGOT, SGPT and ALP between the monoinfected and co-infected non alcoholic patients.

FIG: 14 COMPARISON OF LIVER FUNCTION TESTS BETWEEN THE MONOINFECTED AND CO-INFECTED PATIENTS WITH RESPECT TO NO HISTORY OF ALCOHOL INTAKE

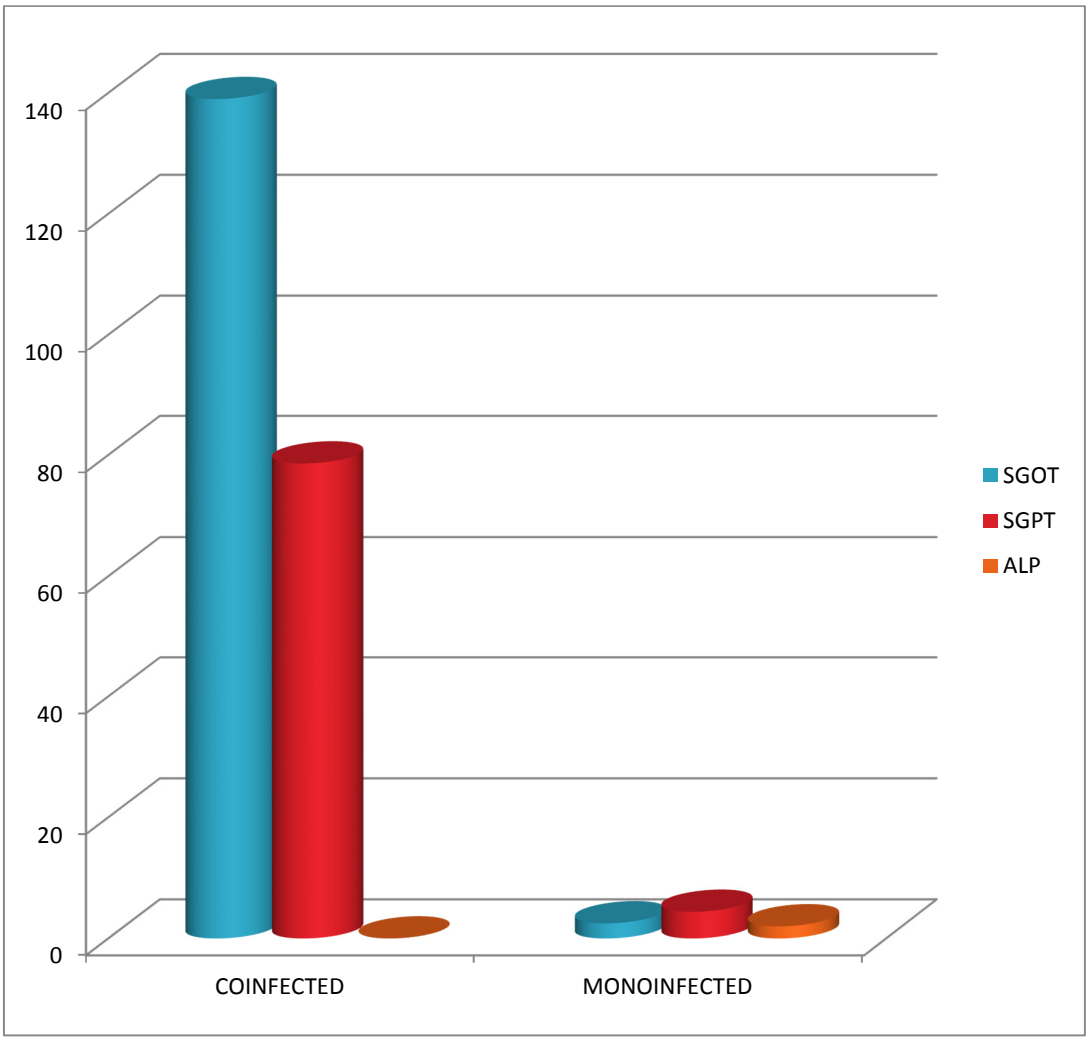
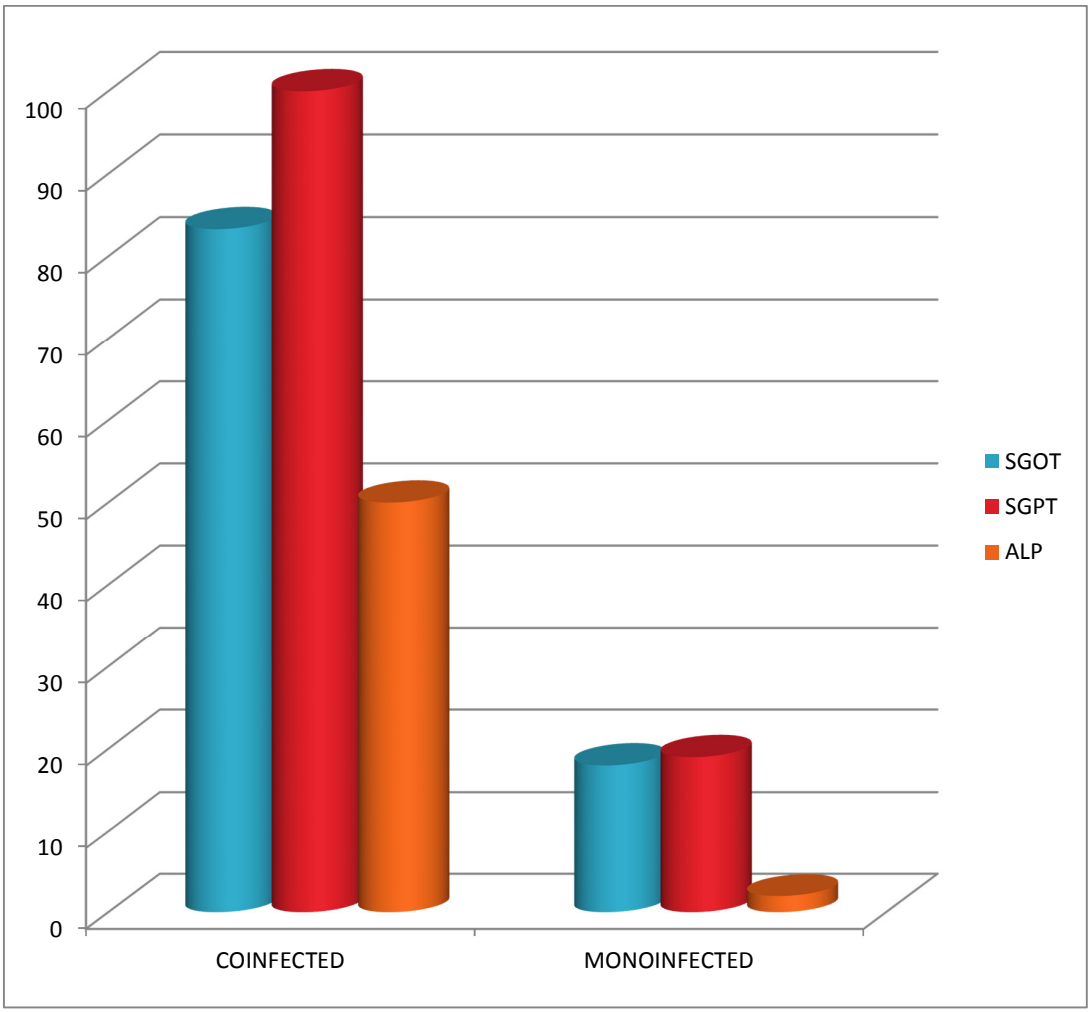


