"A STUDY OF CARDIAC MANIFESTATIONS IN HIV PATIENTS"

By

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Dissertation submitted to BLDE University, Vijayapura

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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Dr. SANDEEP REDDY KALLAM

LIST OF ABBREVATIONS USED

%	-	Percentage
µ	-	Micro
3TC	-	Lamivudine
AIDS	-	Acquired Immunodeficiency Syndrome
ART	-	Antiretroviral therapy
ARV	-	Antiretroviral
AZT	-	Zidovudine
CAD	-	Coronary Artery Disease
cART	-	Combination antiretroviral therapy
CCR	-	Chemokine receptor
CDC	-	Centers for Disease Control
CI	-	Confidence interval
cm	-	Centimeter
CMV	-	Cytomegalovirus
CNS	-	Central nervous system
CRP	-	C-reactive protein
CVD	-	Cardiovascular disease
DF	-	Degree of freedom
DHSS	-	Department of Health and Human Services
DNA	-	Deoxyribonucleic acid
ECG	-	Electrocardiography
EFV	-	Efavirenz
EIA	-	Enzyme immunoassay
ELISA	-	Enzyme-linked immunosorbent assay

FDA	-	Food and Drug Administration
FTC	-	Emtricitabine
HAART	-	Highly active antiretroviral therapy
HIV	-	Human Immunodeficiency Virus
HSV	-	Herpes simplex virus
KS	-	Kaposi sarcoma
L	-	Liter
LDL	-	Low density lipoprotein
LV	-	Left ventricle
mg	-	Milligrams
mg/dL	-	Milligrams per decilitre
min	-	Minute
ml	-	Millilitre
mmHg	-	Millimetres of mercury
mmol	-	Millimoles
MPAP	-	Mean pulmonary artery pressure
MTCT	-	Mother-to-Child transmission
NACO	-	National AIDS Control Organization
NHL	-	Non-Hodgkin lymphoma
NIH	-	National Institutes of Health
NK	-	Natural killer
NNRTIs	-	Non-nucleoside reverse-transcriptase inhibitors
NRTIs	-	Nucleoside reverse transcriptase inhibitors
NVP	-	Nevirapine
OR	-	Odds ratio

PAH	-	Pulmonary arterial hypertension
PCR	-	Polymerase chain reaction
PH	-	Pulmonary hypertension
PI	-	Protease inhibitor
PLHA	-	Person living with HIV and AIDS
POC	-	Point of care
RNA	-	Ribonucleic acid
SAT	-	Subcutaneous abdominal adipose tissue
SC	-	Subcutaneous
SD	-	Standard deviation
STD	-	Sexually transmitted disease
TB	-	Tuberculosis
TC	-	Total count
TDF	-	Tenofovir
TNF	-	Tumor necrosis factor
U.S.	-	United States
UNAIDS	-	United Nations Programme on HIV and AIDS
USPSTF	-	United States Preventive Services Task Force
VAT	-	Visceral adipose tissue
vs	-	Versus
WHO	-	World Health Organization
µg/min	-	Microgram per minute
µU/L	-	Microunit per litre

ABSTRACT

AIM:

The advances in diagnosis, treatment, monitoring of HIV infection and the availability of antiretroviral drugs have lead to improved survival of patients but this has resulted manifestations of late stage disease including cardiac manifestations. This study was intended to find out the cardiac manifestations in HIV patients and to correlate them with CD4 count.

MATERIAL and METHODS :

This was a cross-sectional prospective study carried out in 148 patients with serologically positive HIV status, aged between 18 to 60 years , admitted in BLDE's Shri B.M.Patil hospital with or without opportunistic infections. History collection, clinical examination, ECG, echocardiography, fasting lipid profile and other laboratory investigations were performed as a part of work up and all the patients were treated accordingly.

Results :

Majority of the patients were males (73%) and the commonest age group was 31 to 40 years (38.5%). The CD4 count between 50-199/cum in 33.8% and <50/cum was found in 27.7% of the patients. Cardiac manifestations were present in 56.1% of the patients. Sinus tachycardia was the commonest cardiac manifestation observed on ECG (17.6%). The other prominent cardiac manifestations included pericardial effusion (17.6%) , dilated cardiomyopathy (13.5%) , valvular heart disease (12.2%) , few patients has diastolic and

systolic dysfunctions (8%) and pulmonary arterial hypertension (2.7%). Cardiac manifestations were significantly high in patients with CD4 count <50/cum (74.42%; p=0.002).

Conclusion and interpretation

Patients with HIV infection are at higher risk of developing cardiac manifestations and reduced CD4 count poses maximum risk along with longer durations of HIV status and ART treatment

Keywords

Acquired Immunodeficiency Syndrome; Cardiac manifestations; Human immunodeficiency virus;

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INTRODUCTION

Acquired Immunodeficiency Syndrome (AIDS) caused by infection with human immunodeficiency virus (HIV) is characterized by an acquired, profound, irreversible, immune suppression that predisposes the patient to multiple opportunistic infections and progressive dysfunction of multiple organ systems [1]. HIV/AIDS is a global problem touching virtually every country and every family around the world. It continues to be a major global public health issue, having claimed more than 35 million lives so far. In 2016, 1.0 million people died from HIV-related causes globally.There were approximately 36.7 million people living with HIV at the end of 2016 with 1.8 million people becoming newly infected in 2016 globally [2]. 1 million people died from AIDS-related illnesses in 2016.

The WHO, African Region is the most affected region, with 25.6 million people living with HIV in 2016. The African region also accounts for almost two thirds of the global total of new HIV infections.[2]In 2016, there were 5.1 million people living with HIV in Asia and the Pacific. ▪ There were an estimated 270 000 new HIV infections in the region.[3]

According to the UNAIDS 2016 estimations India had 80,000 (62,000 – 1,00,000) new HIV infections and 62,000 (43000 - 91000) AIDS-related deaths. There were 21,00,000 (17,00,000 – 26,00,000) people living with HIV in 2016, among whom 49% (40% - 61%) were accessing antiretroviral therapy.[3]

HIV cannot be cured but can be suppressed by combination ART which controls viral replication within a person's body and allows an individual's immune system to strengthen and regain the capacity to fight with infections. However, its

prolonged usage can lead to metabolic abnormalities for which regular monitoring and evaluation is needed.[4]

Treatment with these potent ART has increased the survival of patient and transformed HIV into chronic illness which was otherwise a rapidly progressive fatal disease. Under the 2016 World Health Organization (WHO) guidelines, in mid-2017, 20.9 million people living with HIV were receiving antiretroviral therapy (ART) globally.54% of adults and 43% of children living with HIV are currently receiving lifelong antiretroviral therapy (ART). [2]

With the availability of a large armamentarium of ART drugs and recent advances in the diagnosis, treatment and monitoring of persons living with HIV and AIDS (PLHA),options with better tolerability, higher efficacy, and lower rates of treatment discontinuation there has been visible improved survival of such patients. Between 2000 and 2016, new HIV infections fell by 39%, and HIV-related deaths fell by one third with 13.1 million lives saved due to ART in the same period.[2]

Due to the longer survival of PLHA, the manifestations of late stage HIV infection are now being met with more commonly than before, which among is the HIV related cardiac diseases.[5] Although not fully recognized in the early days of HIV epidemic, cardiac involvement has been reported with increasing frequency in recent years.[5] The prevalence of cardiac involvement in AIDS patients have been reported to range between 28% and 73%.[5]The cardiac diseases in HIV infections include pericardial effusion, left ventricular dysfunction, myocarditis, dilated cardiomyopathy, endocarditis, pulmonary hypertension, malignant neoplasm, coronary artery disease and drug related cardiotoxicity.[6]

Patients with HIV/AIDS and symptoms suggestive of cardiac disease represent a diagnostic and therapeutic challenge in clinical practice; An

algorithmic,anatomic approach to diagnosis, localizing disease to the endocardium, myocardium and pericardium can be useful. An intimate knowledge of opportunistic infections affecting the heart, effects of HAART therapy and therapy for opportunistic infections on the heart is needed to be able to formulate a differential diagnosis.[5]

Effects of HAART therapy, especially protease inhibitors on lipid and glucose metabolism, and their influence on progression to premature vascular disease require consideration.

Considering the above facts the present study was planned to assess the various cardiac manifestations in patients with HIV infection and to correlate them with CD4 count.

OBJECTIVES

The objectives of the present study were;

1. To study various cardiac manifestations in patients with HIV infection.

2. To study the correlation of cardiac manifestations with CD4 count.

REVIEW OF LITERATURE

HUMAN IMMUNODEFICIENCY VIRUS:

HIV belongs to the family of retroviridae and a subfamily of lentiviruses. HIV is a blood-borne, sexually transmissible virus. The virus is typically transmitted via sexual contact (both heterosexual and male to male), shared intravenous drug users, and mother-to-child transmission (MTCT), which can occur during the birth process (Intrapartum/ Perinatally) or via breast milk. After 30 years of scrutiny there is no evidence that HIV is transmitted by casual contact or virus can be spread by insects such as mosquito bites.[7]

Two distinct species of HIV (HIV-1 and HIV-2) have been identified, and each is composed of multiple subtypes, or clades. All clades of HIV-1 tend to cause similar disease, but the global distribution of the clades differs.[4]

HIV I is divided into 4 Groups (M,N,O and P).

Subtype O shows 55 to 70% homology with subtype M.[4]

Another group of viruses labeled N for 'new' was reported in 1998.[4]

Group M (major) responsible for most of infections worldwide .M Group comprises of nine subtypes, designated A thorough K (Collectively referred to as group M with subtypes designation A, B, C, D, F, G, H, I, K & 60 circulating recombinant forms and numerous unique recombinant forms).[4]

Subtype C viruses (of the M group) dominates the global pandemic and is more transmissible than other subtypes, worldwide accounting for ≈ 50% of prevalent

infections worldwide. Subtype B viruses are predominant viruses seen in USA, Canada, Western Europe, Australia and account of 12 to 13% of global infection.[4]

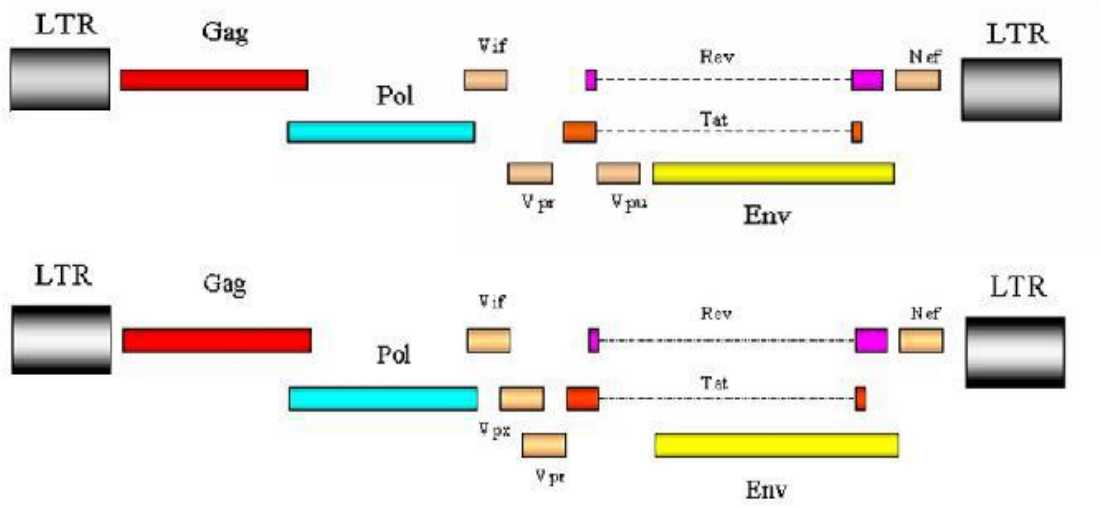


FIGURE 1. GENOME LAYOUT OF HIV–1 AND HIV-2[8]

Various subtypes of HIV-1 have been found in specific geographic areas and in specific high-risk groups. A person can be co-infected with different subtypes. In India Subtype C is predominant with smaller proportions of infections caused by A, G CRF02_A and other subtypes and recombinants.

HIV-2 was first identified in West Africa 1986.Comprises subtypes A through H.Compared with HIV I, HIV 2 infections are characterized by low viral loads, slow rates of clinical progression by low rates of transmission (vertically or sexually) and unique treatment recommendation due to intrinsic resistance to NNRTI's.[9]Despite slow rates of progression mortality rates are similar when adjusted for viral load.[9]

HIV 2 should be suspected in[9]

- Patients of West African origin
- Patients having undetectable virus without therapy
- Patients epidemiologically linked to HIV infection
- In cases where HIV I Western blot is negative, indeterminate or atypical.

HISTORY:

HIV-1 probably originated from one or more cross-species transferred from chimpanzees in central Africa[8]. HIV-2 is closely related to viruses that infect sooty mangabeys in western Africa.[8] Genetically, HIV-1 and HIV-2 are superficially similar, but each contains unique genes and its own distinct replication process.

TIMELINE OF HIV:-

- *1959* - The first known case of HIV in a human occured in a person who died in Congo, and was later confirmed to have HIV infection from his preserved blood samples.[10] The authors of the study did not sequence a full virus from his samples, hence stated that "attempts to amplify HIV-1 fragments of >300 base pairs were unsuccessful . However, after numerous attempts, four shorter sequences were obtained"; these represented small portions of two of the six genes of the complete HIV genome.[11]
- June 28, in New York City, Ardouin Antonio, a 49-year-old Jamaican-American shipping clerk died of *Pneumocystis carinii* pneumonia, a disease closely associated with AIDS. Gordon Hennigar, who performed the postmortem examination of the man's body, found "the first reported instance of unassociated *Pneumocystis carinii* disease in an adult" to be so unusual that he preserved Ardouin's lungs for later study. The case was published in two

medical journals at the time, and Hennigar has been quoted in numerous publications stating that he believed Ardouin probably had AIDS.[12]

- *1960s* – The HIV-2, a viral variant found in West Africa, was thought to have been transferred to people from sooty mangabey monkeys in Guinea- Bissau during that period.[12]
- *1964* - Jerome Horwitz of Barbara Ann Karmanos Cancer Institute and Wayne State University School of Medicine synthesized AZT under a grant from the US National Institutes of Health (NIH). AZT was originally intended as an anticancer drug.[12]
- *1966* - Genetic studies of the virus indicated that HIV first arrived in America, infecting one person in Haiti as many of the Haitians were working in Congo, providing the opportunity for infection.[12]
- *1968* - The disease spread from the 1966 American strand, but remained unrecognized for another 12 years.[12]
- *1969* - A St. Louis teenager, identified as Robert Rayford, died of an illness that baffles his doctors. Eighteen years later, molecular biologists at Tulane University in New Orleans test samples of his remains and find evidence of HIV.[12]
- *1975* - In the residents of Africa certain reports of wasting and other symptoms in patients were later seen who were later determined to have AIDS.[12]
- *1976* - Norwegian sailor Arvid Noe died and was later determined that he contracted HIV/AIDS in Africa during the early 1960s.[12]
- *1977* - Danish physician Grethe Rask died of AIDS contracted in Africa. A San Francisco prostitute gave birth to three children who were later diagnosed

to have AIDS. The children's blood was tested after their deaths and revealed an HIV infection. The mother died of AIDS in May 1987 and her test results showed HIV infection.[12]

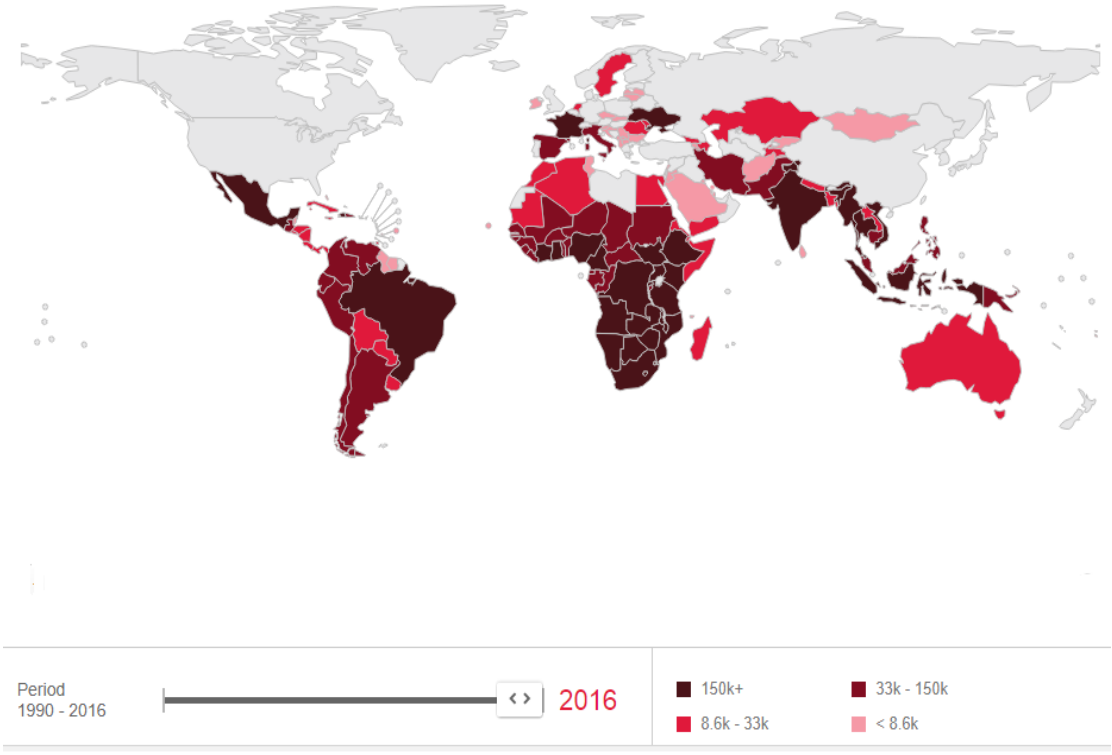
- *1978* - A Portuguese man known as Senhor José dies; he was later confirmed as the first known person with HIV-2 infection. It is believed that he was exposed to the disease in Guinea-Bissau in 1966.[12]
- *1979* - An early case of AIDS in the United States was of a female baby born in New Jersey in 1973 or 1974. She was born to a sixteen-year-old girl, an identified drug-injector, who previously had multiple male sexual partners.The baby died in 1979 at the age of five. Subsequent testing on her stored tissues confirmed that she had contracted HIV-1.[12]
- *Gaetan Dugas* was a Canadian who worked for Air Canada as a flight attendant. In March 1984, a Centers for Disease Control and Prevention (CDC) study tracking the sexual liaisons and practices of gay, and bisexual men in California, New York, and some other states found Dugas to be the center of a network of sexual partners, and he was dubbed "patient zero".[13]After the study, mistaken assertions claimed he brought the HIV virus to the U.S., but Dugas was not the initial carrier of the infection to North America.

EPIDEMIOLOGY:

Worldwide according to recent World Health Organization statistics (WHO) HIV continues to be a major global public health issue, HIV continues to be a major global public health issue, having claimed more than 35 million lives so far. In 2016, 1.0 million people died from HIV-related causes globally. There were approximately 36.7 million people living with HIV at the end of 2016. Africa is the most affected region, with nearly 1 in every 20 adults living with HIV.[2]

FIGURE:2

FIGURE 3 :PEOPLE LIVING WITH HIV



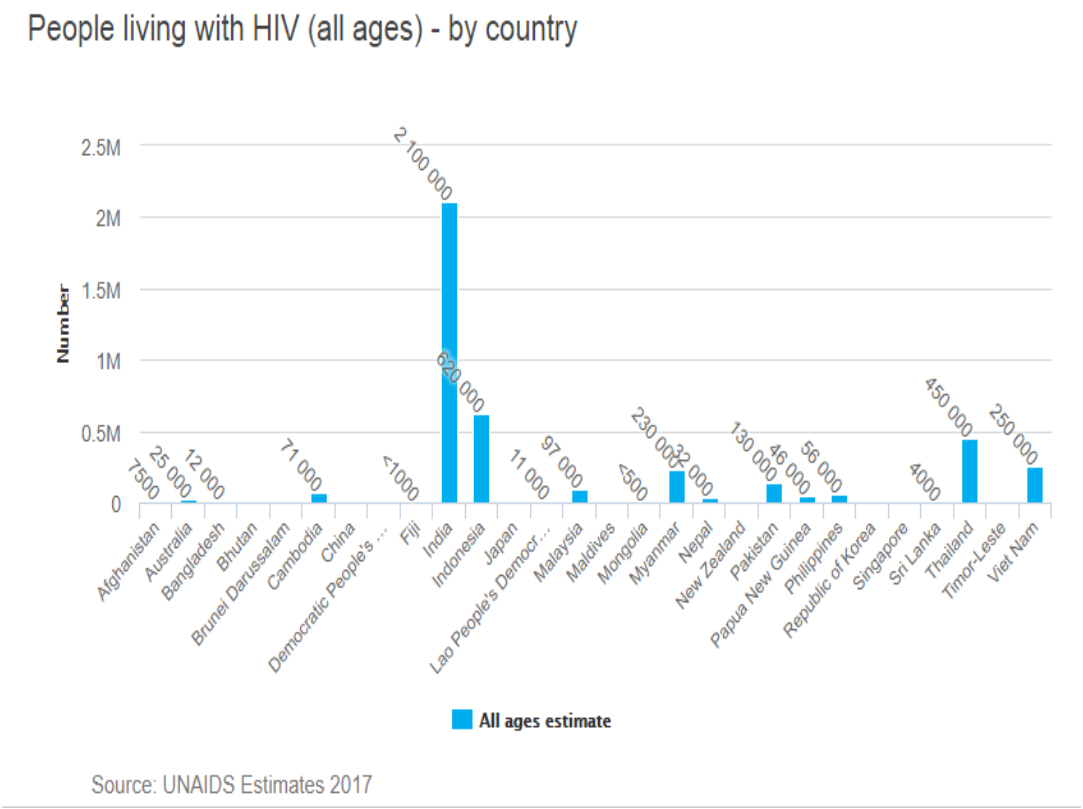
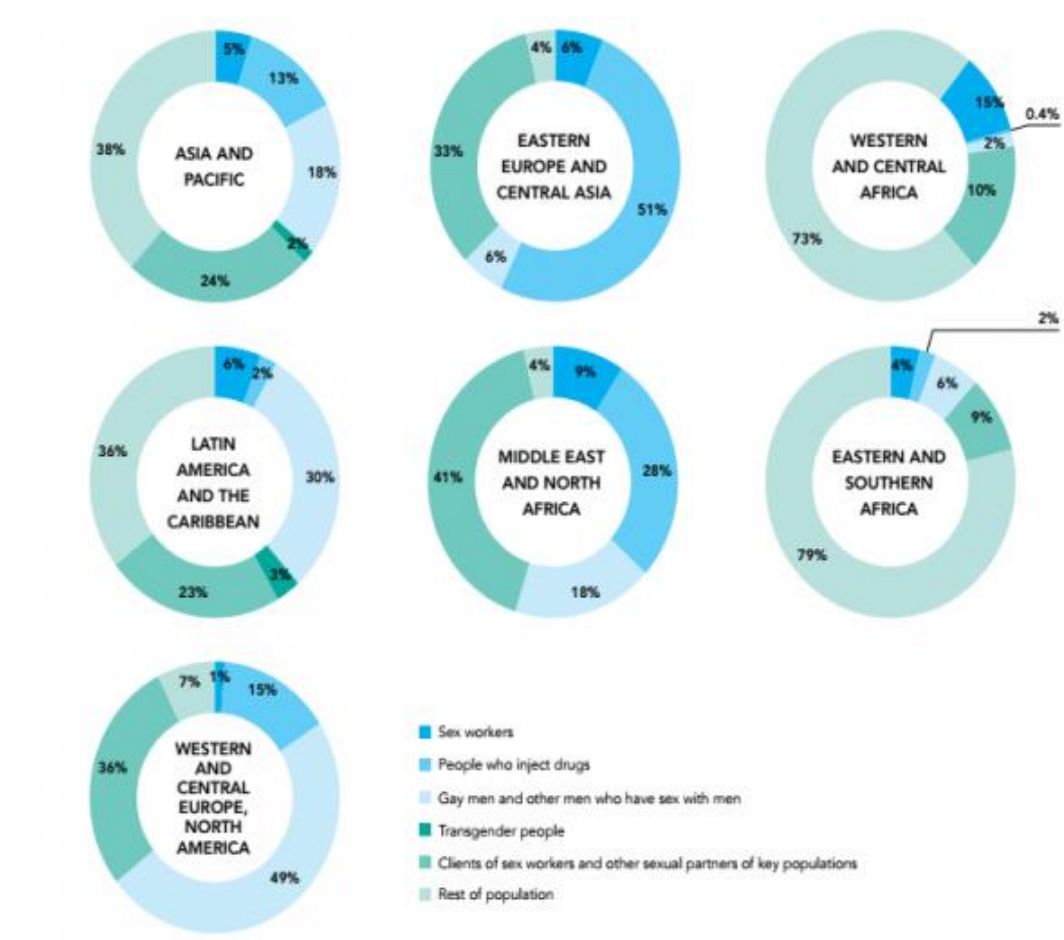

People living with HIV (all ages) - by country
Number
2.5M
2M
1.5M
1M
0.5M
0
7500
25 000
12 000
71 000
<1000
2 100 000
620 000
11 000
97 000
<500
230 000
32 000
130 000
46 000
56 000
4000
450 000
250 000
Afghanistan
Australia
Bangladesh
Bhutan
Brunei Darussalam
Cambodia
China
Democratic People's ...
Fiji
India
Indonesia
Japan
Lao People's Democr...
Malaysia
Maldives
Mongolia
Myanmar
Nepal
New Zealand
Pakistan
Papua New Guinea
Philippines
Republic of Korea
Singapore
Sri Lanka
Thailand
Timor-Leste
Viet Nam
All ages estimate
Source: UNAIDS Estimates 2017

FIGURE :4

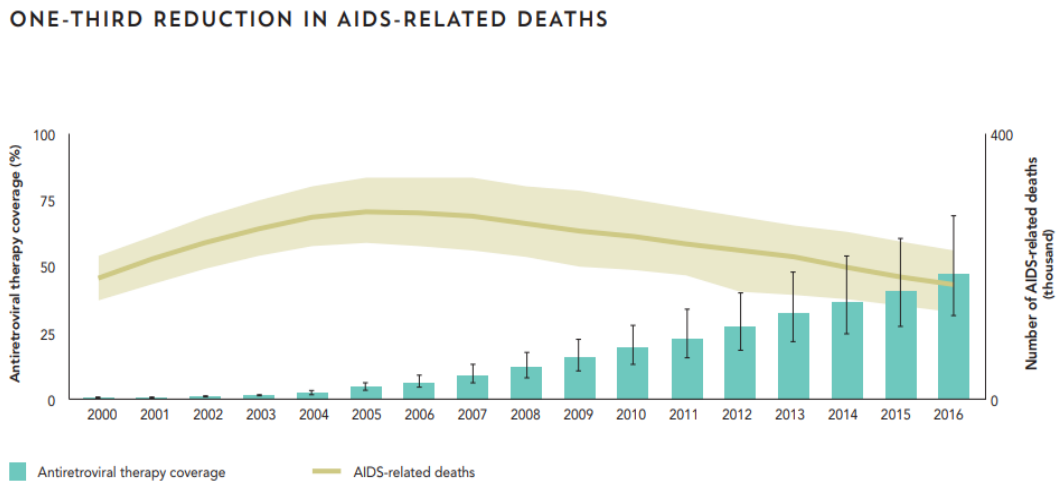
Distribution of new HIV infections among population groups, by region, 2014



Source: UNAIDS special analysis, 2016.

Methodological note: Estimated numbers of new HIV infections by key population were compiled from country Spectrum files submitted in 2015 to UNAIDS (2014 data), available modes-of-transmission studies and additional sources of data drawn from GARPR reports. Where data were lacking, regional medians were calculated from available data and applied to countries' populations.

FIGURE:5



ANTIRETROVIRAL THERAPY COVERAGE AND NUMBER OF AIDS-RELATED DEATHS, ASIA AND THE PACIFIC, 2000–2016

The wider availability of antiretroviral therapy has led to a nearly one-third reduction in deaths from AIDS-related illnesses in the region, down from an estimated 240 000 [190 000–300 000] in 2010 to 170 000 [130 000–220 000] in 2016. AIDS-related deaths fell by an estimated 52% in Myanmar, while AIDS-related deaths in Indonesia increased by 68% and in Pakistan by 319%.

Source: 2017 Global AIDS Monitoring. UNAIDS 2017 estimates.

FIGURE :6

The overall national prevalence of HIV in most countries in Asia and the Pacific remains low. However, considering the size of the regional population low prevalence translates into large numbers of people living with HIV. Low national prevalence also masks higher HIV prevalence and incidence rates in certain geographical areas and among key populations at higher risk.[14]

There are significant variations in HIV epidemics between and within countries. New HIV infections are concentrated among key populations at higher risk, which include people who inject drugs, female and male sex workers and their clients, men who have sex with men and transgender people. Other vulnerable populations include migrants, prisoners, intimate partners of key populations at higher risk and people working in certain industries such as mining, construction,transport services.[14]

In 2016, there were an estimated 5.1 million [3.9-7.2 million] people living with HIV in Asia and the Pacific region.There were an estimated 270000[190000-370000] new Hiv infections in this region and 170000[130000-

220000] people died of AIDS-related illnesses in 2016.Between 2010 and 2016, the number of AIDS-related deaths in the region decreased by 30%. [2]

India has 3rd largest epidemic in the world[3]. In 2016, India had 80 000 (62 000 - 100 000) new HIV infections and 62 000 (43 000 - 91 000) AIDS-related deaths. There were 2 100 000 (1 700 000 - 2 600 000) people living with HIV in 2016, among whom 49% (40% - 61%) were accessing antiretroviral therapy[3].

The key populations most affected by HIV in India are[3]:

- Sex workers, with an HIV prevalence of 2.2%.
- Gay men and other men who have sex with men, with an HIV prevalence of 4.3%.
- People who inject drugs, with an HIV prevalence of 9.9%.
- Transgender people, with an HIV prevalence of 7.2%.

RACIAL, SEXUAL, AND AGE-RELATED DIFFERENCES IN INCIDENCE:

United States, although is the greatest national funder of the HIV epidemic globally, it is still facing a major ongoing HIV epidemic itself. The United States of America (USA) currently has around 1.2 million people living with HIV. Nearly one in eight of these people are unaware they have HIV[1]. The size of the epidemic is relatively small compared to the country's population, but is heavily concentrated among several key affected populations. Most new HIV infections occur among men who have sex with men , with African American/black men who have sex with men most affected. African American/black heterosexual women are also disproportionately affected. [15]

In June 2001, the United Nations General Assembly declared HIV/AIDS to be "a global emergency."The impact of the HIV epidemic in the USA is more serious among some groups than others. These key affected populations can be grouped by transmission category (for example, men who have sex with men) but also by race, with people of colour having significantly higher rates of HIV infection over white Americans.[16]

A complex set of economic and socioeconomic factors drive risk to these populations, including discrimination, stigma, poverty and a lack of access to care.[16] Sexual networks is also a major determining factor, with populations at a high risk to HIV tending to have sex with people in their own communities. HIV continues to be a serious threat to the health of the Hispanic/Latino community. In 2014, Hispanic/Latino people accounted for almost a quarter of all estimated new diagnoses of HIV in the USA, despite representing about 17% of the total US population.[17]

In almost any jurisdiction in the US, there's a concentration of the epidemic in the correctional population[18]. Most (76%) of those living with HIV were male, and 69% of males were gay, bisexual, and other men who have sex with men [15].

In the developed world, HIV infection is much more common in males. Women made up 19% (7,529) of the 39,782 new HIV diagnoses in the United States in 2016. Heterosexual contact accounted for 87% (6,541) of HIV diagnoses among women.[19]

Males were also more likely to acquire HIV infection through contaminated blood products for treatment of hemophilia before universal testing of the blood supply was instituted. The risk of HIV exposure from factor VIII concentrates has been virtually eliminated by viricidal treatment of plasma-derived

factor VIII concentrates, as well as the introduction of recombinant factor VIII concentrates and the gradual elimination of albumin from the production process used for these products.

In the developing world, HIV infection is equally common in males and females. The primary route of HIV transmission in the developing world is heterosexual contact.Young adults tend to be at a higher risk of acquiring HIV, typically through high-risk activities such as unprotected sexual intercourse or intravenous drug use.

In 2015, youth aged 13 to 24[a] accounted for 22% of all new HIV diagnoses in the United States.[20]

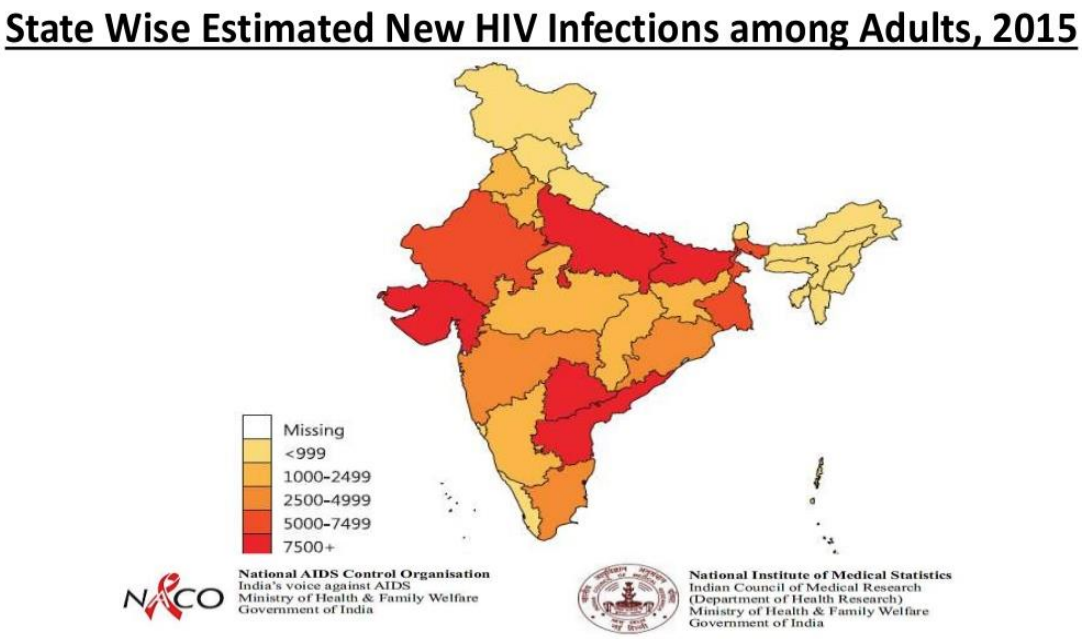


FIGURE:7, NACO STATEWISE REPORT

As per the India Estimation 2015 report, adult(15-49 years) HIV prevalence in India was estimated at 0.26% (0.22%-0.32%) .In 2015, adult HIV prevalence was estimated at 0.30 % among males and at 0.22% among Females.Among the States/UTs, in 2015 , Manipur has shown highest estimated adult

HIV prevalence of 1.15% followed by Mizoram(0.80%), Nagaland (0.78%), Andhra Pradesh & Telangana (0.66%), Karnataka(0.45%) , Gujarat(0.42%) and Goa(.40%).Andhra Pradesh and Telangana have 3.95 lakhs followed by Maharastra 3.01 lakhs,Karnataka 1.99 lakhs, Gujarat 1.66 lakhs , Bihar 1.51 lakhs and Uttar Pradesh 1.50 lakhs.These seven states together account for two thirds (64.4%)of total estimated PLHIV. [21]

ACCORDING TO KARNATAKA STATE AIDS PREVENTION SOCIETY :-

Status report on ART 2014-15.

Indicator	Adult Male	Adult Female	TS/TG	Child Male	Child Female	Total
Pre ART Registration	124383	120624	580	9261	7694	262542
Ever Started on ART	84148	77784	306	5084	3985	171307
Alive on ART	51333	56951	210	4039	3212	115745
Reported Death	24254	13965	70	681	522	39492

TABLE 1

TRANSMISSION:

HIV INFECTION CAN BE TRANSMITTED THROUGH:

- Unprotected sexual intercourse with an infected partner;
- Injection or transfusion of contaminated blood or blood products;
- Sharing unsterilized injection equipment that has been previously used by someone who is infected;
- Maternofetal transmission (during pregnancy, at birth, and through breastfeeding).[2]

The risk of occupational HIV transmission from contaminated needles to healthcare workers was found to be 0.3% (in case series performed prior to the availability of potent anti retroviral therapy).[2]

RISK FACTORS

Behaviours and conditions that put individuals at greater risk of contracting HIV include:[4]

- Having unprotected anal or vaginal sex;
- Having another sexually transmitted infection such as syphilis,herpes,chlamydia, gonorrhoea,and bacterial vaginosis;
- Sharing contaminated needles, syringes and other injecting equipment and drug solutions when injecting drugs;
- Receiving unsafe injections, blood transfusions, medical procedures that involve unsterile cutting or piercing; and Experiencing accidental needle stick injuries, including among health workers.

PATHOPHYSIOLOGY:

HIV produces cellular immune deficiency characterized by the depletion of helper T lymphocytes ($CD4^+$ cells). The loss of $CD4^+$ cells results in the development of opportunistic infections and neoplastic processes.[5]

VIROLOGY OF HIV:

Figure8: Scanning electron micrograph of HIV-1 budding (in green) from cultured lymphocyte[4]

HIV consists of a cylindrical center surrounded by a sphere-shaped lipid bilayer envelope. There are two major viral glycoproteins in this lipid bilayer, gp120 and gp41. The major function of these proteins is to mediate recognition of CD4+ cells and chemokine receptors, thereby enabling the virus to attach to and invade CD4+ cells. The inner sphere contains two single-stranded copies of the genomic material, RNA, as well as multiple proteins and enzymes necessary for HIV replication and maturation: p24, p17, reverse transcriptase, integrase, and protease .[7]

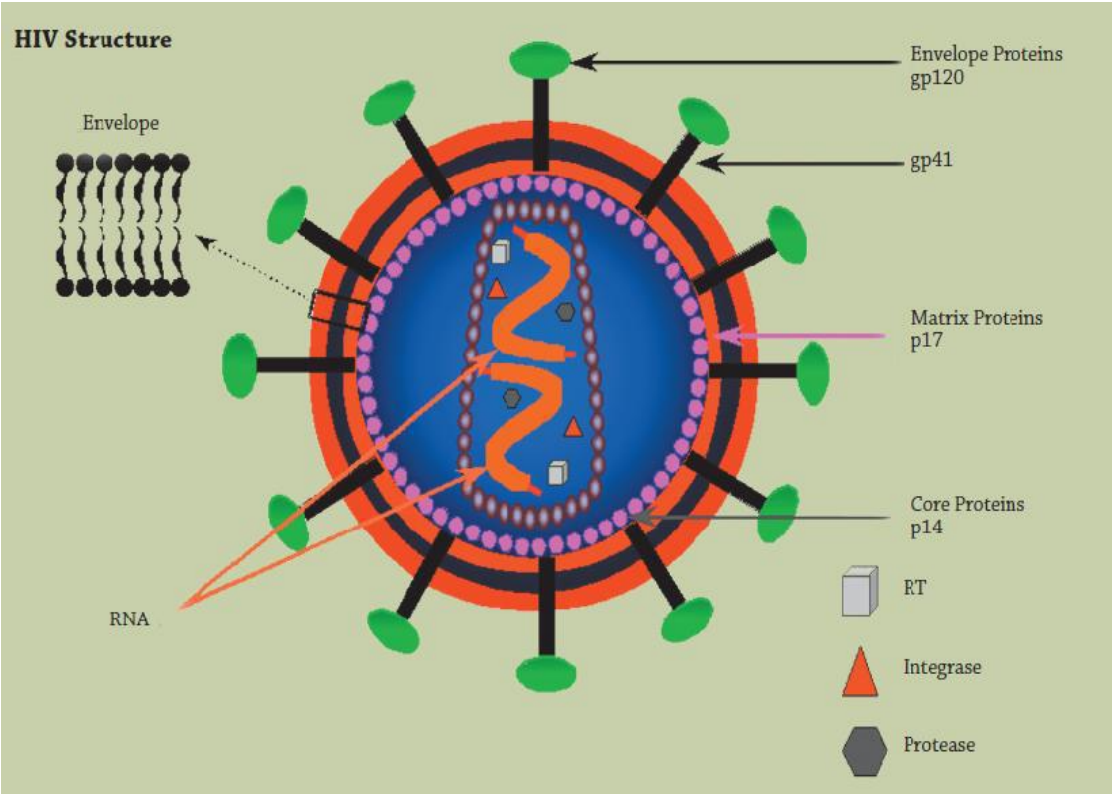


FIGURE 9: HUMAN IMMUNODEFICIENCY VIRUS ANATOMY[7]

Unlike other retroviruses, HIV uses nine genes to code for the necessary proteins and enzymes. The three principal genes are gag, pol, and env. The gag gene encodes core proteins. The pol gene encodes the enzymes reverse transcriptase, protease, and integrase. The env gene encodes the HIV structural components known as glycoproteins. The rest of the genes—rev, nef, vif, vpu, vpr, and tat—are important for viral replication and enhancing HIV's infectivity rate.

HIV'S LIFE CYCLE:

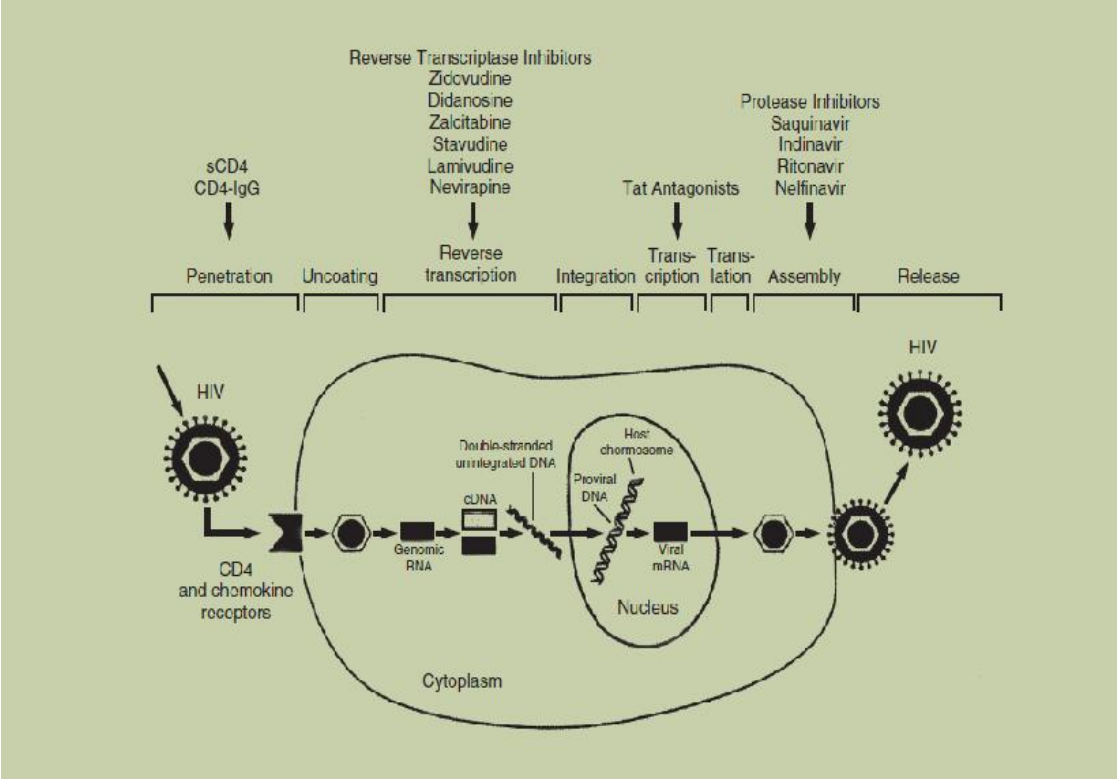


FIGURE 10: HIV LIFE CYCLE AND THE SITES OF ACTION OF ANTIRETROVIRAL AGENTS.[9]

Host cells infected with HIV have a shortened life span as a result of the virus's using them as "factories" to produce multiple copies of new HIV. Thus, HIV continuously uses new host cells to replicate itself. As many as 10 million to 10 billion virions (individual viruses) are produced daily. In the first 24 h after exposure, HIV attacks or is captured by dendritic cells in the mucous membranes and skin.Within 5 days after exposure, these infected cells make their way to the lymph nodes and eventually to the peripheral blood, where viral replication becomes rapid. CD4+lymphocytes that are recruited to respond to viral antigen migrate to the lymphnodes. These become activated and then proliferate via complex interaction of cytokines released in the microenvironment of the lymph nodes.[7] This sequence of

events makes the CD4+ cells more susceptible to HIV infection, and it explains the generalized lymphadenopathy characteristic of the acute retroviral syndrome seen in adults and adolescents. In contrast, HIV-infected monocytes allow viral replication but resist killing. Thus, monocytes act as reservoirs of HIV and as effectors of tissue damage in organs such as the brain.[7]

The HIV life cycle includes six phases: binding and entry, reverse transcription, integration, replication, budding, and maturation.[9]

BINDING AND ENTRY:

The envelope proteins gp120 and gp41 bind to CD4+ cell receptors and coreceptors on the outside of CD4+ cells and macrophages. The chemokine receptors CCR5 and CXCR4 facilitate viral entry. T-cell tropic viruses require CXCR4 to bind, and macrotropic strains of the virus require CCR5. R5 is the most common virus transmitted during acute infection, and later during infection X4 is the virus that is most common. The presence of a homozygous inactive mutation of the CCR5 allele has caused resistance to infection by the R5 virus.[9]

The joining of the proteins and the receptors and coreceptors fuses the HIV membrane with the CD4+ cell membrane, and the virus enters the CD4+ cell and macrophage. The HIV membrane and the envelope proteins remain outside of the CD4+ cell, whereas the core of the virus enters the CD4+ cell. CD4+ cell enzymes interact with the viral core and stimulate the release of viral RNA and the viral enzymes reverse transcriptase, integrase, and protease.[7]

REVERSE TRANSCRIPTION:

The HIV RNA must be converted to DNA before it can be incorporated into DNA of the CD4+ cell. This incorporation must occur for the virus to multiply. The conversion of HIV RNA to DNA is known as reverse transcription and is

mediated by the HIV enzyme reverse transcriptase. The result is the production of a single strand of DNA from the viral RNA. The single strand of this new DNA then undergoes replication into double-stranded HIV DNA.[7]

INTEGRATION

Once reverse transcription has occurred, the viral DNA can enter the nucleus of the CD4+ cell. The viral enzyme integrase then inserts the viral DNA into the CD4+ cell's DNA. This process is known as integration. The CD4+ cell has now been changed into a factory used to produce more HIV.[7]

REPLICATION

The new DNA, which has been formed by the integration of the viral DNA into the CD4+ cell, causes the production of messenger RNA that initiates the synthesis of HIV proteins.[7]