TABLE: 15 PROPORTION OF PATIENTS WITH ELEVATED LIVER ENZYMES BETWEEN MONO INFECTED AND CO-INFECTED NON ALCOHOLIC PATIENTS

S. No	Name of the liver function test	Infected with HIV alone (n=100)		HIV with co-infection (n=6)	
		(n)	(%)	(n)	(%)
1	SGOT (IU/mm^3)	18	18	5	83.3
2	SGPT (IU/mm^3)	19	19	6	100
3	ALP (IU/mm^3)	15	15	3	50

Among the non alcoholics SGOT was elevated among 83.3% of the patients co-infected with hepatitis B or C and SGPT was elevated among all patients with co-infection.

FIG: 15. PROPORTION OF PATIENTS WITH ELEVATED LIVER ENZYMES BETWEEN MONO INFECTED AND CO-INFECTED NON ALCOHOLIC PATIENTS



DISCUSSION

The above study on **PREVALENCE AND CHARACTERISTICS OF HEPATITIS B OR/AND HEPATITIS C CO-INFECTION AMONG PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL** was conducted in the ART department of Thanjavur Medical College and Hospital among 159 HIV positive cases who were on treatment on outpatient basis . It was a cross sectional type of study conducted from march 2017 to august 2017. All HIV positive Patients of age group >12 years of age were included in the study after an informed consent .Patients on ATT or other chronic drug intake except ART and patients with risk factors for hepatic injury like diabetes mellitus, systemic hypertention or hyperlipidemia and patients who were not willing to give consent were not included in the study, detailed history, clinical examination were done along with sample collection for liver enzymes, CD4 counts ,HBsAg, and anti HCV. All the data obtained were compiled and analyzed for the results. The further analysis of the results is as follows:

SOCIO DEMOGRAPHIC CHARACTERISTICS

1. AGE:

The total no of HIV positive cases in the study population was 159 with a mean age group of 42.4 years of age and standard deviation of 10.6. The minimum age in the study population was 16 years and the maximum age was 70 years. Around 66.7% of the study population was between the age group of 30-50 years of age.

2. GENDER:

Among the 159 HIV positive cases 85**(53.5%)** of the patients were male and 74**(46.5%)** of the patients were female with a male:female ratio of **1.15:1** . The maximum number of study population was concentrated between the 30-50 years of age which was 55(64.7%) among which the male:female ratio was 0.9.

3. RISK OF TRANSMISSION

The different modes of transmission and their distribution in the study was as follows Heterosexual- 96.2%

Parent to child-1.9%

Homosexual-1.3%

IUI-0.6%

Most of the patients had heterosexual route as the common mode of trans mission which was **96.2%**

Modes of transmission by needle stick injury, intra venous drug abuse and blood transfusion were not reported in the study may be because of the smaller population of the study group. Only one patient among the study population had intra uterine insemination to be the probable risk for transmission of HIV.

4.ART REGIMEN:

The distribution according to the type of ART regimen in the increasing order of frequency is as follows

TLE- 72(45.3%)

ZLN-69(43.4%)

TLN- 9(5.7%)

ZLE- 6(3.8%)

OTHERS- 2(1.2%)

Almost all the regimen in the study had risk of hepatotoxicity which could also be a cause for elevated liver enzymes in the study

Studies show that hepatotoxicity is the 3 rd most common reason for discontinuation of ART.

5. WHO CLINICAL STAGING:

Distribution of the study population as per the WHO clinical staging showed that **ALMOST 57.9 %** OF the patients were in the stage 1 , and only 1.9% of the patients were in stage 4.

6. BMI

The distribution of HIV positive cases among the different groups of BMI showed that almost **62.9 %** of the patients had a BMI ranging between **19.51-25** kg/m^2

It was also observed that none of the patients were under the obese group.

SEROPREVALENCE OF HIV/HBV and HIV/HCV CO-INFECTION

From the table:7 it is observed that in my study

- The overall prevalence of the HIV patients co-infected with either HBV or HCV was **6.28%**
- The prevalence of HIV/HBV co-infection and HIV/HCV co-infection were **5.03% and 1.25 %** respectively.

Sero prevalence of HIV/HBV OR HIV/HCV varies in different geographical area .It depends on the endemicity of the disease, population exposed to risk and the common mode of transmission in that particular geographical area .Considering hepatitis b it has its highest prevalence in the endemic areas like south American and the African countries where the co-infection rates are very high.