BUDDING

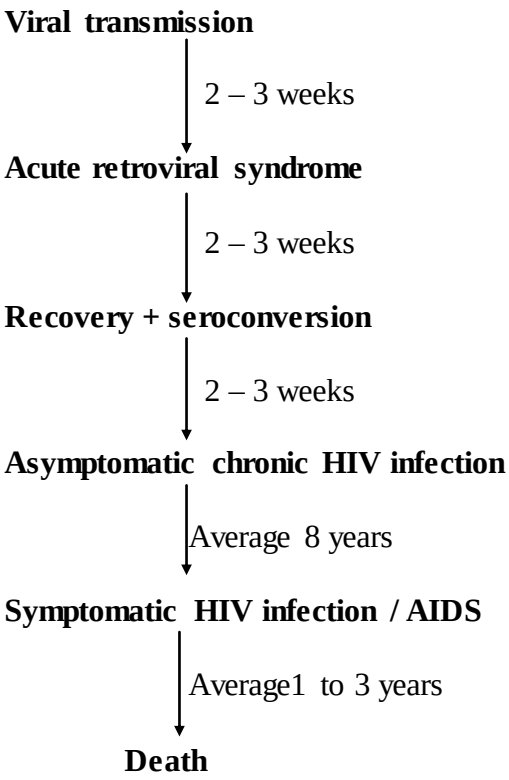
The HIV proteins and viral RNA, all the components needed to make a new virus, gather at the CD4+ cell membrane to form new viruses. These new viruses push through the different parts of the cell wall by budding. Many viruses can push through the wall of one CD4+ cell. These new viruses leave the CD4+ cell and contain all the components necessary to infect other CD4+ cells.[7]

MATURATION

The new virus has all the components necessary to infect other CD4+ cells but cannot do so until it has matured. During this process, the HIV protease enzyme cuts the long HIV proteins of the virus into smaller functional units that then reassemble to form a mature virus. The virus is now ready to infect other cells.[7]

NATURAL HISTORY:

The natural history of untreated HIV infection is divided into following stages.[4]



EFFECTS ON THE IMMUNE SYSTEM:

The pathogenesis of HIV is basically a struggle between HIV replication and the immune responses of the patient, via cell-mediated and immune-mediated reactions. The HIV viral burden directly and indirectly mediates CD4+ T-cell destruction. There is destruction of mature CD4+ cells; CD4+ progenitor cells in bone marrow, the thymus, and peripheral lymphoid organs; as well as CD4+ cells within the nervous system, such as microglia. The result of this destruction is failure of T-cell production and eventual immune suppression.9

There are many mechanisms of CD4+ cell depletion by HIV infection. Direct HIV-mediated cytopathic effects include single-cell killing as well as cell fusion, or syncytium formation. The syncytium is a fusion of multiple uninfected CD4+ cells with one HIV-infected CD4+ cell via CD4–gp120 interaction. This fusion results in a multinucleated syncytium, or giant cell, which may ultimately serve as a means to produce many virions. The host's natural immune responses also play a role in CD4+ cell depletion, mainly through cytotoxic CD8+ T-cells, antibody-dependent cellular cytotoxicity, and natural killer cells. Other mechanisms include autoimmune responses, anergy, superantigen-mediated activation of T cells, and programmed cell death (apoptosis).9HIV can infect many types of cells. The spread of HIV outside lymphoid organs to the brain, spinal cord, lung, colon, liver, and kidney usually occurs late during illness.[7]

The immune systems of HIV-infected children undergo changes that are similar to those in adults. B-cell activation occurs in most children early in the infection, evidenced by the presence of hypergammaglobulinemia (>1.750 g/L) with high levels of anti–HIV-1 antibody. This reflects both dysregulation of T-cell suppression of B-cell antibody synthesis as well as active CD4+ enhancement of B-

lymphocyte humoral response. Also, as HIV disease progresses through more severe immunosuppression and depletion of CD4+ cells, the CD8+ count increases,yielding an overall decrease in the CD4+:CD8+ ratio.[7]

CELLS SUSCEPTIBLE TO HIV INFECTION[7]

- Hematopoietic :T-cells (CD4+ OR CD 8+); Macrophages/monocytes; Dendritic cells; Fetal thymocytes and thymic epithelium; B-cells; NK cells; Megakaryotic cells; Stem cells;
- Central nervous: Microglia; Capillary endothelial cells; Astrocytes;Oligodendrocytes;
- Large Intestine: Columnar epithelium
- Other - Kupffer cells; Synovial cells; Placental trophoblast cells

CLINICAL CATEGORIES OF HIV INFECTION

Children infected with HIV often have severe disease when first evaluated, or they may develop AIDS over time, much like adults infected with HIV. Infants and young children normally have higher CD4+ counts than those of adults. The normal CD4+ count in children varies with age, but it is equal to the adult value by the time the child is 6 years old. Immunologic and clinical categories are used to evaluate the HIV disease status in children and to make treatment decisions.[7]

Primary infection refers to the time when HIV first enters the body. At the time of primary infection with HIV, a person's blood carries a high viral load, meaning that there are many individual viruses in the blood. The number of copies of virus per milliliter of plasma or blood can exceed 1 million. Newly infected adults often experience an acute retroviral syndrome. Signs and symptoms of acute retroviral

syndrome include fever, myalgia (muscle pain), headache, nausea,vomiting, diarrhea, night sweats, weight loss, and rash. These signs and symptoms usually occur 2–3 weeks after infection, subside after a few days, and often are misdiagnosed as influenza or infectious mononucleosis. An important differentiating symptom that is often absent is the presence of a runny nose or nasal congestion.[7]

During primary infection, the CD4+ count in the blood decreases remarkably but rarely drops to less than 200 cells/μL. The virus targets CD4+ cells in the lymph nodes and the thymus during this time, making the HIV-infected person vulnerable to opportunistic infections and limiting the thymus's ability to produce T-lymphocytes. HIV antibody testing using an enzyme-linked immunosorbent assay or enzyme immunoassay may yield positive or negative results depending on the time of seroconversion. DNA PCR and RNA PCR will be positive, but confirmation with Western blot analysis may yield an indeterminate result because seroconversion can take up to 2–8 weeks to occur. The average time to seroconversion is 25 days.[7]

CHRONIC INFECTION[32]

1. Chronic progressors

a. Typical disease progression

b. Usually with V_L > 10000 c/mL and CD4 decline of 50 – 100 cells/mm^3/years

2. Chronic Non Progressors

a. HIV infection without opportunistic infection and CD4 count > 500,>10 years

3. Slow progressors

a. Slow CD4 loss with VL 1000 – 10000 c/mL

4. ELITE controllers

a. V_L < 50 c/mL in absence of therapy make up < 0.5% of person with HIV infection

CLINICAL LATENCY/ASYMPTOMATIC DISEASE (CLINICAL STAGE 1)

Although patients recently infected with HIV usually experience a "clinically latent" period of years between HIV infection and clinical signs and symptoms of AIDS, evidence of HIV replication and host immune system destruction exists from the onset of infection. Early during this time, referred to as Clinical Stage 1 , the immune system produces antibodies in an attempt to protect itself from HIV. This is when the "viral set point" is established. The viral load of the set point can be used to predict how quickly disease progression will occur. People with higher viral load set points tend to exhibit more rapid disease progression than those with lower viral load set points.[7]

During latency, HIV-infected patients may or may not have signs and symptoms of HIV infection though persistent lymphadenopathy is common. In HIV infected adults, this phase may last 8–10 years. The HIV enzyme-linked immunosorbent assay and Western blot or immunofluorescence assay will be positive. The CD4+ count is greater than 500 cells/μL.[9]

MILD SIGNS AND SYMPTOMS OF HIV (CLINICAL STAGE 2)

HIV-infected people may appear to be healthy for years, and then minor signs and symptoms of HIV infection begin to appear. They may develop candidiasis, lymphadenopathy, molluscom contagiosum, persistent hepatosplenomegaly, popular pruritic eruptions, herpes zoster, and/or peripheral neuropathy. The viral load increases, and the CD4+ count falls is between 350-499/uL Once patients are in this stage they remain in stage 2. They can be reassigned stage 3 or 4 if a condition from one of those occurs, but they cannot be reassigned to Clinical Stage 1 or 2 if they become asymptomatic.[7]

ADVANCED SIGNS AND SYMPTOMS OF HIV (CLINICAL STAGE 3)

HIV-infected patients with weakened immune systems can develop life threatening infections. The development of cryptosporidiosis, pulmonary and lymph node tuberculosis, wasting, persistent fever (longer than one month), persistent candidiasis, recurrent bacterial pneumonia, and other opportunistic infections is common. These patients may present with wasting, or losing weight. Their viral load continues to increase, and the CD4+ count falls to less than 200-349 cells/μL.[7]

CLINICAL STAGE 4

Patients with advanced HIV disease, or AIDS, can continue to develop new opportunistic infections, such as Pneumocystis jirovecii pneumonia (formerly Pneumocystis carinii pneumonia), cytomegalovirus infection, toxoplasmosis, Mycobacterium avium complex,ryptococcal meningitis, progressive multifocal leukoencephalopathy, Kaposi sarcoma and other infections that commonly occur with a severely depressed immune system. The viral load is very high, and the CD4+ count is less than 200 cells/μL. At this point in the disease course death can be imminent.[7]

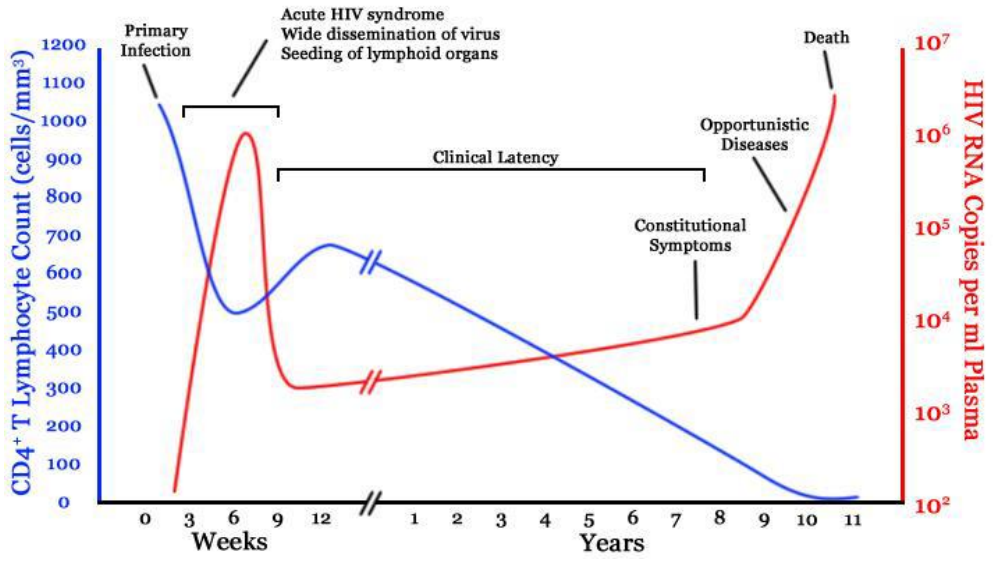


FIGURE 11. TIMELINE OF CD4 T-CELL AND VIRAL-LOAD CHANGES OVER TIME IN UNTREATED HUMAN IMMUNODEFICIENCY VIRUS INFECTION.[22]

IMMUNOLOGIC CONTROL OF HIV

The primary mechanism for immunologic control of HIV appears to be CD8+ cytotoxic T-cells. T-cell responses are correlated with the steady-state of viral load and hence, the rate of progression.[23] Cellular immunity is apparently responsible for some multiple-exposed, but uninfected individuals.[24] Although antibodies against HIV can be detected, it is clear that they are not sufficiently neutralizing to assist with immunologic control of the infection.

The role of NK (Natural Killer) cells may be important in the initial control of HIV. Escape mutations have been detected, implying that immunologic pressure on HIV exists from NK cells.[23]

WHO CLINICAL STAGING SYSTEM FOR HIV INFECTION AND HIV RELATED DISEASE:

World Health Organization has developed a clinical staging system (originally for prognosis), based on clinical criteria. The definition of symptoms, signs and diseases is according to clinical judgement. Clinical condition or performance score, whichever is the higher, determines whether a patient is at clinical stage 1, 2, 3 or 4. Clinical stage is important as a criterion for starting antiretroviral (ARV) therapy.[24]

TABLE 2:

Primary HIV Infection	• Asymptomatic • Acute retroviral syndrome
Stage 1	• Asymptomatic • Persistent generalized lymphadenopathy
Stage 2	• Moderate unexplained weight loss (<10% of presumed or measured body weight) • Recurrent respiratory tract infections sinusitis, tonsillitis, otitis media and pharyngitis) • Herpes zoster • Angular cheilitis • Recurrent oral ulceration • Papular pruritic eruptions • Seborrhoeic dermatitis • Fungal nail infections
Stage 3	• Unexplained severe weight loss (>10% of presumed or measured) • Unexplained chronic diarrhoea for longer than one month

	• Unexplained persistent fever (above 37.6°C intermittent or constant, for longer than one month) • Persistent oral candidiasis • Oral hairy leukoplakia • PulmonaryTB(current) • Severe bacterial infections (such as pneumonia, empyema,pyomyositis,bone or joint infection, meningitis or bacteraemia) • Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis • Unexplained anaemia (<8 g/dl), neutropaenia (<0.5 × 109 per litre) or chronic thrombocytopaenia (<50 × 109 per litre)
Stage 4	• HIV wasting syndrome • Pneumocystis pneumonia • Recurrent severe bacterial pneumonia • Chronic herpes simplex infection (orolabial, genital or anorectal of more than one month's duration or visceral at any site) • Oesophageal candidiasis (or candidiasis of trachea, bronchi or lungs) • Extrapulmonary tuberculosis • Kaposi's sarcoma • Cytomegalovirus infection (retinitis or infection of other organs) • Central nervous system toxoplasmosis • HIV encephalopathy • Extrapulmonary cryptococcosis including meningitis

	• Disseminated non-tuberculous mycobacterial infection • Progressive multifocal leukoencephalopathy • Chronic cryptosporidiosis (with diarrhoea) • Disseminated mycosis (coccidiomycosis or histoplasmosis) • Recurrent non-typhoidal Salmonella bacteraemia • Lymphoma (cerebral or B-cell non-Hodgkin) or other solid HIV associated tumours • Invasive cervical carcinoma • Atypical disseminated leishmaniasis • Symptomatic HIV associated nephropathy or symptomatic HIV associated cardiomyopathy

DIAGNOSIS

SCREENING FOR HIV INFECTION

The U.S. Preventive Services Task Force (USPSTF) strongly recommends that clinicians should screen for HIV in all adolescents and adults at an increased risk for HIV infection, including all pregnant women.25

The Centers for Disease Control and Prevention (CDC) recommends HIV screening for patients in all health-care settings, after the patient is notified that testing will be performed unless the patient declines (opt-out screening). The CDC recommends that persons at high risk for HIV infection to be screened for HIV at least annually.[26]

Citing the benefits of early diagnosis and treatment and the failure of risk based screening to identify a substantial proportion of HIV-infected patients early in

the disease, the American College of Physicians recommends that clinicians shall adapt to routine screening for HIV and encourage all patients to be tested.[26]

HIV TESTING AND COUNSELING[36]

People access HIV treatment and prevention through the gateway of HIV testing and counseling. Currently only 60% of people with HIV know their status. The remaining 40% (over 14 million people) still need to access HIV testing services[27].The people who do know often test late meaning that many people start treatment when they are already significantly immune compromised resulting in poor health outcomes and ongoing HIV transmission.[28]

WHO recommends all HIV testing services must follow the 5 Cs principles informed Consent, Confidentiality, Counselling, Correct test results, Connection (linkage to care, treatment and other services).

WHO recommends all forms of HIV testing should be voluntarily and adhere to five C's. Consent, confidentiality, counseling, correct test results and connections to core treatment and prevention.[28]

STANDARD HIV TESTS[29]

- Standard serologic test consists of screening EIA (Enzyme linked immunoassay performed in lab with whole blood) done as a rapid test at point of care (POC).
- The EIA tests are screening test and require confirmatory western blot
- EIA screening for anti HIV requires a repeatedly reactive test which is the criterion for WB testing. Western blot detects antibodies to HIV I protein.
- WB should be coupled with EIA due to 2% rate of false positive EIA tests.

- Standard serologic assays (EIA and WB or 1FA) show sensitivity in patients with established disease (>3 months after transmission) of 99.5% and specificity of 99.994%.

Positive test should be confirmed with repeat testing or with collaborating clinical or laboratory data.

False negative results

- Window period
- Seroconversion
- Infants
- Patients treated prior to seroconversion
- Patients with late stage disease
- Atypical host response
- Failure to mount immunologic response
- Rapid tests
- Technical or clinical error

False positive results

Reported to range from 0.0004 to 0.0007%

- Autoantibodies (EIA :class 2 antigens, Western blot: cross reactivity to p24/p55)
- Investigational HIV vaccines
- Factitious HIV infection
- False positive oral fluid or a Quick test
- Influenza vaccine
- False positive rapid screening tests
- Technical error

ALTERNATE HIV SEROLOGIC TESTS

- IFA
- Possible advantage – Simple, less expensive, more rapid than WB
- Disadvantages – Resource limited setting
- Home kits
- Saliva test
- Nucleic acid amplification testing
- Rapid test – Recommended for HIV screening as an alternative to EIA. Useful where rapid results are important
- Occupational exposure
- Pregnant women presenting in labour without testing
- Outreach clinics
- In emergency rooms / STD clinics (Where patients are unlikely to return for test results)

CD4 CELL COUNT[30]

This is a standard test to assess

- Prognosis for progression to AIDS to death.
- Formulate differential diagnosis in a symptomatic patient
- To make therapeutic decision regarding antiretroviral treatment and prophylaxis for opportunistic infection.

Technique

Standard technique uses flow cytology and hematology analyzers.

Normal values – Mean of 800 to 1050 with range of two standard deviation of approximately 500 – 1400 cell/mm3.

CD4 slope :

It refers to the rate of decline of CD4 counts. Sequentially collected data for 8729 untreated HIV infected patients from 20 cohorts in Cascade showed that median CD4 count at about 8 months post seroconversion was approximately 610 cells / mm3 With negative slope at 2 years of 130 cell/mm3 and a five year slope of 70 cells /mm3 /year.

Median baseline was about 40 cells/mm3 higher in women versus men and in persons (40 years versus 40 years).

Response to ART[30]

CD4 count typically increases by > 50 cells/mm3 at four to eight weeks after viral suppression with ART and then increases at a rate that correlates with time, baseline CD4 count and virologic suppression. With good virologic response the increase at one year averages 100 to 150 cells/mm3 at ≥ 5 years it averages 20 to 30 cells/mm3/year.

TREATMENT:

Combination antiretroviral therapy (cART) also referred as highly active antiretroviral therapy (HAART) is the cornerstone of management of patients with HIV infection.[31,32,33]

Suppression of HIV replication is an important component in prolonging life as well as in improving the quality of life in patients with HIV infection.[31,32,33]

The continuing success of potent antiretroviral therapy (ART) has resulted in dramatic reductions in HIV-associated morbidity and mortality. HIV-infected individuals are now living longer.33 Incidence of opportunistic infections has decreased in the HAART era in these patients and also the metabolic effects of the Highly Active Anti-Retroviral therapy (HAART), especially the protease inhibitors

(PI) become more prevalent in this patient population. Patients with HIV/AIDS are not only on HAART therapy, but also on prophylaxis for opportunistic infections,depending on their level of immunity competence and prior infections, thereby complicating the picture.[8,34]

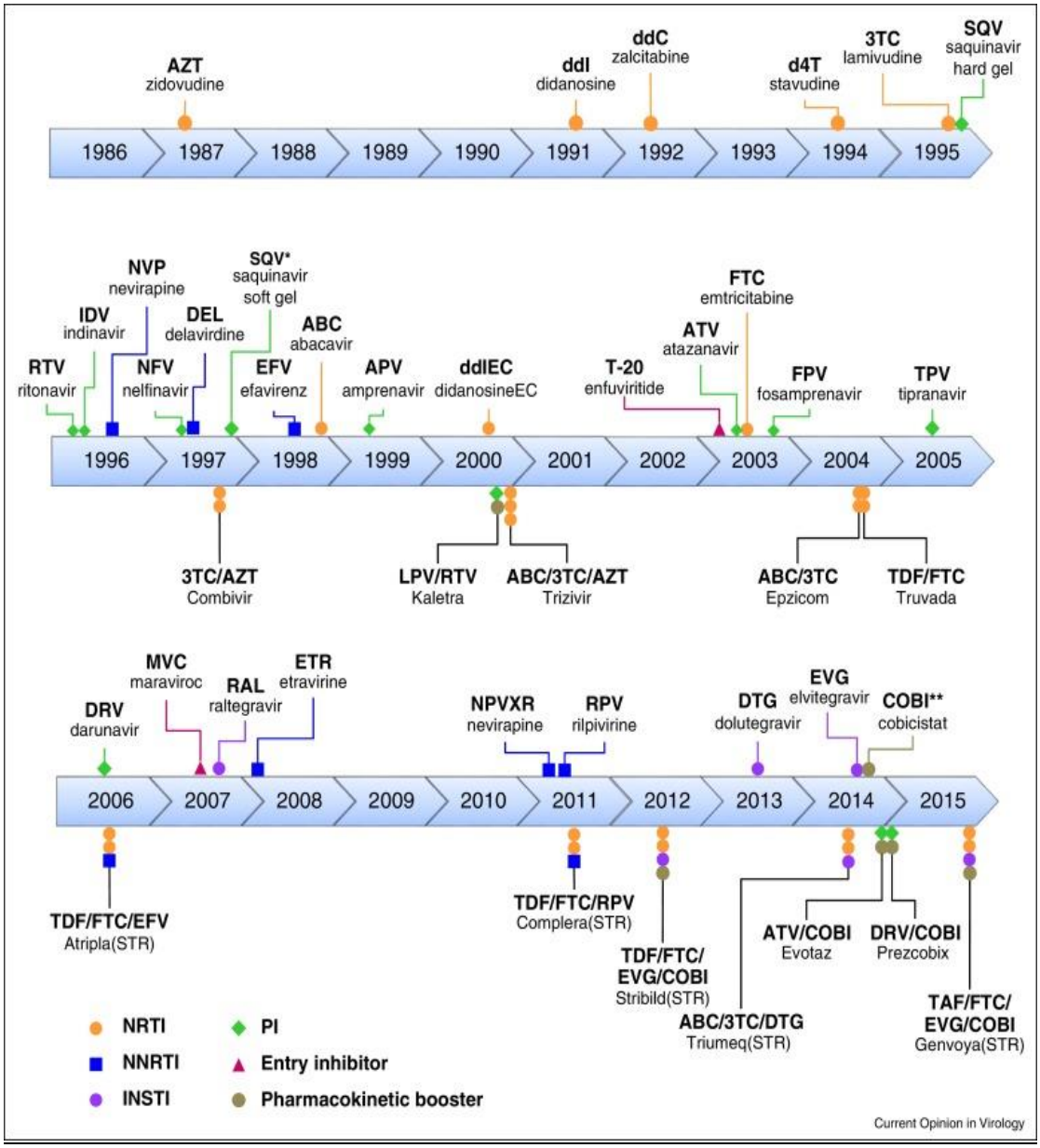


FIGURE 12. ANTIRETROVIRAL DRUGS APPROVED FOR HIV INFECTION[35]

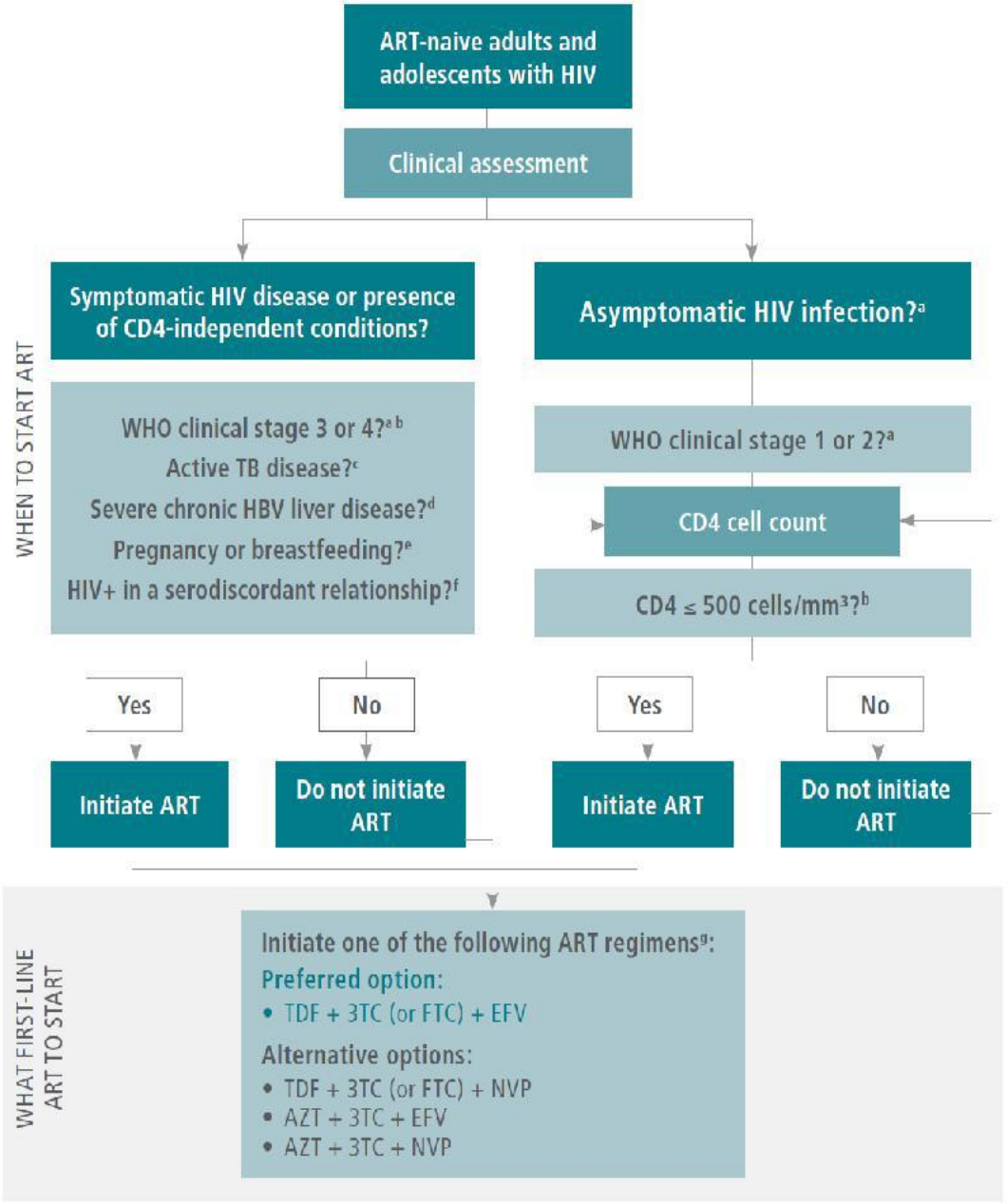


FIGURE :13. WHO 2013 GUIDELINES TO START ART IN PEOPLE LIVING WITH HIV[25]

HIV-RELATED CARDIOVASCULAR DISEASE

It is increasingly common for HIV/AIDS patients to be seen by cardiologists, and cardiovascular disease in HIV/AIDS is becoming increasingly recognized in the developing world[36,39,40] .Despite this, heart disease can be overlooked in HIV positive patients, because symptoms of breathlessness, fatigue, and poor exercise tolerance are frequently ascribed to other conditions associated with HIV infection[37].

Cardiac complications of HIV infection tend to occur late in the disease or are associated with related therapies and are therefore becoming more prevalent as therapy and longevity improve. Complicated drug therapies for HIV infection have sustained life but may increase cardiovascular risk and accelerate atherosclerotic disease and events.[4,39,40]

.A range of cardiac abnormalities associated with HIV infection has been suggested by studies; the conditions, in order of frequency, are pericardial effusion,lymphocytic interstitial myocarditis, dilated cardiomyopathy (frequently with myocarditis), infective endocarditis, and malignancy (myocardial Kaposi sarcoma and B-cell immune blastic lymphoma).[38,39,40]

CARDIAC MANIFESTATIONS OF HIV/AIDS[39,40]

PERICARDIAL EFFUSION

- Idiopathic
- Infectious (viral, bacterial especially tuberculous, and fungal)
- Neoplastic (Kaposi sarcoma and non-Hodgkin lymphoma)

HEART MUSCLE DISEASE

- Myocarditis (idiopathic/lymphocytic, specific infections, toxins)
- Dilated cardiomyopathy and LV dysfunction

ENDOCARDITIS

- Marantic (nonbacterial thrombotic endocarditis)
- Infective

TUMORS

- Kaposi sarcoma
- Lymphoma

RIGHT VENTRICULAR DYSFUNCTION & PULMONARY HYPERTENSION

- Primary
- Secondary (recurrent chest infections, thromboembolism)

PREMATURE ATHEROSCLEROSIS AND CORONARY ARTERY DISEASE

- Adverse drug effects
- Hyperlipidemia
- Proarrhythmia
- Vascular disease
- Autonomic dysfunction

HIV/AIDS AND THE PERICARDIUM[39,40]

Pericardial effusion and pericarditis were the most common cardiac abnormalities found in early HIV/AIDS autopsy studies. HIV-infected patients with pericardial effusions generally have a lower CD4 count than those without effusions,indicating more advanced disease. Effusions are generally small and asymptomatic.Asymptomatic pericardial effusions are common in HIV-infected

patients.The incidence of pericardial effusion in those with AIDS was 11%/year. The prevalence of effusion in AIDS patients increases over time, reaching a mean in asymptomatic patients of about 22% after 25 months of follow-up.

Pericardial effusion may be related to an opportunistic infection, metabolic abnormality, or malignancy, but usually the cause is not clear.Significant pericardial effusions are usually caused by viral or bacterial infection or malignant infiltration particularly with Kaposi sarcoma (KS) or non-Hodgkin lymphoma (NHL). In Africa, pericardial effusion itself is suggestive of HIV infection.

Pericarditis caused by Mycobacterium tuberculosis or Mycobacterium avium–intracellulare is a pressing problem and has been reported as the first manifestation of AIDS in mainland Europe and Asia.

Other unusual pathogens, including Nocardia asteroides and herpes simplex virus (HSV), should be considered along with CMV, which remains prevalent in the HIV population often without a definite anatomic site of infection. Other causes can include uremia from HIV-associated nephropathy or drug nephrotoxicity.

The effusion is often part of a generalized serous effusive process also involving pleural and peritoneal surfaces. This capillary leak syndrome may be related to enhanced cytokine production in the later stages of HIV disease.Effusion markedly increases mortality. These may, however, resolve spontaneously in up to 42% of patients. It is suggested that HIV should be considered as a differential in a young patient presenting with unexplained pericardial effusion or cardiac tamponade.

All HIV-infected patients with evidence of heart failure, Kaposi sarcoma,tuberculosis, or other pulmonary infections should undergo baseline echocardiography and electrocardiographic testing. Patients should undergo

pericardiocentesis if they have pericardial effusion and clinical signs of tamponade(e.g., elevated jugular venous pressure, dyspnea, hypotension, persistent tachycardia, pulsus paradoxus) or echocardiographic signs of tamponade (e.g., continuous-wave Doppler evidence of respiratory variation in valvular inflow, septal bounce, right ventricular diastolic collapse, a large effusion).

Patients with pericardial effusion without tamponade should be evaluated for treatable opportunistic infections, such as tuberculosis, and for malignancy. Highly active antiretroviral therapy (HAART) should be considered if therapy has not already been instituted. Repeated echocardiography is recommended after 1 month,or sooner if clinical symptoms direct.

It is pertinent to note that delay in initiation of antiretroviral therapy with antituberculous treatment because of the concern of IRIS did not confer any advantage as the randomized trial by Abdool Karim et al[41]recorded significant improvement in survival for patients with simultaneous treatment. Recurrence of TB may happen in HIV positive as compared with non-HIV patients even with successful initial therapy.[42]

HIV/AIDS AND THE MYOCARDIUM[39,40]

Diseases of the myocardium in patients with HIV/AIDS include cardiomyopathy, myocarditis, cardiac tumors and drug toxicity.

MYOCARDITIS[39,40]

Numerous pathologic studies have confirmed the presence of varying histologic patterns of lymphocytic myocarditis in HIV patients. The apparent difference in the prevalence of myocarditis in different studies can be related to clinical factors, sampling errors, and possibly the effect of HAART.

Estimates of the prevalence of myocarditis in HIV/AIDS varies from 53 percent in the pre-HAART era to much lower levels today in the developed world.

There are several hypotheses regarding the etiology of myocarditis in AIDS which includes:

a. Primary HIV myocarditis

b. Secondary HIV myocarditis,

c. Opportunistic infection

Autopsy has confirmed a variety of opportunistic infections of the myocardium in patients with AIDS. Infectious agents included Toxoplasma gondii in the hearts of both adults and children, Cryptococcus species, CMV, Candida species, Pneumocystis carinii, Microsporidium, Histoplasma capsulatum, atypical mycobacteria, and Aspergillus organisms involving the myocardium. The majority have been part of a disseminated infection and are infrequently associated with localized myocarditis.

d. Autoimmunity.

Myocarditis can be diagnosed clinically based on symptoms and physical findings, although this is often difficult in the HIV patient. The symptoms are protean and include fatigue, dyspnea, and pleuritic chest pain, which can wrongly be ascribed to other conditions. The finding of an unexplained tachycardia, third heart sound, or a friction rub should alert the physician to the possibility of myocarditis and guide investigation.

DILATED CARDIOMYOPATHY AND LEFT VENTRICULAR DYSFUNCTION IN HIV/AIDS[39,40]

Dilated cardiomyopathy as a complication of HIV infection was first described in 1986 and was identified frequently thereafter. The differential diagnosis

of HIV-related cardiomyopathy includes LV dysfunction secondary to ischemic heart disease, diabetes or hypertension, hypersensitivity reactions to drugs, or foreign injected material and coronary spasm secondary to cocaine use.

Isolated LV dysfunction in HIV-positive patients can resolve spontaneously, suggesting a self-limiting myocarditis, and reflects current thinking on the pathogenesis of non-HIV dilated cardiomyopathy. The presence of dilated cardiomyopathy is ominous and associated with poor survival compared to patients with structurally normal hearts. This poor outlook remained true, even after correcting for CD4 counts.

Mortality in HIV-infected patients with cardiomyopathy is increased independently of CD4 count, age, gender, and HIV risk group. The median survival to AIDS-related death was 101 days in patients with LV dysfunction and 472 days in patients with a normal heart at a similar stage of infection before HAART. Isolated right ventricular dysfunction or borderline LV dysfunction did not place patients at risk.

The mechanisms for the development of LV dysfunction, cardiomyopathy, in AIDS remain unclear. In addition to the role of HIV, lymphocytic myocarditis, and cytokines, the contributions of autoimmune responses, illicit and prescribed medications, nutritional deficiencies, and other factors also appear to be pathogenetically or pathophysiologically important.

DRUG-INDUCED HEART MUSCLE DISEASE[39,40]

Drug toxicity can cause clinically significant myocardial dysfunction; drugs specific to the HIV/AIDS patients that can cause heart failure include zidovudine,[43] interferon alpha, foscarnet, doxorubicin, pentamidine and amphotericin B. The exact role of zidovudine in the pathogenesis of HIV-related heart muscle

disease in humans remains unclear, and evidence for its existence is limited, however, small numbers of HIV-positive patients who have developed cardiac dysfunction while taking the drug were seen to improve following its discontinuation. It seems reasonable to discontinue zidovudine therapy for 1 month in those patients who develop cardiac dysfunction while receiving the drug.

However, this should be followed by reassessment and possible reintroduction of zidovudine if no improvement in cardiac function is noted. The cardiac and other side effects of NRTIs can become more common through improved AIDS survival and increasing cumulative doses of HAART.

TABLE 3:CARDIOVASCULAR ACTIONS AND INTERACTIONS OF DRUGS COMMONLY USED IN HIV THERAPY

Class	Cardiac drug interactions	Cardiac side effects
Antiretroviral		
Nucleoside reverse transcriptase inhibitors	Zidovudine, dipyridamole	Rare—lactic acidosis, hypotension; Accelerated risk with cardiopulmonary bypass; Zidovudine—skeletal muscle myopathy, myocarditis
Non-nucleoside reverse transcriptase inhibitors	Calcium channel blockers, warfarin, beta blockers, nifedipine, quinidine, steroids, theophylline; Delavirdine—can cause serious toxic effects if given with antiarrhythmic drugs and calcium channel blockers	
Protease inhibitors	Metabolized by cytochrome P-450 and interact with other drugs metabolized through this pathway, such as selected antimicrobials, antidepressant, and antihistamine	Implicated in premature atherosclerosis, dyslipidemia, insulin resistance, diabetes mellitus, fat wasting and redistribution

	agents; cisapride, HMG-CoA reductase inhibitors (lovastatin, simvastatin), sildenafil; Potentially dangerous interactions that require close monitoring or dose adjustment; can occur with amiodarone, disopyramide, flecainide, lidocaine, mexiletine, propafenone, quinidine; Ritonavir—most potent cytochrome activator (CYP3A) and P-glycoprotein inhibitor; most likely to interact; Indinavir, amprenavir, and nelfinavir—moderate; Saquinavir—lowest probability to interact; Indinavir, amprenavir, and nelfinavir—moderate; Calcium channel blockers, prednisone, quinine, beta blockers (1.5- to 3-fold increase); Decrease theophylline concentrations	
Anti-infective		
Antibiotics	Rifampin—reduces therapeutic effect of digoxin by inducing intestinal P-glycoprotein, reduces protease inhibitor concentration and effect; Erythromycin—cytochrome P-450 metabolism and drug interactions; Trimethoprimsulfamethoxazole (Bactrim)—increases warfarin effects	Erythromycin—orthostatic hypotension, ventricular tachycardia, bradycardia, torsades (with drug interactions); Clarithromycin—QT prolongation and torsades de pointes; Trimethoprimsulfamethoxazole—orthostatic hypotension, anaphylaxis, QT prolongation, torsades de pointes, hypokalemia; Sparfloxacin (fluoroquinolones)—QT prolongation
Antifungal agents	Amphotericin B—digoxin toxicity; Ketoconazole or	Amphotericin B—hypertension, arrhythmia, renal failure, hypokalemia,

	itraconazole—cytochrome P-450 metabolism and drug interactions; increases levels of sildenafil, warfarin, HMG-CoA reductase inhibitors, nifedipine, digoxin	thrombophlebitis, bradycardia, angioedema, dilated cardiomyopathy; liposomal formulations still have potential for electrolyte imbalance and QT prolongation; Ketoconazole, fluconazole, itraconazole—QT prolongation, torsades de pointes
Antiviral	Ganciclovir—zidovudine	Foscarnet—reversible cardiac failure, electrolyte abnormalities; Ganciclovir—ventricular tachycardia, hypotension.

NUTRITIONAL DEFICIENCIES AND CARDIAC DYSFUNCTION IN HIV/AIDS[39,40]

Nutritional deficiencies are common in HIV infection, particularly in those with late-stage disease. Poor absorption and diarrhea both lead to electrolyte imbalances and deficiencies in elemental nutrients. Deficiencies of trace elements have been associated with cardiomyopathy. For example, selenium deficiency increases the virulence of coxsackie virus to cardiac tissue. Selenium replacement reverses cardiomyopathy and restores LV function in nutritionally depleted patients. Levels of vitamin B12, carnitine, and growth and thyroid hormone can also be altered in HIV disease; all have been associated with LV dysfunction.

Experimentally, carnitine administration reversed myopathic changes induced by zidovudine (AZT) in vitro, but the clinical effects have yet to be established.

ENDOCARDIAL DISEASE IN HIV/AIDS

Endocardial/ valvular disease in patients with HIV/AIDS can be secondary to bacterial or non-bacterial (marantic) endocarditis.[44] Bacterial endocarditis is usually secondary to intravenous drug abuse in this patient population,[45] making Staphylococcus aureus and Streptococcus viridans the most common organisms and the tricuspid valve, the most common valve involved. Unlike in the myocardium, the HIV virus does not affect the endocardium directly. Non-bacterial (marantic) endocarditis is usually clinically silent, affects the tricuspid valve and can lead to embolism into the pulmonary artery, which is also clinically silent. The CD4 count has implications on the risk of developing heart disease, as well as on the prognosis.

Patients with lower CD4 count, especially less than 200, have a higher risk of endocarditis, and more importantly, patients with endocarditis and lower CD4 counts have a much poorer prognosis.[46] Treatment of infective endocarditis in HIVinfected patients does not differ from those who are HIV-negative.[6]Valvular heart disease happens mainly as either bacterial or fungal endocarditis. HIV disease generally does not seem to predispose to increased incidence of endocarditis. Further, patients with HIV have averagely similar manifestations and outcome (85% vs 93%) as compared with HIV-negative patients.[47] However, salmonellal endocarditis is more prevalent as compared with immunocompetent individuals.[48]

In addition intravenous drug users with advanced immunosuppression are more prone to develop IE. However, sustained valvular damage is less likely due to impaired immune response.

Gebo et al[49] noted decreased rates of endocarditis in a review of periods of pre-HAART versus post-HAART eras. They found a decrease in incidence from 20.5

to 6.6 per 1000 persons-years. Generally, mortality is higher in those with CD4 counts of below 200/mm3. Right-sided valves are most commonly affected with the predominant organism being Staphylococcus aureus in up to three-quarter of cases.[50]

Gram negative organisms and fungi also demonstrate higher incidence with mortality higher than in non-HIV patients if left valve is affected and CD4 count is less than 200 cells/mL.[50]

Bacterial pathogens isolated include *Salmonella species, Strepto-coccus pneumoniae* and *Haemophilus influenzae*. Organisms such as *Aspergillus species,Candida species* and *Cryptococcus neoformans* are fungal causes of endocarditis recorded in both IV and non-IV drug abusers in HIV patients.[50]

Further, Losa et al[51] had also re-ported eight cases of infective endocarditis from *Enterococus faecalis*, Staphylococci, *Salmonella enteritidis* and *Coxiella burnetii* in non-IV drug abusing HIV patients in a series over a period of twenty years.

Symptoms and signs of infective endocarditis include fever, lethargy and heart murmurs which were documented in one-third of patients. Repeated blood cultures and transoesophageal echocardiography are essential in reaching diagnosis.

Immediate and appropriate empirical therapy should be started promptly. Once sensitivity of the blood culture is available then therapy should be directed towards the result. Valve replacement surgery should be considered in patients with hemodynamic instability, antibiotic failure and profound valvular destruction. Prophylaxis for endocarditis in patients for dental surgery should be as per recommendation of established guidelines.[50]

CARDIAC TUMORS IN HIV/AIDS[39,40]

Cardiac tumors affecting the heart in patients with HIV/AIDS are more frequently secondary than primary, as in the general population.Kaposi sarcoma (KS) is the most common AIDS-related neoplasia, and in contrast to the classic dermatologic form of the disease, there is often widespread and potentially fatal visceral involvement in HIV-positive individuals

Kaposi's sarcoma is usually a part of disseminated mucocutaneous involvement, only rarely is the heart the sole site of involvement. Lesions are frequently asymptomatic.It affects up to 35% of AIDS patients, particularly homosexuals, with an incidence inversely related to the CD4 count. Autopsy studies have found that 28% of HIV-infected patients with widespread Kaposi sarcoma had cardiac involvement and rarely described it as a primary cardiac tumor.

Primary cardiac malignancy associated with HIV infection is generally caused by cardiac lymphoma. Non-Hodgkin lymphomas are 25 to 60 times more common in HIV-infected individuals. They are the first manifestation of AIDS in up to 4% of new cases. Patients with primary cardiac lymphoma can present with dyspnea, right-sided heart failure, biventricular failure, chest pain, or arrhythmias.

Cardiac lymphoma is associated with rapid progression to cardiac tamponade, symptoms of congestive heart failure, myocardial infarction (MI),, tachyarrhythmias, conduction abnormalities or superior vena cava syndrome.

HAART has not substantially affected the incidence of HIV-related non-Hodgkin lymphomas, but cumulative viremia has been associated, even during HAART therapy. An intracardiac mass in late-stage HIV infection is associated with a uniformly poor prognosis.

RIGHT VENTRICULAR DYSFUNCTION AND PULMONARY HYPERTENSION IN HIV/AIDS[39,40]

Patients with HIV/AIDS can develop pulmonary hypertension that is believed to be secondary to a combination of inflammation and genetic factors.Plexogenic arteriopathy has been described in this patient population.[6]

Primary pulmonary hypertension, occurs in less than 0.5% of patients with HIV infection,[52] the prognosis is usually poor. Echocardiography is useful for the diagnosis of pulmonary hypertension and to rule out secondary forms. Right heart catheterization remains the gold standard for diagnosis. Histologically, plexogenic arteriopathy is found most commonly, similar to the findings in immunocompetent patients.[6,52]

Thrombotic arterial lesions and venoocclusive disease occur far more rarely.Intravenous drug users are prone to the development of pulmonary hypertension,which may be related to intravenous injection of foreign material. This pulmonary hypertension is usually worsened by poor compliance. Treatment of pulmonary hypertension includes calcium-channel blockers, diuretics, anticoagulation, and prostacyclin analogues. The latter, specifically epoprostenol, efficiently reduces pulmonary artery pressure both acutely and in the long term in patients with HIV infection.[6]

The effect of HAART therapy on slowing the progression of pulmonary hypertension is a topic of current research. A study showed improvement of pulmonary arterial pressures with long term HAART therapy.[52] Pulmonary hypertension, associated with HIV/AIDS, differs from idiopathic/ primary pulmonary hypertension in terms of rapidity of progression, is unrelated to CD4 count and is associated with a worse prognosis compared to non- AIDS patients.Bosentan/ PDE

inhibitors and heart-lung transplant are usually the only treatment options that work in these sub-groups of patients.[52]

It has been shown that pulmonary hypertension (PH) is commoner in HIV patients as compared with the general population. Further another study had shown similar incidences despite the access to antiretroviral therapy. It is defined as a mean pulmonary artery pressure (mPAP) >25mmHg at rest with a mean pulmonarycapillary wedge pressure ≤15 mmHg or an mPAP with exercise >30mmHg.[50]

Causative factors implicated include lung infections, venous thromboembolism and left ventricular dysfunction. Animal model had shown that immune response to Pneumocystis jirovicei may be disturbed and prolonged with potential development of chronic disorder like pulmonary hypertension.[53] This novel finding should be evaluated by designing prospective, cohort study on patients who survive Pneumocystis pneumonia in humans. HIV-related PAH is mostly seen in young and male patients with major symptom being progressive shortness of breath, followed by non-productive cough, fatigue, syncope or near syncope and chest pain.[50]

Diagnostic tools employed include chest x-ray, electrocardiography and echocardiography, however, cardiac catheterization is mandatory to definitively diagnose the disease and exclude any underlying cardiac shunt. Modalities of treatment include individualized assessment for anticoagulation, vasodilator agents as tolerated, diuretics, oxygen as required and endothelin antagonists.[50]

A review of the HIV-PAH cases reported in the literature over a twenty-two year period showed a more favorable outcome in patients treated with PAH-specific therapy than in those treated with antiretroviral therapy only[54]. Nevertheless,HAART could delay the development of PAH in HIV-infected patients and is recommended independent of the CD4 counts.[50]

DISORDERS OF RHYTHM ASSOCIATED WITH HIV INFECTION[39,40]

Sudden death and rhythm abnormalities are common in HIV infection and account for up to 20 percent of cardiac-related deaths in this group of patients. These can be secondary to other cardiac pathology, or be a consequence of some forms of treatment.