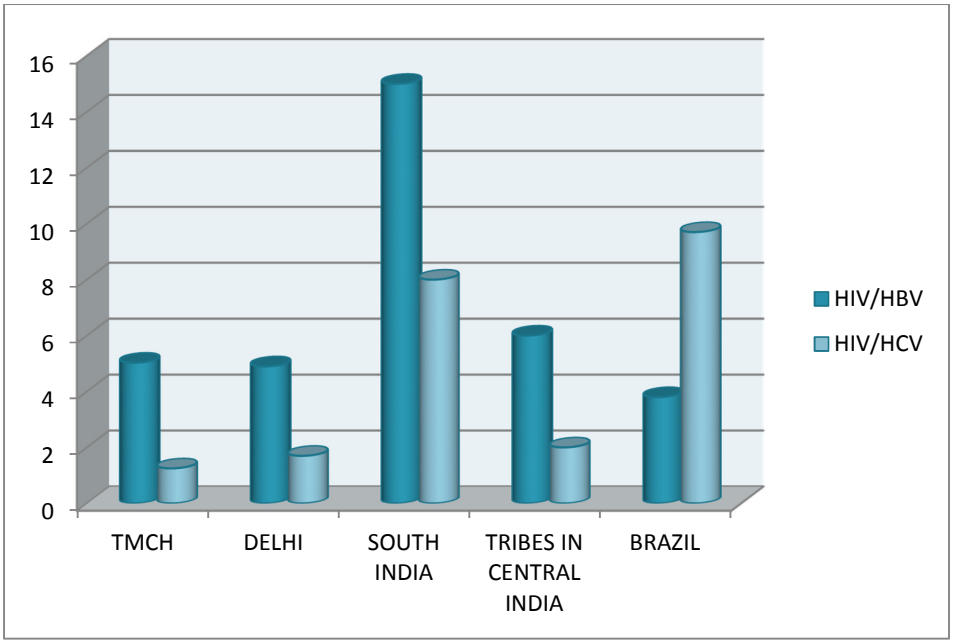
1.Lukman femi owolabi et al[37] has described in his study on the prevalence and burden of HIV and hepatitis B co-infection with data from seventeen states in **Nigeria** that the overall prevalence of HIV/HBV co-infection was **17%** .

2. Ano Novo chandra et al[38] conducted a similar study in around 120 HIV patients in **South India** and estimated the prevalence of HBV and HCV to be 15% and 8% respectively.

3. Natália Alberto et al[39] in his study on the prevalence of HIV/HCV co-infection in **Midwestern Brazil** f found that the prevalence of hepatitis B and C infection among the HIV patients was around 3.8% and 9.7%.

4.Sanjiv Ahuja et al[40] studied seroprevalence of hepatitis B and C co-infection in hiv positive patients at the Dr. r. m. l. Hospital & PGIMER, **New Delhi, India** and found the prevalence to be (4.9%) for HBV and (1.7%) for HCV respectively. This gives an example of the Indian scenario.This is almost similar to the prevalence of the HBV , HCV infection in TMCH as per my study.

FIG: 16 VARIABLE PREVALENCE OF HIV/HBV AND HIV/HCV CO-INFECTION IN DIFFERENT GEOGRAPHICAL AREAS



CD4 COUNT

From the study it is observed that among the patients monoinfected with HIV alone around 54.7% of the patients have CD4 count >500. However among the patients co-infected with either HBV or HCV around 80% of the patients have CD4 counts between 201-500 and 20% of them lie below 20%.

On comparing the mean CD4 counts between the monoinfected and the co-infected individuals it was found that the mean CD4 count was lower in both males(298). and females(265) of the coinfected groups. However the

difference between their mean value was statistically significant only among the females

Review of literature confirms the lower level of CD4 counts among the co-infected individuals which was explained due to the increase in the cytopathic effects of the HIV virus on co-infection with the HBV or HCV.

1.Nebiyou Yemanebrhane[41] et al underwent a study on Magnitude of Hepatitis B Virus and Hepatitis C Virus among HAART Taking Patients and Association with Liver and Renal Function and **CD4+ T Cells Level** among 384 patients in **Ethiopia** . He found that **the mean cd4 count was lower** among the co-infected patients when compared with the mono infected patients

2.This is comparable to the study by **Mayaphi and colleagues[32]** which observed that there was an increased HBV prevalence in HIV patients with CD4 count of ≤100cells/μL and it indeed had a major risk factor of increased HBV replication.

3.In another study done by **Yitayih Wondimeneh et al**[43] among 400 HIV patients **the mean CD4 counts were lower** among the co-infected HIV patients compared to the monoinfected patients and this is again supporting my study

The lower level of CD4 count is an indication that HBV/HCV infection can aggravate the propensity of the pathogenesis of AIDS in HIV infected persons as CD4 count is directly proportional to the level of immunosuppression.

LIVER ENZYME LEVELS

Comparison was done on the level of liver enzymes between the monoinfected and the co-infected patients. The mean levels of SGOT, SGPT, and ALP were assessed in the two groups .

The mean level of SGOT,SGPT and ALP among the co-oinfected individuals were found to be 88.4IU, 83.1IU and 84.4IU respectively which was significantly higher than the levels measured among the monoinfected patients which were 28.3IU, 29.5IU,and 69.1IU.

The difference between these mean levels of liver enzymes were statistically significant with a p value of ($p<0.0001$, $p<0.0001$ and $p<0.04$ for SGOP, SGPT and ALP).

In our study SGOT was elevated in 50% of the coinfected patients and SGPT was elevated in 80% of the coinfected patients.

1.Yitayih wondimeneh et al[43] studied the liver enzyme levels among 400 hiv patients at **North West Ethiopia.** Despite absence of statistically significant difference in the mean levels of the liver enzymes between HIV-monoinfected and HIV-viral hepatitis co-infected individuals, raised AST,ALT and ALP were found in both the monoinfected and the co-infected individuals.

2. However, in a study which was conducted in **South Africa** by **Lodeneo H et al**[44], 70% of HIV-HBV and HIV-HCV co-infected study participants had significantly elevated SGOT, SGPT and 56% of them had elevated ALP

3. Similarly, in a study done by **Tripathi et al** in **Northern India**[45], significantly raised ALT was found in 14% of HIV/HBV co-infections and 20% in HIV/HCV co-infected patients .

The difference in the liver enzyme levels between various studies may be due to difference in study design, duration of the viral hepatitis infection as well as the patient's condition like having chronic alcoholism or other drug induced hepatotoxicity. In addition, HIV can also infect the hepatic or kupffer cells which can further lead to the development of liver fibrosis and elevated liver enzyme levels.

However, the magnitude of the complication of the liver injury may be higher if the HIV positive patients are co-infected with HBV or HCV as indicated above.

LIVER ENZYME LEVELS IN HIV MONOINFECTED AND HBV/HCV COINFECTED NON ALCOHOLIC PATIENTS

In order to analyse the level of liver enzymes among the monoinfected and the co-infected patients eliminating the risk of hepatic injury caused by alcoholic consumption, comparison was done for the serum liver enzyme levels among the non-alcoholics.

It was observed that even among the non alcoholics there was a statistically significant difference in the mean level of SGOT, SGPT and ALP between the monoinfected and co-infected patients.

BMI AMONG THE CO-INFECTED AND MONOINFECTED PATIENTS

Comparison of the mean BMI between the two groups showed a lower level of BMI among the co-infected patients (18.5 kg/m^2) when compared to that of the mono infected patients(21.03 kg/m^2). The difference in mean between the two group was statistically significant with a p value of <0.01

This explains the debilitated state of the HIV positive patients when they are co-infected with HIB/HCV.

SUMMARY

The above study on **PREVALENCE AND CHARACTERISTICS OF HEPATITIS B OR/AND HEPATITIS C CO-INFECTION IN PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL** was conducted in the ART department of Thanjavur Medical College and Hospital among 159 HIV positive cases who were on treatment on outpatient basis . It was a cross sectional type of study conducted from March 2017 to August 2017. All HIV positive Patients of age group >12 years of age were included in the study after an informed consent .Patients on ATT or other chronic drug intake except ART and patients with risk factors for hepatic injury like diabetes mellitus, systemic hypertention or hyperlipidemia and patients who were not willing to give consent were not included in the study, detailed history, clinical examination were done along with sample collection for liver enzymes, CD4 counts, HBsAg, and anti HCV. All the data obtained were compiled and analysed for the results. The observation following further analysis of the results showed that

- The **prevalence of HIV/HBV and HIV/HCV** CO-INFECTION was **5.03%** AND **1.25%** respectively
- Among them 80% of the co-infected patients were unaware of the added infection they had and none of the monoinfected patients were vaccinated against HBV infection.
- The **mean age group** of the study population was **42.4 years** with SD of 10.6. most of the HIV positive patients were grouped between the age group of **30-50 years** of age.
- The commonest mode of transmission was **heterosexual** mode.
- Around 57.9% OF HIV patients were under WHO clinical stage 1. However among the co-1nfected patients most of them were under WHO clinical Stage 2 or 3.
- **The mean BMI** was lower among the HIV/HBV OR HIV/HBC co-infected individuals (**18.1)** when compared with that of the patients monoinfected with HIVonly(21.03) and their difference was statistically significant.
- The **mean CD4T cell count was lower** both among the **male (298)** and the **female (265)** patients co-infected with HIV/HBV or HIV/HCV when compared with that of the male(527.5) and female (659.1) patients monoinfected with HIV only. The difference among their mean was statistically significant among females but not in males.

- The **mean level of the liver enzymes SGOT (88.4), SGPT (83.1) and ALP (84.4) were higher** among the HIV/HBV or HIV/HCV co-infected individuals when compared with that of the mean SGOT, SGPT and ALP in the patients mono infected with HIV alone. Further the difference between the means of all these liver enzymes were statistically significant individually.

CONCLUSION

- It is evident from the study that HIV infected individuals have a higher probability of getting co-infected with HBV or HCV
- The lower level of CD4 count among the HIV/HBV or HIV/HCV co-infected patients is an indication that HBV/HCV infection aggravates the propensity of the pathogenesis of AIDS in HIV infected persons as CD4 count is directly proportional to the level of immunosuppression.
- There is a higher risk of progression of liver disease among the patients with HIV/HBV or HIV/HBC co-infection. The risk is further aggravated by exposure to opportunistic infections, alcoholism and anti retro viral drugs.
- Hence co-infection with HBV OR HCV among the HIV seropositive cases should be estimated earlier before treatment with antiretroviral drugs, for apt choice of drugs and to avoid progression of liver disease and to prevent complications from opportunistic infections , toxicity of antiretroviral drugs and other toxins(alcohol).
- It is also mandatory for a strict monitoring of the liver enzymes and the CD4T cell counts regularly among these individuals in order to minimise the complications of the liver injury and for effective HIV treatment.

LIMITATIONS

- The measurement, HIV viral load, HBV DNA and HCV RNA viremia assays were required but these tests were not done due financial limitations.

- There may be false negative HCV antibody test in the individuals co-infected with HIV due to the impaired antibody formation in the immunosuppressed state

- The low frequency of HCV infection in the study could be due to the lower incidence of transfusion history, IV drug abuse which may be reported to be higher in other parts of the world or other parts of India. Therefore mode of transmission affects the prevalence of co-infection.

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CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR .C. MAGHIL BELINTA ,** Post graduate in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :

Date : Signature of participant

INFORMATION SHEET

We are conducting a cross sectional Study on THE **PREVALANCE AND CHARACTERISTICS OF HEPATITIS B AND/OR HEPATITIS C VIRUS COINFECTION IN PATIENTS WITH HIV IN THANJAVUR MEDICAL COLLEGE AND HOSPITAL ““ (MARCH 2017-AUGUST 2017)** in the Department of General Medicine , Thanjavur Medical College & Hospital, Thanjavur – 613004.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of participant**

Date:

PROFORMA

NAME:

AGE:

SEX:

ART NUMBER :

OCCUPATION:

ADDRESS:

MODE OF TRANSMISSION:

1.Heterosexual

2.Homosexual

3.Intavenous drug abuse/needle stick injury

4.Blood transfusion/organ transplantation

5.Parent to child transmission

6.others

ART REGIMEN:

DURATION OF TREATMENT:

HISTORY:

HISTORY	YES	NO
CHRONIC DRUG INTAKE		
BLOOD TRANFUSION		
INTRA VENOUS DRUG ABUSE		
HEP B VACCINATION		
K/C/O HEP B /HEP C		

CLINICAL EXAMINATION :

GENERAL EXAMINATION:

WHO CLINICAL STAGING

HEIGHT:

WEIGHT:

BMI:

LAB INVESTIGATION

CD4COUNT:

HBsAg:

Anti HCV:

SGOT

SGPT

ALP

OTHERS:

ABREVIATIONS

AIDS-Acquired Immuno Deficiency Syndrome

ALP- Alkaline Phosphatase

ALT-Alanine Transaminase

ART-Anti Retro Viral Therapy

AST-Aspartate Transaminase

ATT-Anti Tuber Culous Treatment

BMI-Body Mass Index

CD-Clusterd Differentiation

DNA-Deoxy Ribo Nucleic Acid

HBV-Hepatitis B Virus

HCV-Hepatitis C Virus

HIV-Human Immuno Deficiency Virus

LFT-Liver Function Test

RNA- Ribo Nucleic Acid

SD-Standard Deviation

SGOT-Serum Glutamic-Oxaloacetic Transaminase

SGPT- Serum Glutamic Pyruvic Transaminase

TLE-Tenofovir, Lamivudine, Efavirenz

TLN-Tenofovir, Lamivudine, Nevirapine

WHO-World Health Organisation

ZLN-Zidovudine Lamivudine, Nevirapine

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
1	Arivanantham	52	M	1	NO	NO	NO	NO	NO	TLE	9	845	1
2	SAROJA	22	F	1	NO	NO	NO	NO	NO	TLE	2	450	1
3	KALAISELVI	45	F	1	NO	NO	NO	NO	NO	TLE	5	101	3
4	SAROJA	50	F	1	NO	NO	NK	NO	NO	TLN	6	510	2
5	ANNADURAI	40	M	1	NO	NO	NO	NO	NO	TLE	2	278	2
6	TAMILMANI	52	F	1	NO	NO	NO	NO	NO	TLE	4	941	1
7	KUMARAVEL	48	M	1	NO	NO	NO	NO	NO	TLE	3	391	2
8	DHANAPAL	45	M	1	YES	NO	NO	NO	NO	ZLN	2	655	1
9	SUGANYA	23	F	1	NO	NO	NO	NO	NO	TLE	1	674	1
10	S.MANIVANNAN	39	M	1	YES	NO	NO	N0	N0	ZLN	4	435	1
11	KAMALA	36	F	1	NO	NO	NO	NO	NO	TLE	4	281	2
12	MOORTHY	34	M	1	YES	NO	NO	NO	NO	TLE	4	79	3
13	ARUMUGAM	40	M	1	YES	NO	NO	NO	NO	ZLN	1	1250	2
14	JANAKI	60	F	1	NO	NO	YES	NO	NO	TLN	11	827	1
15	BALU	55	M	1	NO	NO	YES	NO	NO	ZLN	7	50	2
16	BALAMMAL	50	F	1	NO	NO	YES	NO	NO	TLE	3	644	1
17	SELVI	48	F	1	NO	NO	NO	NO	NO	TLE	3	512	1
18	GOVINDRAJ	65	M	1	NO	NO	YES	NO	NO	ZLN	7	204	3
19	AMUDHA	45	F	1	NO	NO	NO	NO	NO	ZLN	7	1155	1
20	NIRMALA	47	F	1	NO	NO	NO	NO	NO	TLE	1	730	1
21	NARAYANAN	25	M	1	YES	NO	NO	NO	NO	ZLN	1 M	250	
22	RAGUPATHY	48	M	1	YES	NO	NO	NO	NO	TLE	2	516	1
23	DEVA JOTHI	45	F	1	NO	NO	NO	NO	NO	ZLE	7	602	1
24	SENTHIL KUMAR	42	M	1	YES	NO	NO	NO	NO	_	_	320	1
25	JEYARAMAN	42	M	1	YES	NO	NO	NO	NO	ZLN	1M	602	1
26	THILAGAVATHY	40	F	1	NO	NO	NO	NO	NO	TLE	2	309	1
27	KARUNANIDHI	46	M	1	NO	NO	NO	NO	NO	TLE	6	400	1

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
28	ANBALAGAN	52	M	1	NO	NO	NO	NO	NO	ZLN	10	420	2
29	MATHI	58	M	1	NO	NO	NO	NO	NO	ZLN	8	491	1
30	LATHA	35	F	1	NO	NO	NO	NO	NO	ZLN	6	673	1
31	SEKAR	68	M	1	YES	NO	NO	NO	NO	TLE	5	247	2
32	M.SELVI	50	F	1	NO	NO	NO	NO	NO	TLE	6	287	1
33	JEGAN	38	M	1	YES	NO	NO	NO	NO	TLE	1M	321	3
34	ELAYARANI	33	F	1	NO	NO	NO	NO	NO	TLE	2	1422	1
35	PRABAKARAN	43	M	1	YES	NO	NO	NO	NO	TLE	2 DAYS	193	2
36	AMUDHA	40	F	1	NO	NO	NO	NO	NO	TLN	5	805	1
37	SAROJA	58	F	1	NO	NO	NO	NO	NO	TLN	8	701	1
38	THIRUSELVAM	60	M	1	YES	NO	NO	NO	NO	ZLN	4	463	1
39	KALAI ARASI	50	F	1	NO	NO	NO	NO	NO	TLE	5	816	1
40	KASTHURI	41	F	1	NO	NO	NO	NO	NO	TLE	9	975	1
41	ANITHA	36	F	1	NO	NO	NO	NO	NO	ZLN	8	225	1
42	VALARMATHI	41	F	1	NO	NO	NO	NO	NO	ZLN	9	514	1
43	MUTHULAKSHMI	51	F	1	NO	NO	NO	NO	NO	ZLN	5	454	1
44	MEENA	57	F	1	NO	NO	NO	NO	NO	TLE	7	901	1
45	RAVICHANDRAN	53	M	1	YES	NO	NO	NO	NO	ZLN	8	454	1
46	M.BASKER	41	M	1	YES	NO	NO	NO	NO	ZLN	7	354	3
47	ANANTHI	19	F	5	NO	NO	NO	NO	NO	ZLN	4	506	1
48	NAGALAKSHMI	42	F	1	NO	NO	NO	NO	NO	ZLN	7	630	2
49	SASIKALA	44	F	1	NO	NO	NO	NO	NO	TLE	1M	546	1
50	SELVI	52	F	1	NO	NO	NO	NO	NO	ZLN	11	803	1
51	KUMAR	35	M	1	NO	NO	NO	NO	NO	TLN	3	201	2
52	MARIYAMMAL	38	F	1	NO	NO	NO	NO	NO	TLE	2	932	1
53	PANCHALAN	55	M	1	YES	NO	YES	NO	NO	ZLN	11	485	1
54	BUVANESWARI	38	F	1	NO	NO	TES	NO	NO	TLE	11	212	2