Concomitant electrolyte disturbance can be important in the development of cardiac arrhythmia, and careful evaluation of the QT interval and magnesium concentration should be used as a guide to cardiac toxicity. ECG abnormalities and rhythm disturbances are not uncommon findings in HIV-positive patients with myocarditis or heart muscle disease, and ectopic beats, ventricular tachycardia, and sudden death have all been reported.

ACCELERATED ATHEROSCLEROSIS AND CORONARY HEART DISEASE IN HIV/AIDS:

On one hand, although HAART therapy slows the progression to HIV associated cardiomyopathy, HAART therapy, especially the protease inhibitors, have clinically significant effects on metabolism; causing hyperlipidemia, insulin resistance, lipodystrophy and hyperglycemia. Different classes of HAART appear to have varying effects on the lipid profile, notably, PIs raising low density lipoproteins (LDL) and NNRTIs raising HDL cholesterol. Accelerated atherosclerosis appears to be one of the unexpected side-effects of HAART. The relationship between antiretroviral therapy and coronary artery disease is a topic of much debate and uncertainty. Suffice, to say, the current literature suggests that HAART therapy decreases cardiovascular risk in the short term, but prolonged use of HAART therapy, especially protease inhibitors has been shown to be associated with increased risk of CAD/ MI.[6]

Patients on HAART therapy have a 26% increased relative risk of a myocardial infarction, per year of treatment.[55] More recently, it has also been shown that NNRTIs have a low to no increased risk of myocardial infarction compared to protease inhibitors.[56]

It has also been shown that Ritonavir, protease inhibitors, is associated with increase in carotid intimal wall thickness. The incidence of peripheral arterial disease appears to be increased in this patient population, independent of traditional cardiovascular risk factors.[8]

There is substantial clinical evidence for the development of vascular disease in HIV infected patients. The large vessel vasculitis involving the aorta and its major branches is increasingly being recognized in young Africans who have no evidence of atherosclerosis, syphilis or any other cause of vascular disease.[50]

The vascular disease shows similar histology to the intracranial variant, HIV associated intracranial aneurysmal vasculopathy, first described in children in the 1980s and over the last decade has been increasingly reported in adult patients.[50]

The typical pathologic process has been described as either an idiopathic focal necrotizing vasculitis with aneurysmal dilatation or a granulomatous vasculitis with fibro proliferative occlusion. Interestingly, vasculopathy is also observed in simian immunodeficiency virus-infected rhesus monkeys and in a mouse model of HIV vasculopathy using a defective HIV pro-virus. The transgenic mice develop a diffuse vasculopathy with intimal hypertrophy, primarily a result of smooth-muscle proliferation, disruption of the elastic lamina and fibrosis of the media and adventitia– findings similar to those seen in HIV-associated vasculopathy.[51]

CLINICAL APPROACH TO HEART DISEASE IN HIV/AIDS PATIENTS[39,40]

The history and physical examination must be used to detect symptoms and signs of cardiovascular disease in patients with HIV/AIDS. The history must include details of previous opportunistic infections, traditional risk factors for atherosclerosis, details regarding present and prior anti-retroviral therapy. One of the important questions clinicians should ask themselves is whether an HIV-positive individual is immunocompetent or immunodeficient- on the basis of a recent CD4 count and if not available, this would be necessary for further diagnostic evaluation and decisions regarding treatment and prognosis.

If the patient is not already on anti-retroviral therapy and presents with cardiac symptoms, this may require referral to an infectious disease specialist for decision making regarding anti-retroviral therapy. Co-ordination of care between infectious disease and cardiology can improve the quality of care and aid in developing an individualized treatment plan based on all of the above factors.

Routine use of electrocardiography or echocardiography in these patients is discouraged, especially because of the lack of evidence for finding sub-clinical disease. Shortness of breath is a common complaint, and in patients with HIV/AIDS,requires consideration of cardiomyopathy and pulmonary hypertension as possible etiologies. Transthoracic echocardiography is required for further evaluation.

Drug therapy of for heart failure is not different from HIV-negative individuals, except for consideration of drug-drug interactions, especially with antiretroviral therapy. Endomyocardial biopsy may be needed in HIV/AIDS patients with ventricular dysfunction on echocardiography to identify potentially treatable causes of myocarditis/cardiomyopathy.

Lastly, cardiotoxic medications may need to be stopped in patients who have pre-existing or those who have developed significant cardiovascular disease.

Management of pericardial disease, especially tuberculous effusions, is different in this patient population: addition of steroids is indicated and pericardiocentesis is needed even in the absence of tamponade.

Treatment of endocarditis does not differ from HIV negative individuals. In this new era of significantly improved prognosis in patients with HIV/AIDS, both cardiac procedures and cardiovascular surgery, including valve replacement and coronary artery bypass grafting should be done in these patients, except in the setting of advanced immunosuppression or high risk of mortality from AIDS related complications. Increased incidence of coronary artery disease, peripheral vascular disease and deep venous thrombosis has been shown in this patient population and requires careful consideration of the adverse effects of the different classes of anti-retroviral therapy.

A patient with HIV/AIDS and symptoms suggestive of cardiac disease, a growing problem, represents a diagnostic and therapeutic challenge in clinical practice. An intimate knowledge of opportunistic infections affecting the heart, effects of HAART therapy and therapy for opportunistic infections on the heart need to be considered in the differential diagnosis. Effects of HAART therapy, especially protease inhibitors on lipid and glucose metabolism, and their influence on progression to premature vascular disease require consideration. Finally, management of these patients can vary from non-infected patients, based on drug interactions, differences in responsiveness, and other factors; and this area requires further research.

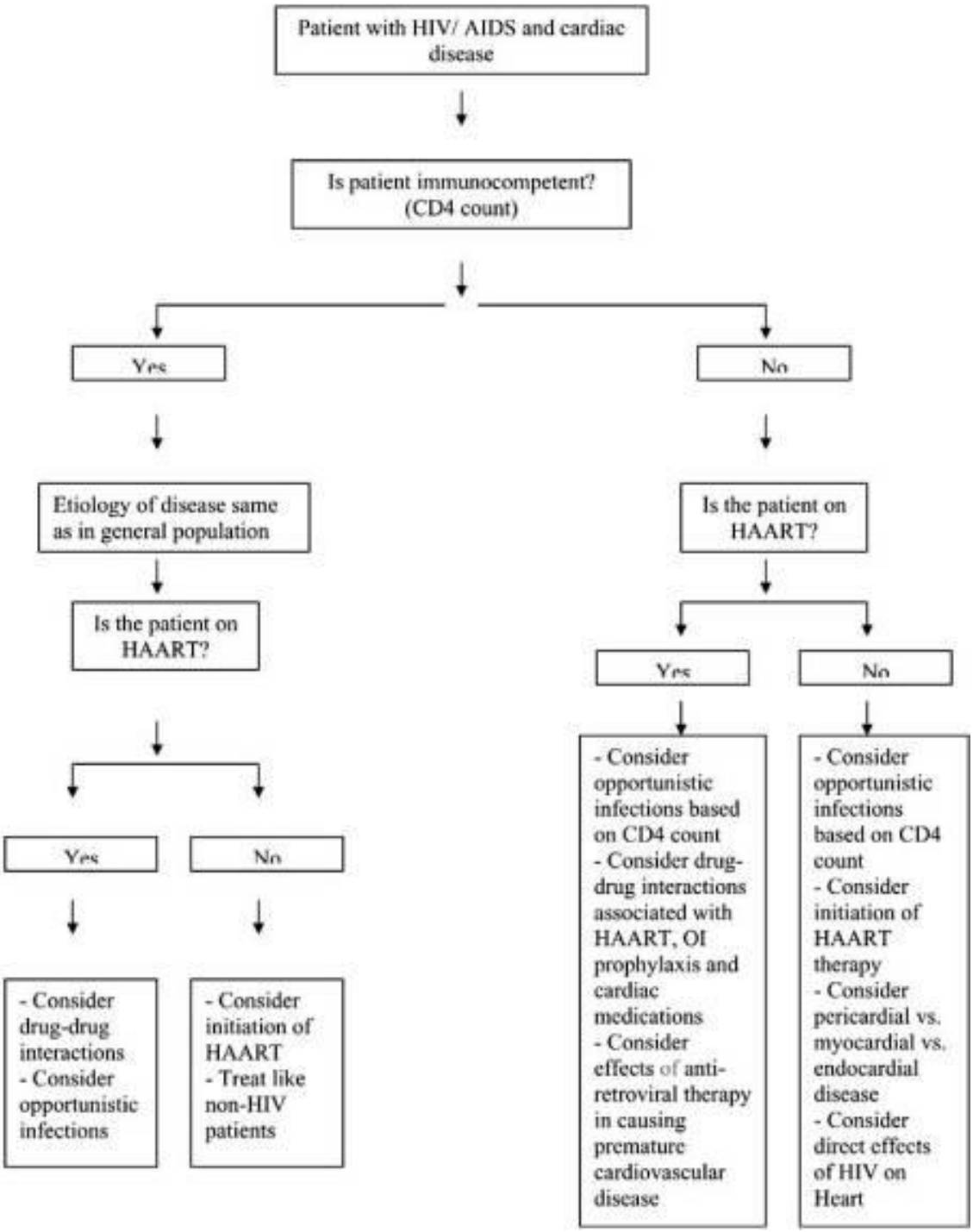


FIGURE 14. AN ALGORITHMIC APPROACH TO CARDIAC PROBLEMS IN HIV/AIDS [6]

MATERIALS AND METHODS

SOURCE OF DATA:

- The study will include outpatients and inpatients of BLDEU Shri B.M.Patil Medical College hospital and research centre, Vijayapura who are diagnosed as sero-positive for HIV.
- The patients will be informed about study in all respects and informed consent will be obtained.
- Period of study will be from September 2015 to March 2018.

SAMPLE SIZE:

- With 95% confidence level anticipated prevalence of non-cardiac HIV patients as 89% among total HIV patients and desired precision as ±5%.
- Minimum sample size is 116 (with finite population correction)

THE FORMULA:-

$$n=NZ^2 P(1-P) / [d^2(N-1) + Z^2 P(1-P)]$$

n=population size(with finite population correction)

N=population size(500 approx/year from hospital records)

Z=z static for level of confidence

P=expected proportion

d= precision

STATISTICAL ANALYSIS:

Data will be analyzed using

- Mean ± SD
- Diagrams
- Correlation coefficient
- Chi-square test (if necessary)

METHOD OF COLLECTION OF DATA

STUDY DESIGN

A cross sectional study

SAMPLE COLLECTION

A detailed history, general physical examination, systemic examination and investigations will be performed on all patients having HIV and fulfilling the inclusion criteria, both sex who are admitted in BLDEU's Shri B M PATIL Medical college, Hospital and Research Centre Vijayapura, between September 2015 to March 2018 .Every case will be included after detailed history, clinical examination and laboratory investigations. Oral and written consent will be taken from the subjects prior to the collection of specimens.

INCLUSION CRITERIA:

- ALL HIV SEROPOSITIVE PATIENTS.
- AGE MORE THAN 18YEARS

EXCLUSION CRITERIA:

- AGE LESS THAN 18 YEARS AND MORE THAN 60 YEARS
- PREVIOUSLY KNOWN TO HAVE FOLLOWING

1. ISCHEMIC HEART DISEASE
2. HYPERTENSIVE HEART DISEASE,
3. RHEUMATIC HEART DISEASE
4. CONGENITAL HEART DISEASE

INVESTIGATIONS

1. CD4 count
2. Complete blood count (with differential counts)
3. Blood urea and Serum Creatinine
4. Random Blood Sugar
5. liver function tests and Fasting lipid profile
6. An erect x-ray chest on deep inspiration
7. ECG and 2D Echo-Cardiograph with color flow Doppler

   Along with the above investigations other relevant investigations will be performed if required.

PROCEDURE

CD4 count was done for all patients using flowcytometry using a BD FACS Count system. The CD4 count was done using kits supplied by the National AIDS Control Organisation of India (NACO) to Anti Retroviral Therapy (ART) Centres.

The ECG was done on 12 lead surface ECG machine. All the patients were evaluated using M Mode and Two dimensional transthoracic echocardiography and colour flow doppler examination. Each two dimensional study consist of parasternal long and short axis, and apical two and four chamber views.

Investigations or interventions required in this study are routine standardized procedures. There is no animal experiment involved in this study.

OUTCOME VARIABLES:

The ECG findings were noted.The 2D Echocardiography findings were evaluated as for pericardial effusion, dilated cardiomyopathy, systolic/diastolic dysfunction, regional wall motion abnormalities, clot, vegetation and ejection fraction

STATISTICAL ANALYSIS

The data obtained was coded and entered into Microsoft Excel Worksheet (Annexure III). The categorical data was expressed as rates, ratios and proportions and comparison was done using chi-square test. The continuous data was expressed as mean ± standard deviation (SD) and comparison was done using independent sample 't' test. A probability value ('p' value) of less than or equal to 0.05 was considered as statistically significant.

RESULTS

The present cross-sectional study titled "A Study of Cardiac Manifestations in HIV Patients" was carried out in the department of medicine ,BLDE's Shri B.M.Patil Hospital. During the study period from September 2015 to March 2018, a total of 148 HIV positive patients were studied. The findings /observations and final results are tabulated as below.

TABLE 4: **TOTAL NUMBER OF PATIENTS**

	N
TOTAL NUMBER OF PATIENTS	148

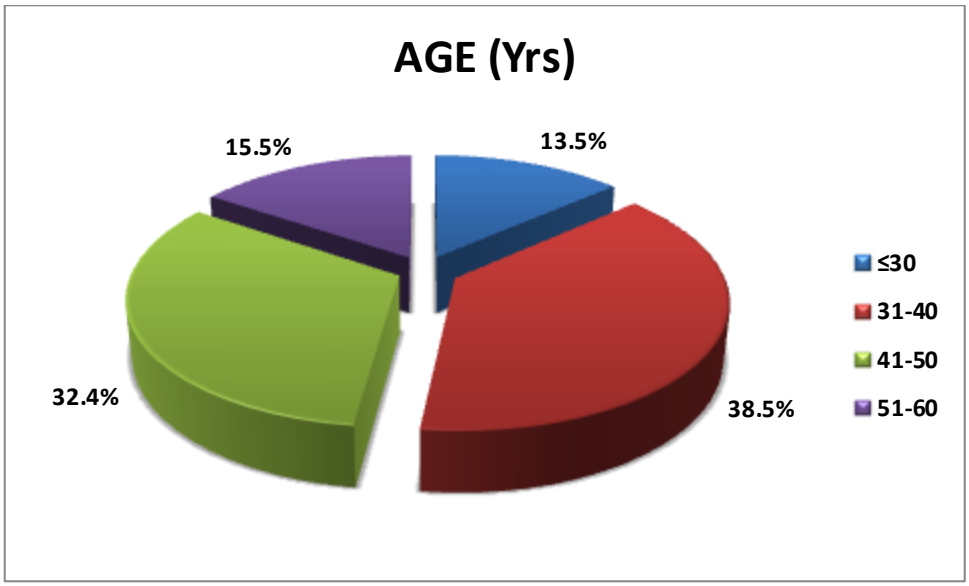
TABLE 5.1: DESCRIPTIVE STATISTICS OF AGE

	Min	Max	Mean	SD
AGE (Yrs)	18	60	41.0	10.0

TABLE 5.2: DISTRIBUTION OF CASES ACCCORDING TO AGE

AGE (Yrs)	N	%
≤30	20	13.5
31-40	57	38.5
41-50	48	32.4
51-60	23	15.5
Total	148	100

CHART 1: DISTRIBUTION OF CASES ACCCORDING TO AGE

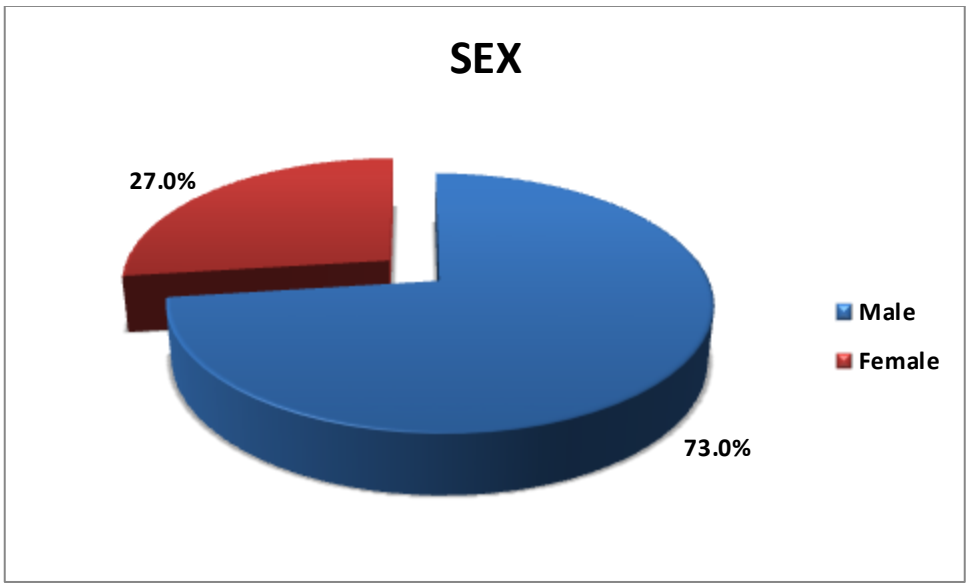


Patients age ranged from 18 to 60 years, maximum number of cases were in the age group of 31 to 40 that is 57 patients (38.5%), between 41-50 years 48 patients (32.4%), between 51-60 years 23 patients (15.5%), below 30 years 20 patients (13.5%). The mean age of the study population was 41 years.

TABLE 6: DISTRIBUTION OF CASES ACCCORDING TO SEX

SEX	N	%
Male	108	73
Female	40	27
Total	148	100

CHART 2: DISTRIBUTION OF CASES ACCCORDING TO SEX



Out of 148 patients 108 (73%) were males and 40 patients (27%) were females, accounting a ratio of male to female 2.7:1.

Inference : Male preponderance was observed.

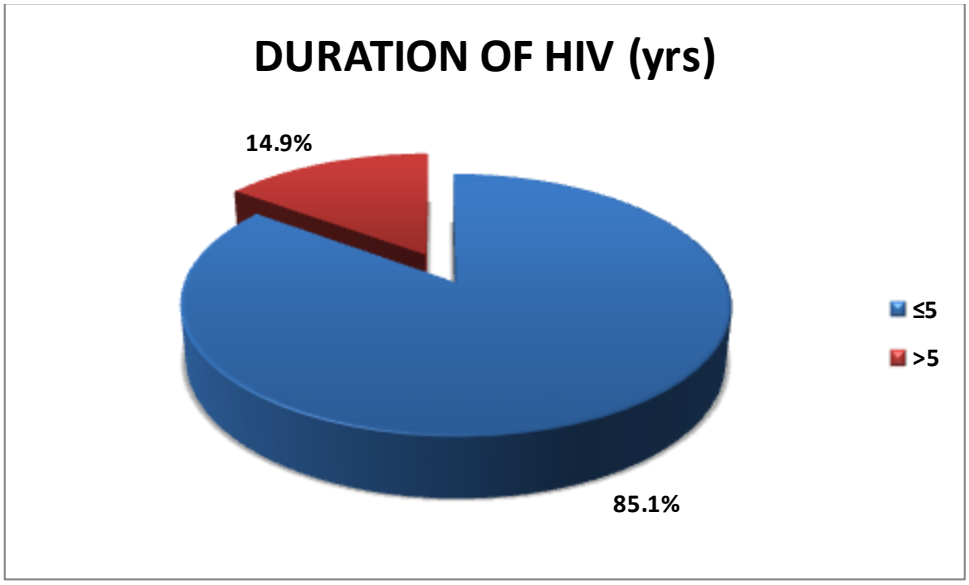
TABLE 7.1: DESCRIPTIVE STATISTICS OF DURATION OF HIV

	Min	Max	Mean	SD
DURATION OF HIV (YRS)	0	20	2.9	3.1

TABLE 7.2: DISTRIBUTION OF CASES ACCCORDING TO DURATION OF HIV

DURATION OF HIV (yrs)	N	%
≤5	126	85.1
>5	22	14.9
Total	148	100

CHART 3: DISTRIBUTION OF CASES ACCCORDING TO DURATION OF HIV

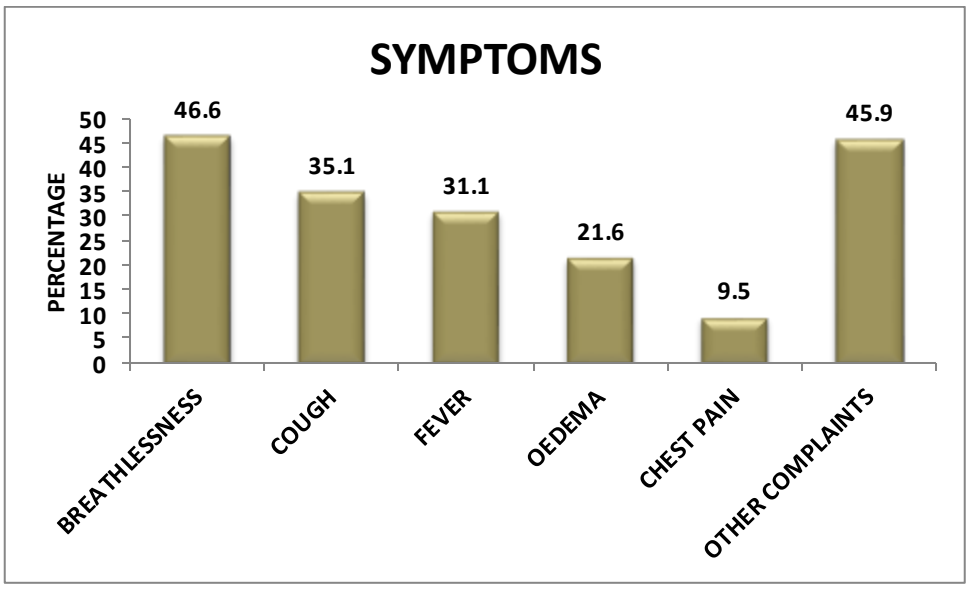


In the present study, we observed the duration of HIV infection varied from 1 month to 20 years. In 126 patients (85.1%) duration was either 5 or less than 5 years. In 22 patients (14.9%) it was more than 5 years .

TABLE 8: DISTRIBUTION OF CASES ACCCORDING TO CLINICAL PRESENTATION

SYMPTOMS	N	%
BREATHLESSNESS	69	46.6
CHEST PAIN	14	9.5
OEDEMA	32	21.6
FEVER	46	31.1
COUGH	52	35.1
OTHER COMPLAINTS	68	45.9

CHART 4: DISTRIBUTION OF CASES ACCCORDING TO SYMPTOMS

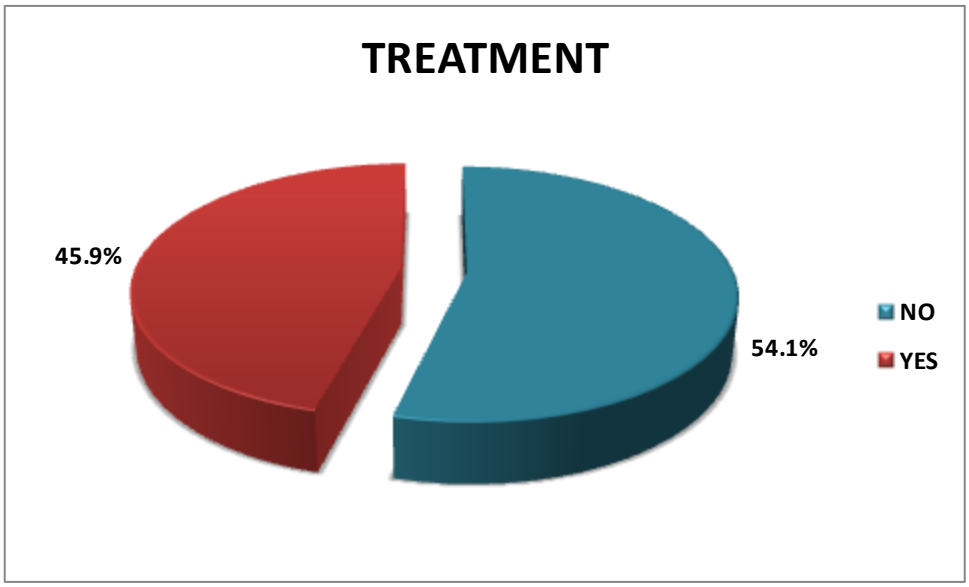


In our study presentation of complaints was as follows overlapping symptoms (like breathlessness, chest pain, edema, fever) were seen in 80 patients (54.1%) of which breathlessness seen in 68(46.6%)patients ,Cough 52(35.1%),fever 46(31.1%),Pedal Edema 32 (21.6%), chest pain 14 patients (9.5%) and complaints other than these in 68 (45.9%)patients.