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
55	KALAISELVI	45	F	1	NO	NO	NO	NO	NO	TLE	4	211	2
56	PALANIVEL	37	M	1	YES	NO	NO	NO	NO	TLE	7	467	1
57	RAVICHANDRAN	54	M	1	NO	NO	NO	NO	NO	TLE	1	741	1
58	SHANMUGAM	46	M	1	NO	NO	NO	NO	NO	TLE	1	818	1
59	VANITHA	32	F	1	NO	NO	NO	NO	NO	TL/L/R	2	920	1
60	JAYALAKSHMI	54	F	1	NO	NO	NO	NO	NO	ZLN	8	602	3
61	SELVAM.K	46	M	1	YES	NO	NO	NO	NO	TLE	2	94	3
62	V.SELVARANI	32	F	1	NO	NO	NO	NO	NO	ZLN	7	281	2
63	VISHWANATHAN	40	F	1	YES	NO	NO	NO	NO	TLE	2	1302	1
64	RAJENDRAN	47	M	1	YES	NO	NO	NO	NO	ZLN	7	968	1
65	JEGADISH	40	M	1	YES	NO	NO	NO	NO	ZLN	4	564	1
66	KALYANASUNDAR/	43	M	1	YES	NO	NO	NO	NO	TLE	4	165	3
67	MAHESWARI	34	F	1	NO	NO	NO	NO	NO	TLE	3	771	1
68	SASIPRIYA	32	F	1	NO	NO	NO	NO	NO	TLE	1	325	2
69	ANANDHAN	48	M	1	NO	NO	NO	NO	NO	ZLN	6	684	2
70	ANNADHURAI	47	M	1	NO	NO	NO	NO	NO	TLE	10	1009	1
71	BAVANI	49	F	1	NO	NO	NO	NO	NO	TLE	2M	469	1
72	HELEN LATHA	40	F	1	NO	NO	NO	NO	NO	ZLE	11	700	1
73	NANDHAKUMAR	45	M	1	YES	NO	NO	NO	NO	TLE	5	355	1
74	MANIMEGALAI	43	F	1	NO	NO	NOI	NO	NO	TLE	2	541	1
75	JEYANTHI	37	F	1	NO	NO	NO	NO	NO	ZLN	5	606	1
76	JOTHIVEL	55	M	1	YES	NO	NO	NO	NO	TLE	2	501	1
77	SHAKTHIVEL	47	M	1	NO	NO	NO	NO	NO	TLE	1	214	3
78	JEYASEELAN	38	M	1	NO	NO	NO	NO	NO	ZLN	7	276	2
79	INDRA	50	F	1	NO	NO	NO	NO	NO	TLE	11	737	1
80	VETRISELVAN	37	M	2	YES	NO	NO	NO	NO	ZLN	10	266	3
81	SUBRAMANIYAN	55	M	1	YES	NO	NO	NO	NO	TLE	1	612	1

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
82	SWAMINATHAN	37	M	1	YES	NO	NO	NO	NO	TLE	7	837	1
83	SAMIYAMMAL	60	F	1	NO	NO	NO	NO	NO	TLE	4	523	1
84	CHELIAN	38	M	1	NO	NO	NO	NO	NO	ZLE	3	226	1
85	JENCY	33	F	1	NO	NO	NO	NO	NO	TLE	1	842	1
86	SAMINATHAN	48	M	1	YES	NO	NO	NO	NO	TLE	11	854	1
87	PERIYASAMI	42	M	1	YES	NO	NO	NO	NO	TLE	10	432	1
88	DURGADEVI	28	F	1	NO	NO	NO	NO	NO	TLE	3	791	1
89	BALAKRISHNAN	32	M	1	NO	NO	NO	NO	NO	TLE	7	131	2
90	SELVARANI	39	F	1	NO	NO	YES	NO	NO	TLE	9	999	1
91	SASIMAL	30	F	1	YES	NO	NO	NO	NO	ZLN	9	835	1
92	A.ARMUGAM	35	M	1	YES	NO	YES	NO	NO	TLE	11	500	1
93	BHAVANI	38	M	1	YES	NO	NO	NO	NO	TLE	1	520	1
94	KAMALAM	63	F	1	YES	NO	NO	NO	NO	TLN	11	1037	2
95	RAJALAKSHMI	40	F	1	NO	NO	NO	NO	NO	ZLE	11	1074	1
96	MADHAVI	30	F	1	NO	NO	NO	NO	NO	ZLE	7	559	2
97	MUTHULAKSHMI	35	F	1	NO	NO	NO	NO	NO	TLE	1	508	1
98	SIVAKUMAR	35	F	1	YES	NO	NO	NO	NO	ZLN	1	605	1
99	S.UMA	41	F	1	NO	NO	NO	NO	NO	ZLN	8	850	1
100	RAJENDRAN	36	M	1	NO	NO	NO	NO	NO	TLE	2	297	1
101	S.PREETHI	18	F	5	NO	NO	NO	NO	NO	TLE	9	587	1
102	M.KAVITHA	40	F	4	NO	NO	NO	NO	NO	ZLN	3	599	3
103	S.SIVA	21	M	5	NO	NO	NO	NO	NO	ZLN	8	455	1
104	SIVANANDHA	35	M	1	YES	NO	NO	NO	NO	ZLN	1	236	3
105	AROKIANATHAN	49	M	1	YES	NO	NO	NO	NO	ZLN	4	402	2
106	GUNASEKARAN51	51	M	1	YES	NO	NO	NO	NO	ZLN	2	1073	1
107	VASUKI	30	F	1	NO	NO	NO	NO	NO	TLE	2	200	2
108	CHINNATHA	40	F	1	NO	NO	NO	NO	NO	TLE	2	189	2

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
109	JEYAKODI	51	F	1	NO	NO	NO	NO	NO	TLE	1	98	2
110	M.VINCENT	61	M	1	NO	NO	NO	NO	NO	TL/A/R	2	243	1
111	SELVI.K	35	F	1	NO	NO	NO	NO	NO	TLE	4	390	2
112	SURESH	34	M	1	YES	NO	NO	NO	NO	ZLN	3	680	1
113	SATHYAMOORTHY	49	M	1	NO	NO	NO	NO	NO	TLE	2	360	1
114	J.SAIMU	23	F	1	NO	NO	NO	NO	NO	ZLN	9	607	2
115	KARTHIKEYAN	16	M	1	NO	NO	NO	NO	NO	ZLN	8	1182	1
116	POONGODHAI	32	F	1	NO	NO	NO	NO	NO	ZLN	10	1261	1
117	MALA	45	F	1	NO	NO	NO	NO	NO	TLE	4	654	2
118	RANGARAJ	63	M	1	NO	NO	NO	NO	NO	ZLN	5	381	1
119	EZHILMATHI	40	F	1	NO	NO	NO	NO	NO	TLE	5	396	2
120	SUMATHI	31	F	1	NO	NO	NO	NO	NO	TLN	2	498	2
121	JEEVA	40	F	1	NO	NO	NO	NO	NO	ZLN	3	388	1
122	CHITRA	29	F	1	NO	NO	NO	NO	NO	ZLN	5	189	3
123	PREMALATHA	39	F	1	NO	NO	NO	NO	NO	ZLN	3	1466	1
124	ISAK	58	M	1	YES	NO	NO	NO	NO	ZLN	11	423	2
125	ELAKIYA.E	30	F	1	NO	NO	NO	NO	NO	TLE	4	94	2
126	KANNAPPAN	40	M	1	YES	NO	NO	NO	NO	ZLN	5	196	3
127	RAMESH	47	M	1	NO	NO	NO	NO	NO	ZLN	5	424	2
128	NADESAN	63	M	1	YES	NO	NO	NO	NO	TLE	6	245	2
129	ROSSAMMAL	70	F	1	NO	NO	YES	NO	NO	TLE	7	478	1
130	AMMAKANNU	40	F	1	NO	NO	NO	NO	NO	ZLE	1	1311	1
131	VASANTHAKUMAR	42	F	1	NO	NO	NO	NO	NO	TLN	9	709	1
132	RADHAKRISHNAN	59	M	1	YES	NO	NO	NO	NO	TLE	2	145	1
133	JOHN	47	M	1	NO	NO	NO	NO	NO	ZLN	7	156	2
134	VIJAYAN	31	M	1	NO	NO	NO	NO	NO	ZLN	4	154	2
135	ARULKUMAR	26	M	1	YES	NO	NO	NO	NO	TLE	5	658	2

S.NO	NAME	AGE	SEX	MOT	ALCOHOL	O.C.D.I	BT/OT	IV. DA	HEP B Vac	ART REG	DOT(yrs)	CD4	WHO.CS
136	PONNILAVAR	57	M	1	YES	NO	NO	NO	NO	ZLN	6	458	2
137	THAMBIKANNU	45	M	1	NO	NO	NO	NO	NO	TLE	1	589	1
138	DHINAKARAN	44	M	1	YES	NO	NO	NO	NO	ZLN	1	345	2
139	SENTHILKUMAR	48	M	1	YES	NO	NO	NO	NO	ZLN	2	547	4
140	AASAITHAMBI	29	M	1	YES	NO	NO	NO	NO	TLE	6	285	4
141	KARTHIKEYAN	55	M	1	YES	NO	NO	NO	NO	ZLN	7	980	1
142	VALLUVAN	32	M	1	YES	NO	NO	NO	NO	ZLN	3	285	2
143	PAULRAJ	36	M	1	NO	NO	NO	NO	NO	ZLN	1	1021	3
144	ARIVUMANI	55	M	1	NO	NO	NO	NO	NO	ZLN	7	1256	1
145	GANESH	41	M	1	NO	NO	NO	NO	NO	ZLN	5	652	2
146	GOPINATH	23	M	1	YES	NO	NO	NO	NO	ZLN	1	987	2
147	PETER	46	M	1	NO	NO	NO	NO	NO	ZLN	4	890	1
148	SIVAKUMAR	48	M	1	YES	NO	NO	NO	NO	ZLN	7	64	2
149	PONNUSAMY	44	M	1	NO	NO	NO	NO	NO	ZLN	2	841	3
150	KUMARESAN	64	M	1	YES	NO	NO	NO	NO	ZLN	7	980	3
151	SEETHA	29	F	1	NO	NO	NO	NO	NO	TLN	4	154	4
152	UMARANI	35	F	1	NO	NO	YES	NO	NO	ZLN	7	560	3
153	BANGARU	44	M	1	YES	NO	NO	NO	NO	ZLN	6	621	3
154	ANBUMANI	40	M	1	NO	NO	NO	NO	NO	TLE	1	521	3
155	ARAVIND	45	M	1	YES	NO	NO	NO	NO	ZLN	5	681	2
156	AYAPPAN	46	M	1	NO	NO	NO	NO	NO	ZLN	1	566	1
157	SANTHANAN	28	M	2	NO	NO	NO	NO	NO	ZLN	3	325	1
158	MOHAN	22	M	1	NO	NO	NO	NO	NO	TLE	5	1452	1

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
1	24.91	27	30	46	neg	neg
2	16.64	17	20	87	pos	neg
3	15.55	17	21	91	neg	neg
4	24.4	14	15	48	neg	neg
5	26.03	18	21	102	neg	neg
6	22.2	18	23	46	neg	neg
7	17.6	24	26	36	neg	neg
8	23.7	44	42	50	neg	neg
9	27.3	47	50	38	neg	neg
10	23.3	34	32	45	neg	neg
11	23.7	29	31	54	neg	neg
12	21.22	22	19	54	neg	neg
13	25.8	21	20	48	neg	neg
14	23.78	15	16	107	neg	neg
15	24.1	33	42	49	neg	neg
16	19.5	19	18	72	neg	neg
17	26.15	46	42	36	neg	neg
18	23.12	20	19	38	neg	neg
19	20.88	46	49	36	neg	neg
20	27.03	30	64	80	neg	neg
21	23.24	27	20	53	neg	neg
22	23.2	31	35	63	neg	neg
23	19.02	18	16	70	neg	neg
24	13.33	48	58	82	pos	neg
25	33.2	23	25	76	neg	neg
26	24.14	56	45	39	neg	neg
27	15.17	23	25	75	neg	neg