TABLE 9: DISTRIBUTION OF CASES ACCCORDING TO TREATMENT

TREATMENT	N	%
NO	80	54.1
YES	68	45.9
Total	148	100.0

CHART 5: DISTRIBUTION OF CASES ACCCORDING TO TREATMENT



We observed 68 patients (45.9%) were on the treatment with Anti-Retroviral drugs and the remaining 80 patients (54.1%) were not on ART.

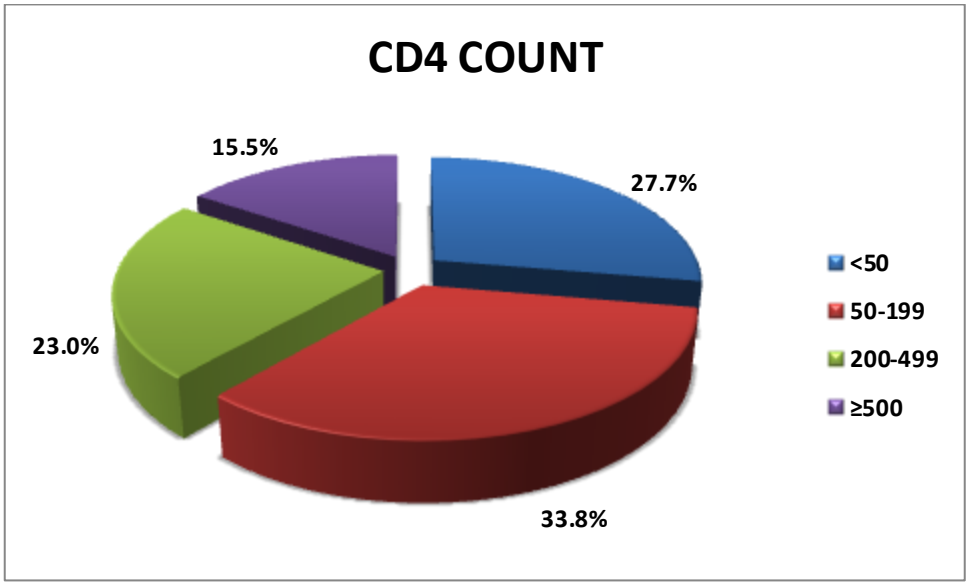
TABLE 10.1 : DESCRIPTIVE STATISTICS OF CD4 COUNT

	Min	Max	Mean	SD
CD4 COUNT	10	1048	230.9	223.1

TABLE 10.2: DISTRIBUTION OF CASES ACCCORDING TO CD4 COUNT

CD4 COUNT	N	%
<50	41	27.7
50-199	50	33.8
200-499	34	23
≥500	23	15.5
Total	148	100

CHART 6 : DISTRIBUTION OF CASES ACCCORDING TO CD4 COUNT

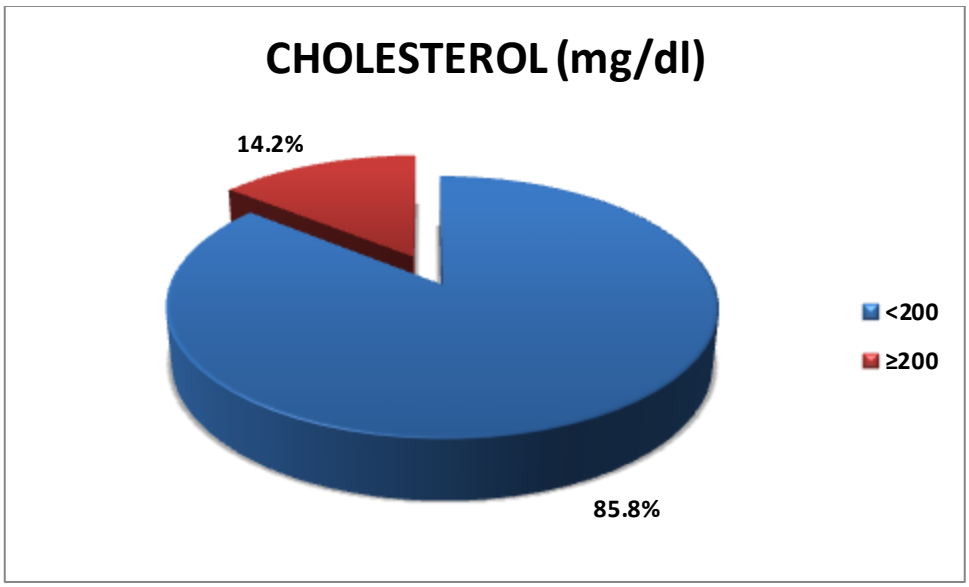


Most of our patients who had presented had CD4 count of more than 50 but less than 200 are 50 patients (33.8%) followed by CD4 counts of less than 50 that is 41 patients (27.7%) , 34 patients (23%) had CD4 count of more than 200 and less than 500. Only 23 patients (15.5%) had count of more than 500.

TABLE 11: DISTRIBUTION OF CASES ACCCORDING TO TOTAL CHOLESTEROL

CHOLESTEROL (mg/dl)	N	%
<200	127	85.8
≥200	21	14.2
Total	148	100

CHART 7: DISTRIBUTION OF CASES ACCCORDING TO TOTAL CHOLESTEROL

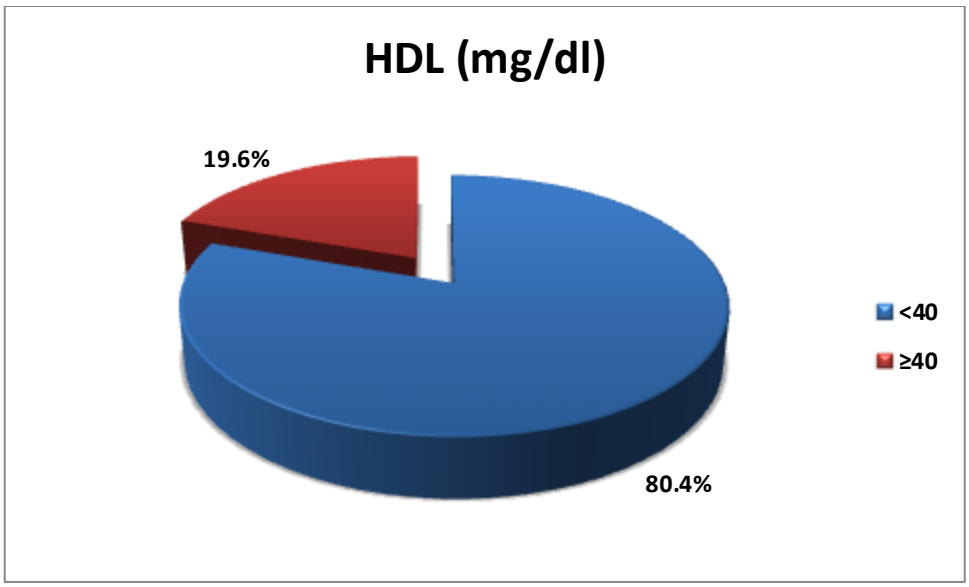


In our study 127 patients (85.8%) had cholesterol of less than 200 mg/dl and in 16 patients (14.2%) was more than 200 mg/dl.

TABLE 12: DISTRIBUTION OF CASES ACCCORDING TO HDL

HDL (mg/dl)	N	%
<40	119	80.4
≥40	29	19.6
Total	148	100

CHART 8 : DISTRIBUTION OF CASES ACCCORDING TO HDL

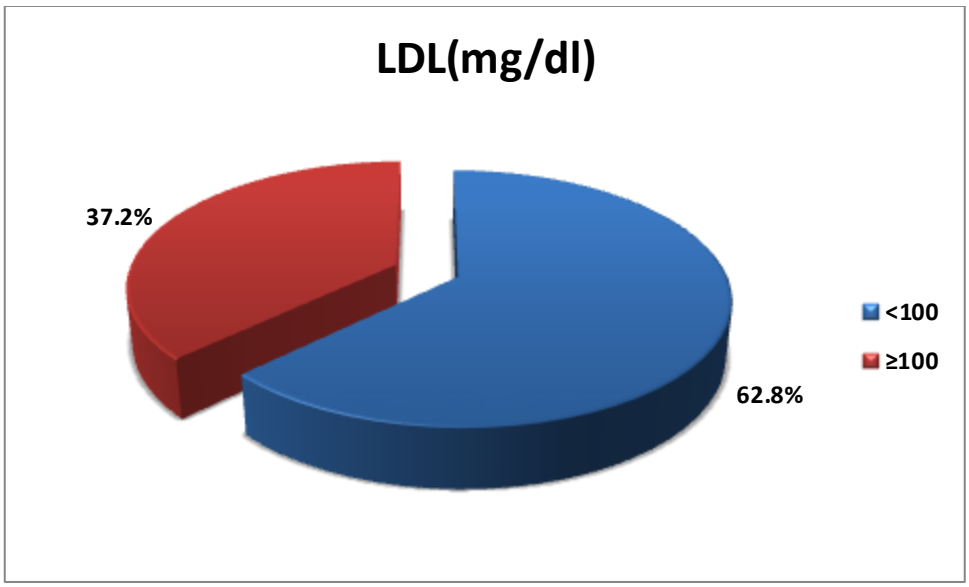


Majority of our patients that is 119 patients (80.4%) had HDL of less than 40mg/dl, 29 patients (19.6%) had levels more than 40 mg/dl.

TABLE 13 : DISTRIBUTION OF CASES ACCCORDING TO LDL

LDL(mg/dl)	N	%
<100	93	62.8
≥100	55	37.2
Total	148	100

CHART 9 : DISTRIBUTION OF CASES ACCCORDING TO LDL

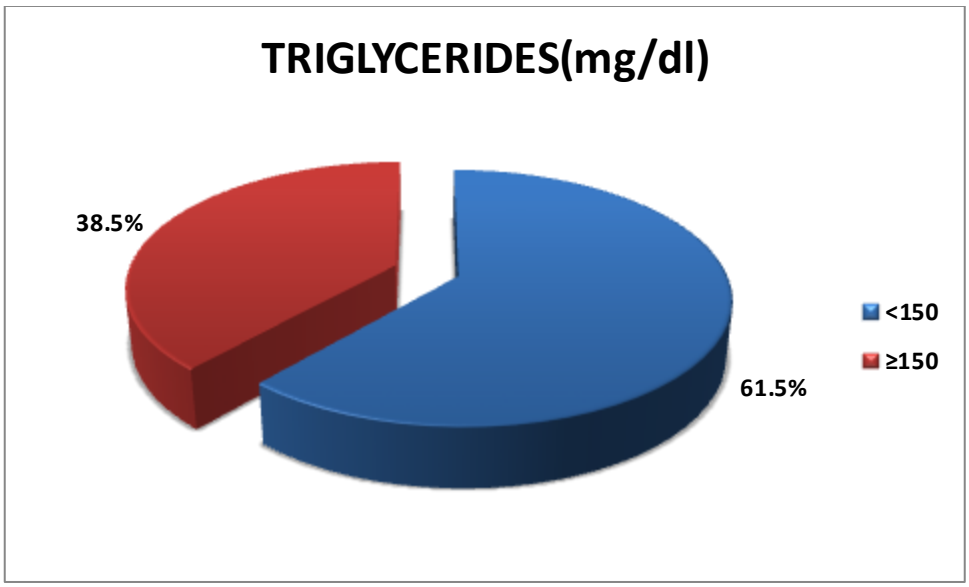


It was observed in 93 patients (62.8%) the LDL was less than 100 mg/dL and in remaining 55 patients (37.2%) it was equal to or more than 100 mg/dl.

TABLE 14: DISTRIBUTION OF CASES ACCCORDING TO TRIGLYCERIDES

TRIGLYCERIDES(mg/dl)	N	%
<150	91	61.5
≥150	57	38.5
Total	148	100

CHART 10 : DISTRIBUTION OF CASES ACCCORDING TO TRIGLYCERIDES

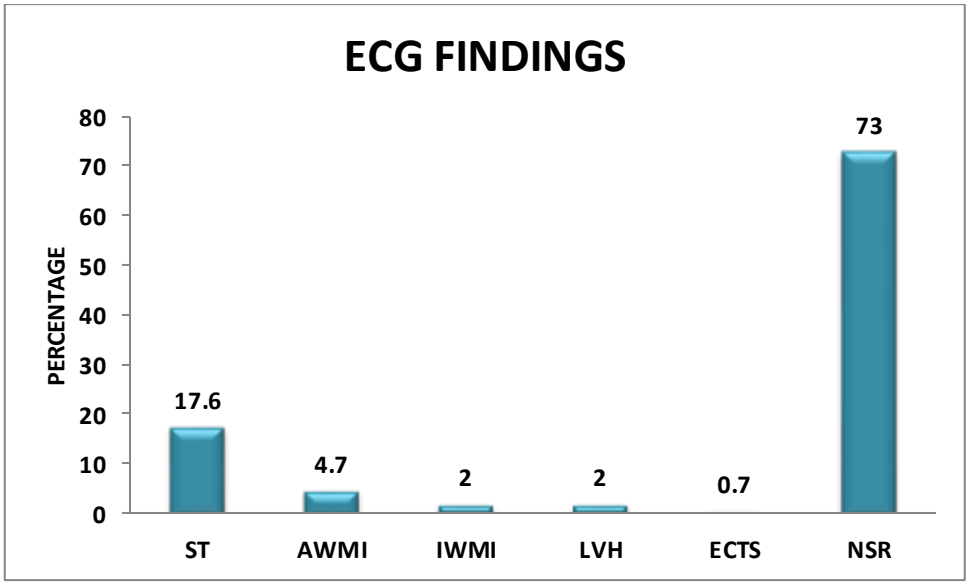


In our study 91 patients (61.5%) had Triglycerides below 150 mg/dl, 57 patients (48.5%) had equal to or more than 150 mg/dl.

TABLE 15 : DISTRIBUTION OF CASES ACCCORDING TO ECG FINDINGS

ECG FINDINGS		N	%
ST		26	17.6
MI	AWMI	7	4.7
	IWMI	3	2
LVH		3	2
ECTS		1	0.7
Normal		108	73

CHART 11 : DISTRIBUTION OF CASES ACCCORDING TO ECG FINDINGS

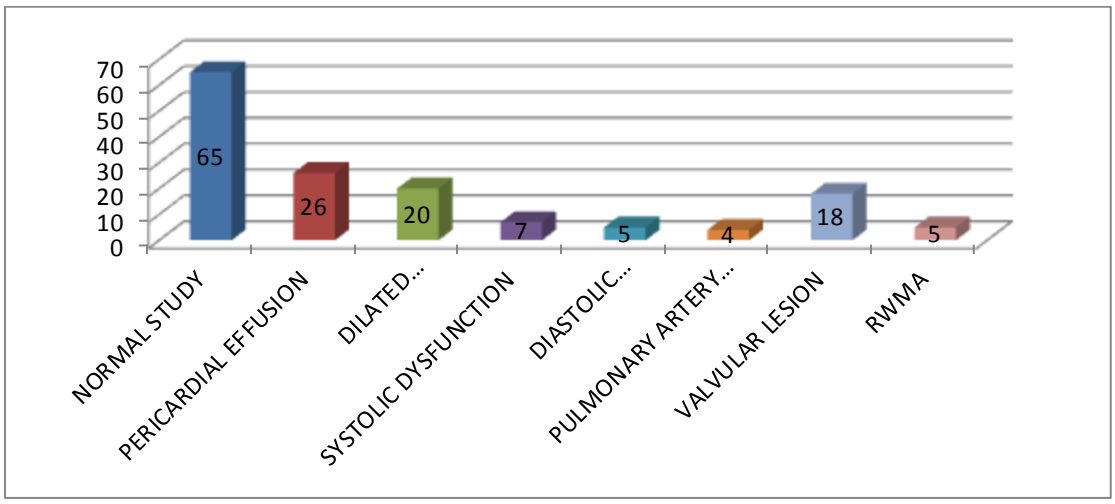


In 108 patients (73 %) ECG study was normal, 26 patients (17.6%) had sinus tachycardia, 10 patients had evidence of Myocardial Infarction (7 patients(4.7%)-Anterior wall MI, 3 patients(2%)- Inferior wall MI), 3 patients (2%%) had left ventricular hypertrophy and 1 patient(0.7%) had ectopics.

TABLE 16: DISTRIBUTION OF CASES ACCCORDING TO ECHOCARDIOGRAPHY

2D ECHO CARDIOGRAPHY	N	%
NORMAL STUDY	65	43.9%
PERICARDIAL EFFUSION	26	17.6%
DILATED CARDIOMYOPATHY	20	13.5%
SYSTOLIC DYSFUNCTION	7	4.7%
DIASTOLIC DYSFUNCTION	5	3.4%
PULMONARY ARTERY HYPERTENSION	4	2.7%
VALVULAR LESION	18	12.2%
RWMA	5	3.4%

CHART 12 : DISTRIBUTION OF CASES ACCCORDING TO ECHO CARDIOGRAPHY

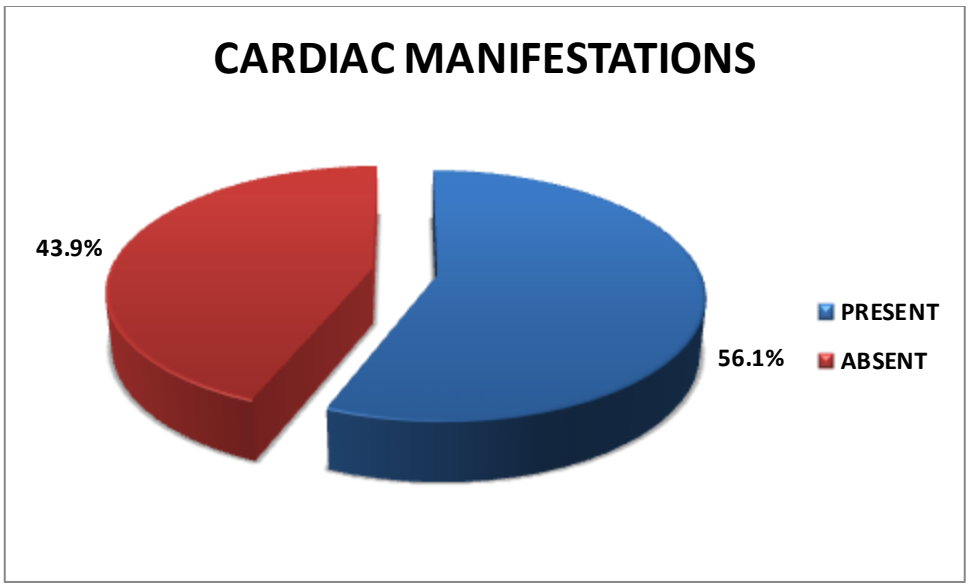


In our study 65 patients(43.9%) had normal study, however 83 patients (56.1%) had various cardiac abnormalities – Dilated cardiomyopathy-20(13.5%), Pericardial effusion- 26(17.6), Regional wall motion abnormality-5(3.4%), Systolic dysfunction 7(4.7%),diastolic dysfunction-5(3.4%), Pulmonary arterial hypertension-4(2.7%), Valvular lesions-18(12.2%).

TABLE 17 : DISTRIBUTION OF CASES ACCCORDING TO CARDIAC MANIFESTATIONS

CARDIAC MANIFESTATIONS	N	%
PRESENT	83	56.1
ABSENT	65	43.9
Total	148	100.0

CHART 13 : DISTRIBUTION OF CASES ACCCORDING TO CARDIAC MANIFESTATIONS



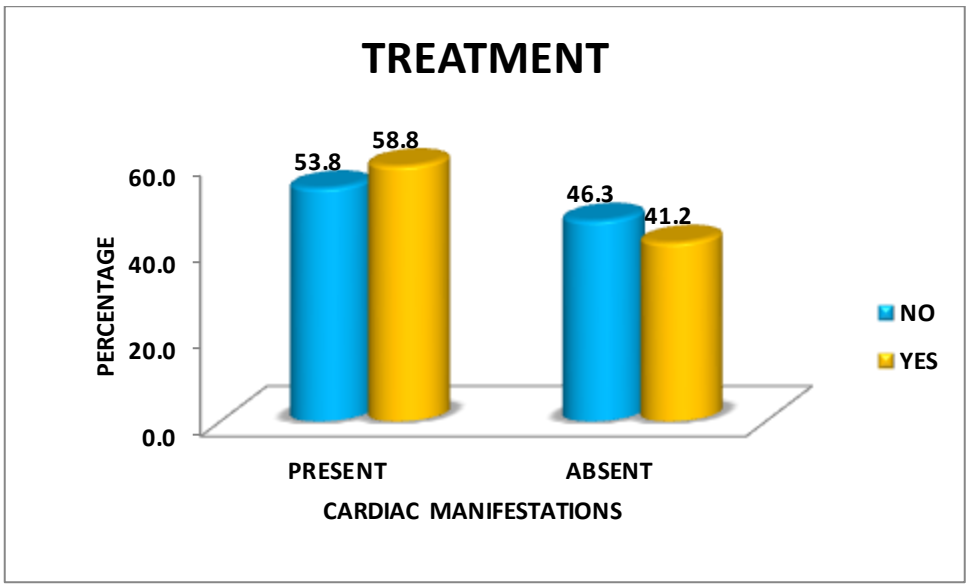
In our study with 2d-echocardiography abnormal cardiac manifestations are seen in 83 (56.1%) patients and were normal in 65(43.9%) patients

TABLE 18: ASSOCIATION OF CARDIAC MANIFESTATIONS AND TREATMENT

TREATMENT	CARDIAC MANIFESTATIONS					p value
	PRESENT		ABSENT		TOTAL	
	N	%	N	%	N	
NO	43	53.8	37	46.3	80	0.34
YES	40	58.8	28	41.2	68	0.039*
Total	83	56.1	65	43.9	148	

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

Chart 14 : ASSOCIATION OF CARDIAC MANIFESTATIONS AND TREATMENT



In our study of 148 patients 80 patients who are not on treatment 43 (53.8%) patients were not having any abnormal findings on echocardiographic study and 37(46.3%) patients have abnormalities with pvalue of 0.34. Among 68 patients who are on regular treatment 40(58.8%) patients have cardiac manifestations with p value 0.039 and 28(41.2%) patients normal echocardiographic study.