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
28	17.3	29	24	47	neg	neg
29	20.2	36	35	50	neg	neg
30	25.77	17	16	72	neg	neg
31	19.03	34	37	89	neg	neg
32	16	41	48	70	pos	neg
33	21.97	29	25	52	neg	neg
34	25.91	24	26	42	neg	neg
35	20.27	27	25	37	neg	neg
36	17.12	43	52	103	neg	neg
37	20.45	18	15	85	neg	neg
38	21.97	25	24	47	neg	neg
39	21.4	39	41	51	neg	neg
40	20.45	28	25	51	neg	neg
41	21.4	397	250	117	pos	neg
42	17.8	26	24	40	neg	neg
43	19.8	19	16	67	neg	neg
44	22.89	22	25	102	neg	neg
45	31.4	30	35	68	neg	neg
46	25.71	26	68	47	neg	neg
47	20.2	63	74	61	neg	neg
48	23.87	16	15	57	neg	neg
49	25.81	22	24	73	neg	neg
50	20.81	21	24	54	neg	neg
51	15.57	22	26	120	neg	neg
52	21.4	22	24	81	neg	neg
53	20.95	34	35	51	neg	neg
54	20.45	28	30	90	neg	neg

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
55	21	102	104	112	pos	neg
56	25.95	31	25	43	neg	neg
57	14.19	24	29	62	neg	neg
58	23.11	23	24	106	neg	neg
59	19.12	26	27	34	neg	neg
60	19.21	55	54	106	neg	neg
61	19.79	64	70	51	pos	neg
62	14.8	22	20	59	neg	neg
63	23.6	26	24	47	neg	neg
64	21.6	30	27	53	neg	neg
65	20.2	29	30	24	neg	neg
66	21	16	20	120	neg	neg
67	21.1	18	19	43	neg	neg
68	20	34	38	37	neg	neg
69	23.66	22	25	38	neg	neg
70	16.5	23	25	68	neg	neg
71	22.06	53	54	1.3	neg	neg
72	30.2	13	20	49	neg	neg
73	16.4	38	40	55	pos	neg
74	24.14	20	26	73	neg	neg
75	18	164	184	79	neg	neg
76	25	22	23	53	neg	neg
77	23.32	21	25	78	neg	neg
78	18.35	23	28	72	neg	neg
79	21	17	21	104	neg	neg
80	18	20	24	60	neg	pos
81	21.1	22	26	41	neg	neg

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
82	16.2	21	23	197	neg	neg
83	20.3	22	23	105	neg	neg
84	25	28	26	43	neg	neg
85	26.2	19	25	62	neg	neg
86	20.3	16	15	78	neg	neg
87	19.4	19	21	173	neg	neg
88	18.3	23	24	120	neg	neg
89	18.05	25	17	52	neg	neg
90	17.2	40	45	56	neg	neg
91	21.09	19	18	63	neg	neg
92	23	35	40	56	neg	neg
93	20.1	56	51	69	neg	neg
94	20.8	35	41	89	neg	neg
95	21.8	22	26	95	neg	neg
96	20.2	28	23	103	neg	neg
97	20	16	15	86	neg	neg
98	28	15	14	62	neg	neg
99	22.5	45	51	96	neg	neg
100	26.17	22	26	58	neg	neg
101	18.3	12	17	35	neg	neg
102	20	15	18	56	neg	neg
103	19.6	42	40	98	neg	pos
104	19.34	35	39	102	neg	neg
105	24	24	22	92	neg	neg
106	17.2	52	41	85	neg	neg
107	23.2	22	21	56	neg	neg
108	16.75	15	19	96	neg	neg

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
109	21	19	25	65	neg	neg
110	23.1	25	22	85	neg	neg
111	24.1	22	18	124	neg	neg
112	21.2	18	15	102	neg	neg
113	20.4	22	45	78	neg	neg
114	19.3	21	31	105	neg	neg
115	20.6	45	47	94	neg	neg
116	23.1	18	17	59	neg	neg
117	20.8	17	16	45	neg	neg
118	20.1	18	20	59	neg	neg
119	20.5	45	54	85	neg	neg
120	23.4	17	19	48	neg	neg
121	20.4	19	15	57	neg	neg
122	19.2	32	31	65	neg	neg
123	25.3	21	32	52	neg	neg
124	20.1	22	29	98	neg	neg
125	15.2	47	45	102	neg	neg
126	16.5	18	12	45	neg	neg
127	18.5	25	26	96	neg	neg
128	20.5	22	26	85	neg	neg
129	19.2	45	39	56	neg	neg
130	25.4	15	19	95	neg	neg
131	22.8	18	17	84	neg	neg
132	25.3	14	16	79	neg	neg
133	17.24	28	26	85	neg	neg
134	19.72	18	14	56	neg	neg
135	17.93	34	36	102	neg	neg

S.NO	BMI	SGOT	SGPT	ALP	HBsAg	ANTI HCV
136	19.82	39	45	91	neg	neg
137	19.2	16	18	56	neg	neg
138	16.05	26	22	48	neg	neg
139	16.87	29	34	76	neg	neg
140	16.25	32	35	89	neg	neg
141	19.5	26	29	34	neg	neg
142	20.61	22	28	59	neg	neg
143	18.54	24	15	62	neg	neg
144	18.3	64	61	51	neg	neg
145	17.3	28	24	90	neg	neg
146	19.6	22	25	81	neg	neg
147	16.78	16	24	57	neg	neg
148	19.5	44	38	50	neg	neg
149	19.5	24	26	36	neg	neg
150	18	19	46	54	neg	neg
151	19.29	75	86	112	pos	neg
152	17.38	55	45	106	neg	neg
153	19.6	23	25	106	neg	neg
154	18.98	26	24	47	neg	neg
155	18.24	21	26	120	neg	neg
156	19.52	22	27	73	neg	neg
157	17.62	31	36	43	neg	neg
158	20.63	34	36	51	neg	neg