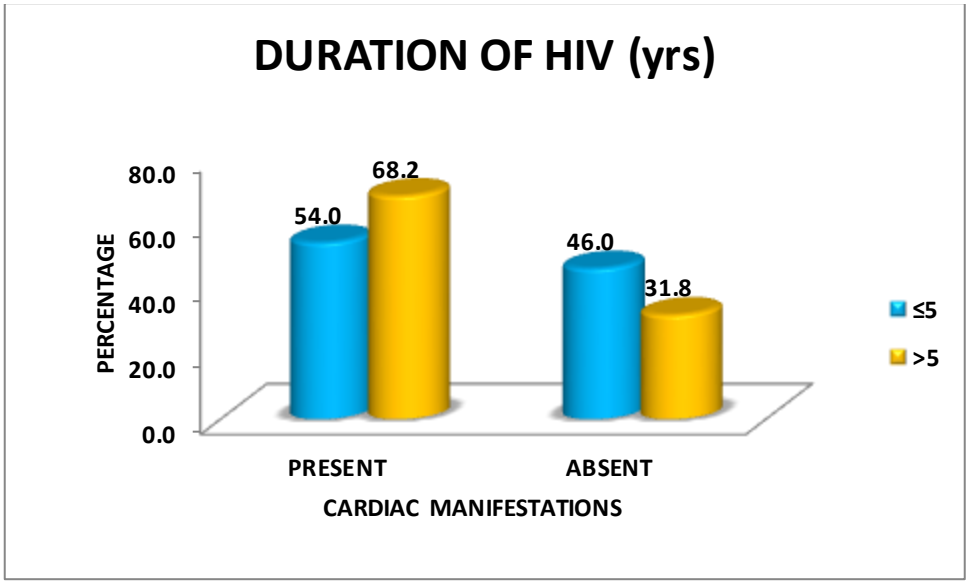
INFERENCE: p-value shows significance among patients on treatment with ART than patients not on ART.

TABLE 19: ASSOCIATION OF CARDIAC MANIFESTATIONS AND DURATION OF HIV

DURATION OF DISEASE (yrs)	CARDIAC MANIFESTATIONS					p value
	PRESENT		ABSENT		TOTAL	
	N	%	N	%	N	
≤5	68	54.0	58	46.0	126	0.21
>5	15	68.2	7	31.8	22	0.015*
Total	83	56.1	65	43.9	148	

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

Chart 15 : ASSOCIATION OF CARDIAC MANIFESTATIONS AND DURATION OF HIV



We observed 126 patients who had infection of 5 years or less, 68 had cardiac abnormalities, 58 patients did not have abnormal findings on 2d echo study, p value was 0.21. Whereas 22 patients who had infection of >5 years, 15 patients showed cardiac abnormalities, 7 patients have normal 2d echo findings, p value was 0.015.

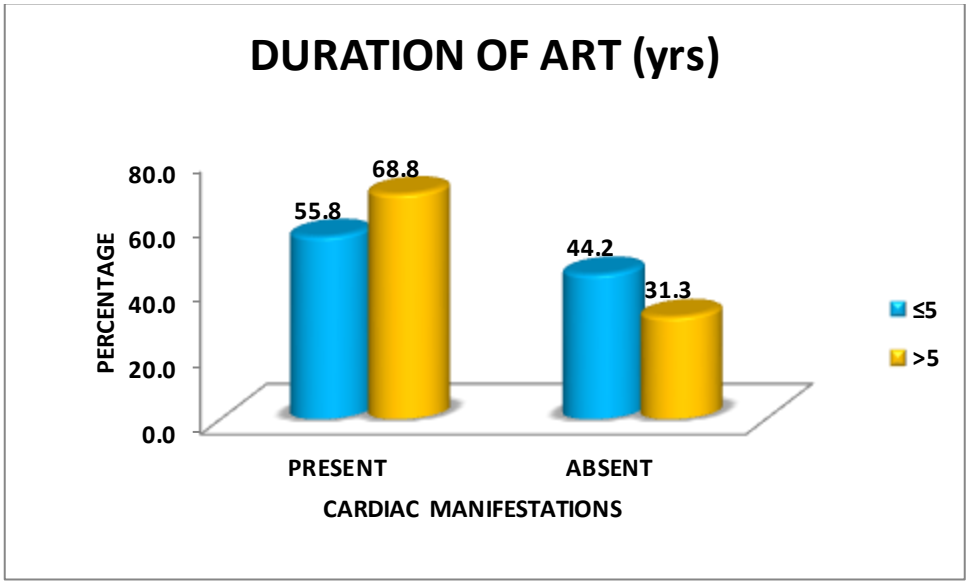
INFERENCE: p-value was significant among patients with HIV duration greater than 5 years.

TABLE 20: ASSOCIATION OF CARDIAC MANIFESTATIONS AND DURATION OF TREATMENT WITH ART

DURATION OF DISEASE (yrs)	CARDIAC MANIFESTATIONS					p value
	PRESENT		ABSENT		TOTAL	
	N	%	N	%	N	
≤5	29	55.8	23	44.2	52	0.24
>5	11	68.8	5	31.3	16	0.03*
Total	40	58.8	28	41.2	68	

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

CHART 16: ASSOCIATION OF CARDIAC MANIFESTATIONS AND DURATION OF TREATMENT WITH ART



Among 68 patients who are on ART,52 patients were on ART equal to or less than 5 years of which 29 (55.8%) had cardiac manifestations and 23 (44.2%) patients did not have cardiac manifestations, p value 0.024.

Among 16 patients who are on ART greater than 5 years, 11(68.8%) had cardiac manifestations and 5(31.3%) did not have any abnormalities on echo cardiographic study, p value 0.05.

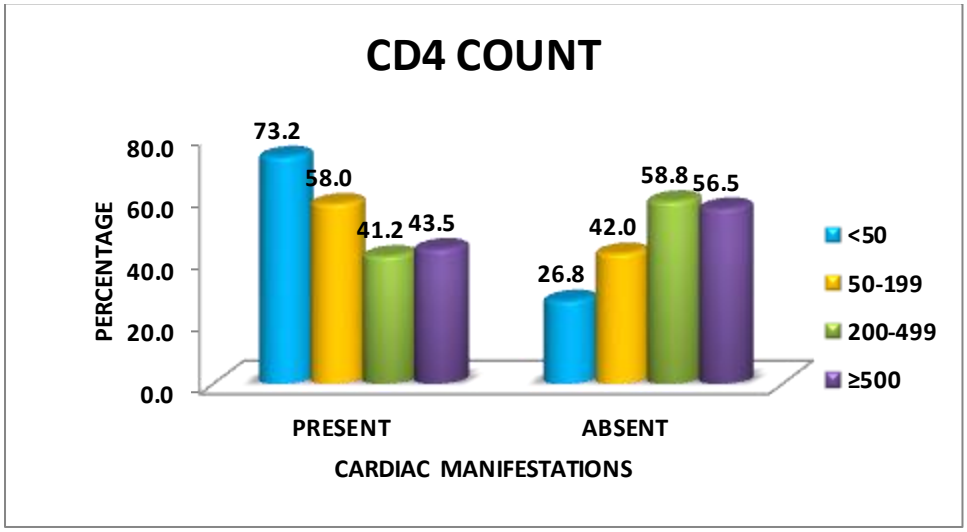
INFERENCE: p-value significant among patients on ART more than 5years.

TABLE 21 : ASSOCIATION OF CARDIAC MANIFESTATIONS AND CD4 COUNT

CD4 COUNT	CARDIAC MANIFESTATIONS					p value
	PRESENT		ABSENT		TOTAL	
	N	%	N	%	N	
<50	30	73.2	11	26.8	41	<0.001*
50-199	29	58.0	21	42.0	50	0.11
200-499	14	41.2	20	58.8	34	0.15
≥500	10	43.5	13	56.5	23	0.38
Total	83	56.1	65	43.9	148	

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

CHART 17 : ASSOCIATION OF CARDIAC MANIFESTATIONS AND CD4 COUNT



In this study when Cardiac manifestations were compared with CD4 counts.In patients with CD4 counts less than 50 ,abnormalities were found in 30 patients(p value 0.001) and in 11 patients no Echocardiographic abnormalities were seen. In patients with CD4 Counts between 50-199, 29 had manifestations and 21 did not and the p value was 0.11.

I n patients with Counts between 200-499, 14 had cardiac manifestations and 20 did not and p value was 0.15.In patients with CD4 counts $\geq$ 500 , 10 had cardiac manifestations and 13 did not, p value 0.38.

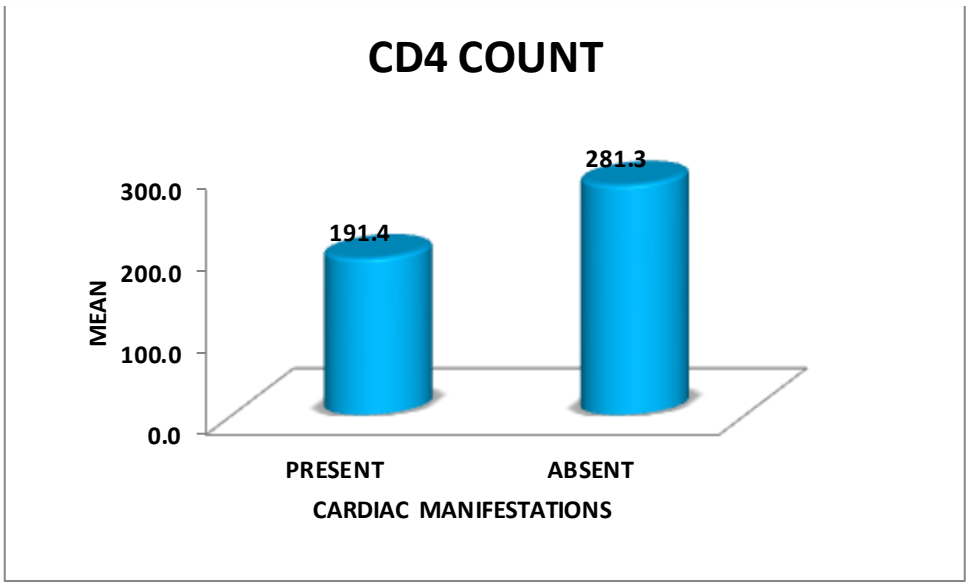
INFERENCE: p-value significant among patients with CD4 counts <50

TABLE 22: ASSOCIATION OF CARDIAC MANIFESTATIONS AND MEAN CD4 COUNT

CARDIAC MANIFESTATIONS	PRESENT		ABSENT		p value
CD4 COUNT	Mean	SD	Mean	SD	
	191.4	206.2	281.3	235.1	0.015*

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

CHART 18: ASSOCIATION OF CARDIAC MANIFESTATIONS AND MEAN CD4 COUNT



In an attempt to compare mean CD4 count with cardiac manifestations it showed cardiac abnormalities present in 191.4± 206.2 and were absent in 281.3± 235.1. Statistically p value was significant (p=0.015).

DISCUSSION

In the present study of 148 patients with HIV infection, different cardiac manifestations were observed and same was compared with various factors.

AGE:

In our study patients age ranged from 18 to 60 years, maximum number of cases were in the age group of 31 to 40 that is 57 patients (38.5%), between 41-50 years 48 patients (32.4%), between 51-60 years 23 patients (15.5%), below 30 years 20 patients (13.5%) . HIV infection was found more common in younger age group, same was observed by NACO annual report 2016- 2017.[21] Similar observations were made by Das S et al[56] and Currie et al.[37]

SEX:

Taking sex into consideration we observed in our study males were more affected (108 patients-73%) as compared to females (40 patients -27%).Same sex difference was observed by NACO annual report (2016-2017).[21] Same conclusion was drawn by Das et al[56]and Aggarwal et al.[57]

DURATION OF HIV INFECTION:

The duration of HIV infection varied from newly diagnosed to 20 years. Maximum number of patients were 126(85.1%) with a duration, either five years or less than five years. Only 22 patients (14.9%) had duration more than 5 years. I n our study with sample of 148 correlation(p 0.015) was seen with duration of HIV (>5years) and cardiac manifestations. Similarly in a study by Lyons A et al [58] , they have found that rate of heart attack increased with duration of infection, from 0.43 per 1000 person-years in people infected for five years or less, to 0.86 per 1000 person-

years in those infected for five to ten years, 1.06 per 1000 person-years in those infected for ten to fifteen years and 2.65 per 1000 person years of follow-up in those infected for more than 15 years.

TREATMENT:

In our study most of patients were not on treatment with ART. Only 68 patients (28%) were on ART. Similar observation was made by NACO annual report (2015-2016)[21] 2.1 million people living with HIV/AIDS with 0.3% adult prevalence of which only 50% adults are on ART.

CLINICAL PRESENTATION:

The clinical presentations in our patients, majority i.e. 69 patients (46.6%) had breathlessness ,chest pain 14 patients (9.5%), fever 46 patients (31.1%) pedal edema 32 patients (21.6%) and cough 52 patients (35.1%) and complaints other than these in 68 patients(45.9%) . This is in sharp contrast to studies done by Das et al[56] and Anita et al [59]who found symptoms pertaining to pulmonary infections.

CD4 COUNTS:

When attempt was made to compare our study with various lab parameters,we observed following factors- In majority of our patients CD4 count was between 50-199 i.e.50 patients (33%), CD4 count was less than 50 in 41 patients (27.7%), 34 patients (23%) had CD4 count of more than 200 and less than 500, and in only 23 patients (15.5%) count was more than 500. Similar observation was made by

Das et al.[57] The low CD4 count in our study was probably due to advanced disease state as all our patients were admitted cases in hospital.

FASTING LIPID PROFILE:

The fasting lipid studies – Total cholesterol, 127 patients (85.8%) had cholesterol of less than 200 mg/dl and in only 21 patients (14.2%) it was more than 200 mg/dl. HDL was less than 40 mg/dl in 119 patients (80.4%),equal or more than 40 mg/dl in 29 patients (19.6%). In 93 patients (62.8%) the LDL was less than 100 mg/dl and in remaining 55 patients (37.2%) it was equal to or more than 100 mg/dl. 91 patients (61.5%) had Triglycerides below 150 mg/dl and 57 patients (38.5%) had equal to or more than 150 mg/dl. When compared to studies by Baker et al,[60] El- Sadr et al[61] they found low CD4 count associated with lower LDL-C, HDL-C. Another study by Riddler et al [62] in patients of HIV seroconversion was associated with decreased triglycerides, LDLC, HDL-C levels. In our study majority i.e. 85.8 % had total cholesterol less than 200 this difference is little difficult to explain owing to our small sample size.

ECG :

ECG findings revealed normal tracing in 108 patients (73%), 26 patients (17.6%) had sinus tachycardia, 10 patients had evidence of Myocardial Infarction (7patients-Anterior wall MI(4.7%), 3 patients- Inferior wall MI (2%)), 3 patients (2%) had left ventricular hypertrophy and 1 patient had ventricular ectopics.A study by Anita B et al[59] in their patients sinus tachycardia was observed in 72%; Study by Herdy GV et al[63] had normal tracing in 33% and ST-T changes in 37%: Study by Hadadi et al[64] observed 9.7 % ST-T changes.

ECHOCARDIOGRAPHY:

Echocardiographic evaluation of these 148 patients, normal study was observed in 65 patients (43.9%.) In remaining 83 patients (56.1%) various cardiac abnormalities were observed. pericardial effusion- 26(17.6%),Dilated cardiomyopathy-20(13.5%),Regional wall motion abnormality-5(3.4%), Systolic dysfunction in 7 patients(4.7%) and diastolic dysfunction in 5 patients (3.4%), Pulmonary artery hypertension in 4 patients(2.7%), Valvular lesions in 18 patients(12.2%), .Various studies done by Das et al,[56] Aggarwal et al[57] (India) ,Hakim et al[65] in Zimbabwe and Reinsch N et.al [66] in U.S , similar cardiac abnormalities were observed with varying percentage related to cardiac abnormalities.

DURATION OF TREATMENT:

We observed taking into consideration duration of treatment with ART and cardiac abnormalities in them , of 68 patients on ART we found 29 patients among treatment duration equal to or less than 5years and 11 patients on treatment with ART for more than 5 years had cardiac abnormalities. It was found that p-value was 0.03 in patients on treatment greater than 5 years. Similar observations were made by Muriella M.K et al [67] Nirdesh et.al [68] in their observational studies.

When comparison of cardiac manifestations with CD4 count was attempted. In patients with count less than 50/cmm abnormalities were found in 30 patients(p-value <0.001,statistically significant) and in 11 patients no abnormalities. In patients with Counts between 50-199, 29 patients had manifestations and 21 patients did not have. Counts between 200-499, 14 patients had manifestations and 20 did not have. And in patients >500 counts, 10 patients had manifestations and 13 patients did not have.

When mean CD4 count was considered there was a positive correlation between mean CD4 count and cardiac manifestation. ($p=0.015$; statistically significant) Similar observation was made by Aggarwal et al.[57] The comparison of CD4 count by various authors has shown cardiac abnormalities increase with the level of immunosuppression and low CD4 count which is also observed in our study.

It has become easier in the recent years to find various cardiac abnormalities in patients with HIV infection because of availability of sophisticated investigations routinely in most of hospitals/centres.

The various cardiac abnormalities observed in our small sample size of 148 patients is little difficult to explain, it could be direct HIV infection, may be opportunistic infection, progressive disease state or may be ART drug toxicity, needs to be addressed by detailed study and large sample size.

CONCLUSION:

In present study of 148 patients with HIV infection the predominant age group was 31-40 years .

When sex is taken into account, it was seen that males were affected more than females .

In this study, majority of patients presented with one or more of the following typical complaints like breathlessness, chest pain, fever, pedal edema and cough .

In our study, found to have different cardiac manifestations to the tune of 56.1% because of availability of 2D echocardiography while with ECG only 27 % of patients were found to have cardiac abnormality.

The cardiac manifestations observed were pericardial effusion, dilated cardiomyopathy, systolic and diastolic dysfunctions, pulmonary artery hypertension and valvular lesions , among which pericardial effusion was predominantly found followed by dilated cardiomyopathy

When the cardiac manifestations were compared with CD4 counts, majority of patients belong to CD4 counts between 50-199 and significant correlation was seen with CD4 count less than 50 and mean CD4 among patients with cardiac manifestations is 191.4.

When cardiac manifestations compared with duration of HIV and treatment duration significant correlation was found with duration of HIV and treatment duration greater than 5years.

Whereas in this study we were not able to found any correlation of cardiac manifestation with lipid profile.

It is necessary to evaluate further these patients to find out exact reasons for various cardiac abnormalities.

We feel it is worth to study by adjusting the co-morbid conditions/ confounding factors and comparing the cardiac abnormalities by detailed studies with cardiac specific biochemical markers, coronary angiography and histopathological studies which may reflect whether these abnormalities per se are because of HIV infection or may be affected by factors like age, sex, duration of HIV or treatment with ART.

Owing to our small sample size (148 patients) large sample size, may be required to overcome these bias.

SUMMARY

The present study of 148 patients with HIV infection admitted in Department of Medicine, in BLDE's Shri B.M.PATIL HOSPITAL, Vijayapura during the study period of September 2015 to March 2018, to find out the various cardiac manifestations and its correlation with CD4 count.

The results observed were significant correlation of cardiac abnormalities with low CD4 count and mean CD4 count and also with longer duration of HIV infection and ART treatment.

However we did not find significant correlation with variable factors – age, sex.

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ANNEXURE I

ETHICAL CLERANCE CERTIFICATE

ANNEXURE II

INFORMED CONSENT FORM

TITLE OF RESEARCH	**:**	**"A STUDY OF CARDIAC MANIFESTATIONS IN HIV PATIENTS"**
GUIDE	**:**	**DR R.M. HONNUTAGI**
P.G.STUDENT	**:**	**DR SANDEEP REDDY KALLAM**

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to access the cardiac manifestations in HIV patients and their correlation with CD4 count.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved in this study and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help to study the cardiac manifestations in HIV positive individuals in this part of state.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further medical compensation.

_____ _____

(Signature of Guardian) (Signature of patient)

STUDY SUBJECT CONSENT FORM:

I confirm that Dr.Sandeep Reddy Kallam has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

SIGNATURE OF PARTICIPANT DATE

SIGNATURE OF WITNESS DATE

ANNEXURE III

BLDEU'S SHRI B.M.PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR

"THE STUDY OF CARDIAC MANIFESTATIONS IN HIV PATIENTS"

PROFORMA

Name of the patient:

Age in years:

Sex:

Address:

Religion:

Occupation:

IP no/OP no:

Presenting Complaints:

Past history:

Personal history:

1. Tobacco chewing

2. Smoking

3. Alcoholism

4. Diet-

Family history:

GENERAL PHYSICAL EXAMINATION :

Built :

Nourishment :

Ht(Cm) :

Wt(Kg) :

BMI:

Pallor

Icterus

Clubbing

Cyanosis

Edema

6. Vital parameters a. Pulse :

b. BP :

c. Respiratory rate :

d. Temperature

SYSTEMIC EXAMINATION:

ABDOMEN EXAMINATION

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

CENTRAL NERVOUS SYSTEM

BIOCHEMISTRY

1)LIVER FUNCTION TESTS	
Total bilirubin	
Conjugated	
Unconjugated	
SGOT SGPT	
Total protein Albumin Albumin: Globulin	
2)Random blood sugar	
3)LIPID PROFILE	
Total Cholesterol	
Triglycerides	
HDL-Cholesterol	
LDL-Cholesterol	
VLDL-Cholesterol	
4) Serum creatinine	
5) Blood urea	

PATHOLOGY 1)Urine routine	
Albumin	
Sugar	
Urine microscopy RBC's Pus cells Cast's Epithelial cells	
2)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count Neutrophils	%
Lymphocytes	%

Eosinophils	%
Basophils	%
Monocytes	%
ESR	At end of 1st hour.
CD4 COUNT	

2D-ECHO

LVIVSd : cm	LVIDd : cm	Aorta : cm
LVPWd : cm	LVISd : cm	LA (AP): cm
RVIDd : cm	EF% : %	PA : cm

VALVES :

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

CHAMBERS :

Left Ventricle :

Right Ventricle :

Left Atrium :

Right Atrium :

SEPTAE :

GREAT ARTERIES

Aorta :

Pulmonary Artery :

DOPPLER STUDY

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

REGIONAL WALL MOTION ABNORMALITIES :

PERICARDIAL EFFUSION :

CLOT/VEGETATION :

CONCLUSION :

FUNDOSCOPY:

ECG:

CONCLUSION:

DATE: **SIGNATURE**

S.NO	NAME OF PATIENT	IP/OP NUMBER	AGE	SEX	OCCUPATION	DURATION OF DISEASE	TREATMENT	PRESENTATION					CD4 COUNT	INVESTIGATIONS										2D ECHO CARDIOGRAPHY																				
														HAEMOGRAM							LIPIDS									HYPOKINESIA/AKINESIA								VALVULAR LESION								
																													AKINESIA																
								BREATHLESSNESS	CHEST PAIN	OEDEMA	FEVER	COUGH		HAEMOGLOBIN (gm%)	TOTAL COUNT (/cum)	NEUTROPHILS	LYMPHOCYTES	MONOCYTES	EOSIONOPHILS	BASOPHILS	CHOLESTEROL (MG/DL)	HDL (mg/dl)	LDL(mg/dl)	TRIGLYCERIDES(mg/dl)	ECG FINDINGS	PERICARDIAL EFFUSION	DILATED CARDIOMYOPATHY	DYSFUNCTION SYSTOLIC	DYSFUNCTION DIASTOLIC	PULMONARY ARTERY HYPERTENSION	GLOBAL	ANTERIOR WALL	APICAL SEPTUM	APEX	RWMA	INFERIOR WALL	LEFT VENTRICLE	LATERAL WALL	MR	TR	AR	MS	CALCIFICATION	CLOT	VEGITATION	EJECTION FRACTION
1	JAYASHREE	9333/16	55	F	HOMEMAKER	2y	-	-	-	-	+	-	416	8.2	4300	48	40	4	8	0	142	42	78	148	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
2	BANEPPA	7220/16	40	M	CLERK	3y	-	+	-	-	-	+	42	13.2	6300	75	23	2	0	0	98	20	60	88	NSR	+	-	-	-	ML	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
3	HANAMANT	9885/16	35	M	COBBLER	1y	-	-	+	-	-	-	29	9.4	5500	86	8	6	0	0	192	35	125	276	IW MI	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	60%
4	SAIBANNA	10001/16	28	M	BARBER	2y	-	-	-	-	+	-	35	9.3	8900	85	15	0	0	0	92	14	46	73	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
5	JAKAVVA	9489/16	48	F	HOMEMAKER	2y	-	+	-	-	-	-	86	11	9900	76	16	8	0	0	212	47	131	168	ST	-	-	+	-	ML	-	-	-	-	-	-	-	-	-	-	-	-	-	-	35%
6	ANNAPURNA	10729/16	25	F	HOMEMAKER	1y	-	-	-	-	-	+	537	11.4	4900	70	20	8	2	0	123	26	90	119	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
7	RENUKA	11307/16	35	F	HOMEMAKER	4y	-	-	+	+	-	-	326	10.6	3000	56	33	10	1	0	132	26	93	179	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
8	DURGAPPA	11180/16	45	M	DRIVER	3y	-	+	-	+	-	+	47	11.4	8600	59	28	7	6	0	251	28	181	239	NSR	-	+	+	-	MD	+	-	-	-	-	-	-	-	2	2	-	-	-	-	-	25%
9	SUKHDEV	11014/16	40	M	FARMER	4y	-	-	+	-	-	-	147	10.2	7900	82	10	4	4	0	112	28	63	119	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
10	BOURAMMA	14106/16	33	F	FARMER	3y	-	+	-	+	+	-	672	12.6	8400	76	16	6	2	0	192	45	87	138	NSR	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	50%
11	MAHADEVI	2228/16	30	F	HOMEMAKER	4y	+	-	-	+	-	-	46	10.9	4030	60	38	1	1	0	98	30	56	134	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
12	KALYANSINGH	12080/16	70	M	SHOPKEEPER	9y	-	+	-	-	-	-	177	9.4	13110	80	16	2	2	0	124	21	90	147	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
13	BABU	6928/16	43	M	SHOPKEEPER	4y	+	-	+	-	-	-	48	7.8	21200	90	7	2	1	0	131	48	90	156	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
14	SHIVAJI	5254/16	45	M	DRIVER	2m	-	-	-	-	+	-	43	17.7	5300	56	36	5	3	0	145	35	112	169	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
15	SHIVAPPA	12654/16	45	M	FARMER	8m	-	+	-	-	-	-	34	9.7	7200	58	36	4	2	0	161	30	134	203	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
16	MAHAVEER	11474/16	32	M	LABOURER	10m	-	-	-	+	-	-	325	13.4	8300	48	40	4	8	0	86	10	44	73	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
17	INDRABAI	4664/16	45	F	FARMER	4y	-	+	-	-	+	+	40	10.8	3500	60	19	7	14	0	80	12	48	103	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
18	TIPANNA	13721/16	39	M	DRIVER	1y	-	-	+	-	-	-	105	12.6	4950	70	23	5	2	0	112	36	90	143	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
19	SIDDANNA	13914/16	48	M	FARMER	2y	-	+	-	+	+	+	148	4.8	6600	87	6	7	0	0	74	19	35	98	NSR	-	+	+	-	MD	+	-	-	-	-	-	-	-	3	2	-	-	-	-	-	30%
20	PANDIT	21924/16	30	M	DRIVER	3y	-	+	-	-	-	-	155	9.6	4900	80	10	8	2	0	94	14	58	112	LVH	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	AML	-	-	60%
21	AHAMED	14551/16	35	M	DRIVER	1y	-	-	-	-	+	-	29	10.4	2100	88	10	0	2	0	142	43	87	96	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
22	BHAGANNA	21460/16	55	M	FARMER	4y	+	+	-	-	-	+	45	9	4010	55	40	3	2	0	208	36	115	259	NSR	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	40%
23	KHEMU	14815/16	52	M	COOLIE	1y	+	-	-	-	+	+	161	6.4	11300	47	43	8	2	0	89	27	58	86	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
24	B KRISHNA	15011/16	50	M	DRIVER	8y	-	+	-	-	-	-	47	12.8	29000	64	33	2	1	0	122	14	37	354	NSR	-	+	+	-	ML	+	-	-	-	-	-	-	-	1	1	-	-	-	-	-	40%
25	RAJSHEKHAR	16271/16	45	M	BARBER	4y	+	-	+	-	-	-	44	11.7	3900	57	28	10	5	0	246	20	189	185	MI	-	-	-	-	-	-	-	+	+	-	-	-	+	-	-	-	-	-	-	-	55%
26	RUKMINI	32404/16	31	F	HOMEMAKER	3m	-	+	-	-	+	-	167	12.4	13500	55	39	4	2	0	289	29	156	356	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
27	SHEKAPPA	18597/16	35	M	DRIVER	6m	-	-	-	+	-	-	382	14.5	3300	38	60	0	2	0	132	37	96	140	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
28	SHRISHAIL	19392/16	60	M	FARMER	9m	-	-	+	-	-	-	82	14.9	9100	68	30	0	2	0	278	27	139	306	MI	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
29	MAHESH PATIL	19397/16	42	M	LABOURER	3m	-	+	-	+	-	+	42	6.5	10500	65	28	7	0	0	175	35	129	148	ST	-	-	+	-	MD	-	-	-	-	-	-	-	-	-	2	-	-	-	-	45%
30	SHRISHAIL	19930/16	47	M	DRIVER	1y	+	-	-	+	-	-	667	9.6	3800	54	44	1	1	0	156	23	129	206	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
31	SHANMUKH	19737/16	32	M	FARMER	1y	-	+	-	+	-	+	42	9.4	9800	85	8	7	0	0	152	21	103	143	NSR	+	-	+	-	ML	+	-	-	-	-	-	-	-	1	1	-	-	-	-	-	30%
32	ANASUYA	19409/16	38	F	FARMER	5y	+	+	-	-	+	-	397	9.2	10300	76	14	4	4	2	120	46	90	136	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
33	SHANTABAI	22276/16	38	F	FARMER	5y	+	-	-	-	+	+	212	11.7	5500	79	14	5	2	0	103	23	71	109	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
34	MUDAKAPPA	21426/16	45	M	FARMER	1y	-	+	-	+	-	+	40	11.2	1900	60	25	8	7	0	136	19	84	167	NSR	+	-	+	-	MD	-	-	-	-	-	-	-	-	-	-	-	-	-	-	45%
35	RAMESH	18513/16	25	M	STUDENT	1y	-	+	-	+	+	+	50	13	7300	86	9	5	0	0	237	20	136	289	NSR	-	+	+	-	ML	+	-	-	-	-	-	-	-	2	2	-	-	-	-	-	35%

36	IRAMMA	20167/16	60	F	HOMEMAKER	1y	-	+	-	-	-	-	168	9.6	3800	54	44	1	1	0	156	23	129	206	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
37	DODDAWWA	24680/16	32	F	DRIVER	1m	-	+	-	+	-	+	433	10.7	5200	60	30	8	2	0	83	18	79	64	NSR	+	-	-	+	ML	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
38	SHOBHA	1576/16	35	F	HOMEMAKER	3m	-	-	-	+	-	-	969	10	5630	71	27	0	2	0	134	41	87	97	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
39	SANGAPPA	23376/16	35	M	SHOPKEEPER	4m	-	+	-	-	-	+	45	8.9	4860	78	20	0	2	0	208	36	115	259	NSR	+	-	+	-	MD	+	-	-	-	-	-	-	2	2	-	-	-	-	-	-	30%
40	PARASHURAM	23469/16	32	M	SHOPKEEPER	4m	-	-	-	-	+	-	567	12.4	3800	66	30	0	4	0	107	18	73	114	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
41	BASAVARAJ	26881/16	38	M	FARMER	5m	-	-	-	-	+	+	442	11.4	8600	59	28	7	6	0	251	28	181	239	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
42	NINGAPPA	27259/16	30	M	LABOURER	6m	+	+	-	+	-	-	40	10.1	3400	70	24	6	0	0	230	36	131	257	NSR	-	+	+	-	ML	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	40%
43	RENUKA	13685/17	18	F	STUDENT	6m	-	+	-	+	-	-	42	8.7	4900	87	9	1	3	0	149	31	120	138	NSR	-	+	+	-	MD	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-	35%
44	BASANNA	31349/16	52	M	FARMER	2y	-	+	-	-	+	+	141	12.2	9700	56	33	8	3	0	116	31	92	148	NSR	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	MA	-	-	-	35%
45	SIDDALINGAPPA	14484/17	56	M	FARMER	2y	-	+	-	-	+	-	548	10	9000	84	6	10	0	0	182	29	164	206	NSR	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	40%
46	DAYANAND	14640/17	35	M	DRIVER	1y	+	+	-	+	+	+	145	5.4	19900	52	30	0	5	0	90	43	102	127	NSR	+	-	+	-	ML	+	-	-	-	-	-	-	2	1	-	-	-	-	-	-	35%
47	CHANNABASU	4258/16	38	M	COOLIE	4y	+	+	+	-	-	-	601	7.9	14700	85	7	8	0	0	156	40	89	89	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
48	BHIMARAYA	2374/16	35	M	FARMER	2m	-	+	-	+	-	+	209	14.3	7900	90	4	6	0	0	168	35	68	126	ST	-	-	-	-	SV	-	-	-	-	-	-	-	3	-	-	-	-	-	-	-	60%
49	SHRIMANTH	12044/16	45	M	DRIVER	7m	+	+	-	+	-	-	43	9	3900	60	36	2	2	0	218	29	141	256	NSR	-	+	+	-	MD	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	35%
50	MADIVALAPPA	8010/16	45	M	DRIVER	3Y	-	-	-	-	+	+	456	9.3	3400	58	38	2	2	0	167	25	132	238	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	65%
51	KISHAN	23711/15	45	M	LABOURER	1m	-	+	-	-	+	+	63	7.6	8200	78	20	0	2	0	93	40	68	86	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	58%
52	MALLIKARJUN	10364/16	35	M	FARMER	1y	-	+	-	-	+	-	43	11.4	7900	88	10	1	1	0	154	39	86	189	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
53	BASAVARAJ	7958/16	35	M	FARMER	2y	-	+	-	-	+	+	86	9.9	3900	69	21	10	0	0	145	35	112	169	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
54	RAYAGOND	12580/16	42	M	LABOURER	1y	-	-	-	-	+	+	256	10	12800	83	10	6	1	0	88	32	78	98	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
55	SHIVAPPA	12654/16	45	M	FARMER	2y	-	-	-	-	+	+	456	14.2	6400	76	14	8	2	0	167	25	132	238	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
56	SHARANAGOUDA	13484/17	35	M	SHOPKEEPER	1y	-	+	-	+	-	-	26	12.9	8400	92	6	2	0	0	133	48	102	146	ST	+	-	+	-	ML	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-	40%
57	KALAVATHI	13000/16	32	F	HOMEMAKER	2y	-	+	-	-	+	-	43	7.1	3300	79	18	2	1	0	154	39	86	189	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
58	ATULRAY	28866/16	42	M	SHOPKEEPER	1y	-	-	+	-	-	-	584	13.2	4500	33	54	10	3	0	216	29	136	319	MI	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	60%
59	BHARATHI	13802/17	28	F	HOMEMAKER	1y	-	+	-	-	-	-	189	9.5	8100	84	13	3	0	0	116	39	76	149	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
60	MALLAPPA	11192/17	30	M	LABOURER	3y	-	+	-	+	-	-	29	7.1	7300	42	40	8	10	0	76	29	65	89	NSR	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	40%
61	ASHOK	12033/17	35	M	CLERK	5m	-	+	-	-	-	+	81	12.1	11030	85	10	3	2	0	159	32	125	119	IW MI	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	50%
62	YOUNUS	40281/16	40	M	MECHANIC	6m	-	+	-	+	-	+	40	10	6600	75	17	8	0	0	148	22	106	98	ST	+	-	+	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	--	40%
63	AJEET	41220/16	41	M	PAINTER	5m	-	+	-	+	+	+	42	8.9	6400	71	26	2	1	0	182	44	108	229	NSR	-	+	+	-	-	+	-	-	-	-	-	-	1	1	-	-	-	-	-	-	40%
64	MALAKAPPA	10922/17	48	M	FARMER	1y	-	-	-	-	+	+	411	13.3	3900	79	12	8	1	0	88	19	54	86	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
65	YANKAPPA	10833/17	34	M	DRIVER	2y	-	-	-	-	+	+	167	6.7	1600	74	14	9	3	0	169	34	112	192	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
66	KASTURI	4304/16	34	F	HOMEMAKER	4y	+	-	-	-	+	+	1048	8.5	8520	60	35	4	1	0	149	45	112	237	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
67	LAXMIBAI	11152/16	47	F	HOMEMAKER	2y	+	+	-	-	-	+	46	9.5	4450	80	16	2	2	0	86	34	90	110	NSR	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
68	SHANTABAI	3480/16	45	F	HOMEMAKER	3y	+	+	-	+	-	-	43	10.4	4600	72	20	8	0	0	176	34	99	216	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
69	SUSHILABAI	9677/17	34	F	HOMEMAKER	1y	-	+	-	+	+	+	148	11.8	7700	61	27	10	2	0	134	34	77	119	ST	-	+	+	-	MD	+	-	-	-	-	-	-	3	2	-	-	-	-	-	-	30%
70	BORAMMA	40697/16	36	F	FARMER	10m	-	+	-	-	-	-	39	7.2	3800	68	25	7	0	0	133	30	62	104	NSR	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	40%
71	SARASWATHI	39634/16	35	F	FARMER	6y	-	+	-	+	-	+	121	13.2	11500	85	10	5	0	0	155	28	110	83	NSR	+	-	+	-	ML	+	-	-	-	-	-	-	2	-	-	-	-	-	-	-	30%
72	SAMEER	12460/17	19	M	STUDENT	1y	+	+	-	-	-	-	144	12.6	2800	48	40	8	4	0	182	29	164	206	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
73	MAHADEVA	3378/15	47	M	FARMER	3m	-	+	+	-	-	-	512	12.7	7100	70	22	6	2	0	156	40	89	89	MI	-	-	-	-	-	-	+	+	+	-	-	-	3	-	-	-	-	+	-	40%
74	SHIVANAND	1674/15	38	M	DRIVER	5m	-	+	+	-	-	-	48	7.9	7100	71	19	7	3	0	216	30	186	308	IW MI	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	50%
75	BASAPPA	162/15	52	M	FARMER	1y	-	+	-	-	-	+	527	12.1	8800	72	26	2	0	0	79	18	86	106	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
76	ALLABAKASH	2235/15	40	M	WELDER	4y	-	-	-	-	+	+	32	7.7	8000	68	22	10	0	0	163	36	87	202	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	50%
77	SHARANAPPA	157/16	42	M	DRIVER	9y	+	+	-	-	+	-	42	7.7	3010	83	12	3	2	0	218	29	141	256	ST	-	+	+	-	MD	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	35%
78	BASAPPA	524/15	60	M	LABOURER	7y	-	+	-	-	+	+	412	10.3	33100	79	12	7	2	0	133	34	97	188	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
79	VIJAYKUMAR	2034/14	48	M	CLERK	4y	+	+	+	-	-	-	47	10.6	5520	58	30	4	8	0	88	20	62	113	MI	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	45%
80	KRISHNA	1543/14	43	M	POLICE	4y	+	+	-	-	+	-	32	10.9	5600	41	32	7	0	0	246	39	140	319	ST	-	+	+	-	MD	+	-	-	-	-	-	-	3	-	-	-	-	-	-	--	30%
81	SHOBHA	456/15	35	F	TAILOR	8y	+	+	-	-	-	+	86	9.8	3300	60	38	1	1	0	134	30	86	171	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%

82	RAGHAVENDRA	4677/14	32	M	LABOURER	2y	-	-	-	-	+	+	560	12.3	3600	57	39	2	2	0	156	31	106	148	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
83	SHIVANAND	876/14	32	M	FARMER	4y	+	+	-	-	-	-	155	15	6400	72	21	1	6	0	94	14	58	112	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	AML	-	-	60%
84	SURYAKANTH	199/14	44	M	DRIVER	2y	-	-	-	-	+	+	141	10.6	8900	90	10	0	0	0	116	31	92	148	ST	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	MA	-	-	60%
85	MARUTI	3874/15	36	M	LABOURER	8y	+	+	-	-	+	+	40	13.3	6300	75	23	2	0	0	80	12	48	103	ST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
86	KUMAR	3078/14	45	M	FARMER	6y	+	-	-	+	-	-	559	10.8	5900	52	43	3	2	0	76	35	87	134	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
87	PANDU	541/16	45	M	LABOURER	1m	+	+	-	+	-	+	645	12.5	5500	63	33	2	2	0	90	43	102	127	ST	+	-	+	-	ML	+	-	-	-	-	-	-	2	1	-	-	-	-	-	35%
88	GURAPPA	2423/15	38	M	FARMER	3m	-	+	+	-	-	-	512	12.7	7100	70	22	6	2	0	156	40	89	89	MI	+	-	-	-	-	-	+	+	+	-	-	-	3	-	-	-	-	+	-	40%
89	PANDU DODAMANI	25355/16	45	M	SHOPKEEPER	3Y	+	+	-	-	+	-	659	12.2	4620	72	24	0	4	0	180	50	107	98	NSR	+	+	+	+	+	+	-	-	-	-	-	-	2	3	-	-	-	-	-	25%
90	SHRISHAIL	16393/16	35	M	BUSINESS	2Y	+	-	-	-	-	-	430	12.9	6400	58	35	3	4	0	156	34	102	98	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
91	ASHOK	3758/17	59	M	EDITOR	2D	-	-	-	-	-	-	154	3.3	3300	68	27	0	5	0	110	39	56	77	NSR	-	-	-	-	MD	-	-	-	-	-	+	-	-	-	1	-	-	-	-	60%
92	BHIMANNA	19612/16	60	M	FAMER	10Y	+	+	-	-	-	+	650	9.3	2300	72	22	6	0	0	220	60	122	190	NSR	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	50%
93	PUSHPA	13313/16	47	F	HOMEMAKER	7Y	+	+	-	-	-	-	764	12.2	9780	58	37	3	2	0	262	36	145	404	NSR	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	50%
94	SHANTAPPA	20503/16	40	M	LABOURER	3Y	-	-	-	-	-	-	240	15.4	8260	76	20	2	2	0	140	23	97	100	MI	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	50%
95	SUJATHA	2407/17	40	F	HOMEMAKER	7Y	+	+	-	-	+	-	18	9.9	9130	89	10	1	0	0	137	23	95	96	NSR	-	-	-	+	ML	-	-	-	-	-	-	-	1	1	-	-	-	-	-	60%
96	SIDDAPPA	9276/16	40	M	BUSINESS	8Y	+	-	-	-	-	+	110	11.1	5870	94	5	1	0	0	low	low	low	81	NSR	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
97	SURESH	6779/16	50	M	FARMER	12Y	+	-	-	-	+	-	126	10.7	9080	82	12	1	5	0	250	low	low	268	NSR	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
98	SUNDRAWWA	15144/16	60	F	HOMEMAKER	20Y	-	-	-	-	+	-	500	10.2	6400	76	16	0	8	0	164	30	112	98	NSR	+	-	-	+	ML	-	-	-	-	-	-	-	1	1	-	-	-	-	-	50%
99	TIMMAPPA	13204/17	45	M	POLICE	1	+	-	-	-	-	-	143	6.9	7000	70	25	0	5	0	114	23	63	140	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
100	SADASHIV	14957/16	28	M	LABOURER	3	+	-	-	-	+	-	286	12.2	8170	86	12	1	1	0	118	25	65	136	NSR	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
101	RASHMI MAHADEV	19158/16	18	F	STUDENT	4y	+	-	-	-	-	-	210	10.9	3030	35	60	3	2	0	120	26	66	134	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
102	MUDAKAMMA	93483/16	20	F	STUDENT	6D	-	-	-	-	-	-	700	11.6	7170	46	40	2	12	0	155	30	111	68	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
103	GOPAL	25103/16	26	M	LABOURER	5Y	+	-	-	-	-	+	540	10.2	6270	86	10	2	2	0	164	44	100	81	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
104	DURANATH	3776/17	40	M	LABOURER	4Y	+	-	-	-	-	-	98	11.1	6000	80	16	0	4	0	192	57	95	199	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
105	BHASKAR	16580/16	48	M	DRIVER	2Y	+	-	-	-	-	+	227	10.6	10170	84	12	4	0	0	140	26	95	100	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
106	VIDYA	17723/16	45	F	HOMEMAKER	3Y	-	-	-	-	-	-	350	11.2	6820	72	18	2	6	0	136	24	92	94	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
107	BASAMMA	25340/16	35	F	LABOURER	2Y	+	+	-	-	-	-	370	13.1	7820	92	6	2	0	0	148	28	98	94	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
108	GANGAWWA	14471/16	45	F	HOMEMAKER	1D	-	-	-	-	-	-	540	6.2	4470	78	15	4	3	0	134	23	92	94	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
109	GIRIJA	960/17	38	F	HOMEMAKER	13Y	+	-	-	-	+	+	100	11.6	7000	68	27	0	5	0	189	46	117	131	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
110	ASHWAT	4650/17	46	M	FARMER	5Y	-	-	-	-	-	+	177	11.2	6324	80	16	0	4	0	119	28	45	231	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
111	BASAPPA	41157/16	54	M	FARMER	6Y	+	-	-	-	-	-	368	9	22890	93	6	1	0	0	192	57	95	199	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
112	VIJAYALAXMI	12788/16	28	F	HOMEMAKER	3Y	-	-	-	-	-	-	116	11.7	6700	63	31	0	6	0	192	57	95	184	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
113	MADIWALAPPA	8010/16	45	M	FARMER	6Y	+	-	-	-	-	-	560	5.5	6060	90	6	4	0	0	138	27	90	107	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
114	ADEWWA	3997/17	40	F	HOMEMAKER	1D	-	-	-	-	-	-	240	3	2860	91	7	1	1	0	146	28	98	116	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
115	SANGEETHA	16670/16	26	F	HOMEMAKER	2Y	+	-	-	-	-	-	488	7.2	6000	76	16	0	8	0	164	30	112	98	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
116	ALLABAKSHA	16674/16	40	M	AUTODRIVER	3Y	+	+	-	-	+	-	106	8.4	5470	92	7	1	0	0	146	26	97	104	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	55%
117	SIDDARAM PATIL	44248/17	45	M	LABOUR	3Y	-	+	-	-	-	+	27	9.2	7880	85.5	9.6	4.8	0	0.1	97	25	47	121	NSR	+	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
118	RAJSHEKHAR WADDAR	3579/18	55	M	FARMER	12Y	+	+	-	-	-	-	16	5.7	11100	87	8	0	5	0	122	14	37	354	NSR	+	-	-	+	ML	-	-	-	-	-	-	-	1	-	-	-	+	-	-	55%
119	MURGAYYA HIREMATH	42906/17	38	M	SHOPKEEPER	4Y	+	-	-	-	-	-	950	8.2	21850	78.6	14	6.4	1	0.1	low	low	low	low	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	1	1	-	-	-	-	-	60%
120	MAHADEVI ANTIN	1494/18	60	F	HOUSEWIFE	4M	+	-	-	-	-	-	152	8.8	6600	73	22	0	5	0	154	30	105	97	LVH	-	-	+	-	-	+	-	-	-	+	-	-	-	1	-	1	-	-	-	-	45%
121	LAXMAN KATTIMANI	73/18	50	M	LABOUR	7Y	+	+	-	+	-	-	86	2.5	10470	75.1	14	11	0.6	0	60	18	28	71	NSR	-	-	+	-	SV	-	-	-	-	-	-	-	-	-	3	-	-	-	-	-	55%
122	MALLAPPA SHIROL	41808/17	60	M	SHOPKEEPER	5M	+	+	-	+	+	-	94	10.1	9710	82.9	11	1.5	4.1	0.1	138	25	74	193	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
123	MALLAPPA PUJARI	40316/17	45	M	FARMER	10Y	+	-	-	-	-	-	79	3	3160	62.6	34	3.2	0	0	138	25	74	193	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
124	ADIVEPPA GODACHI	42713/17	50	M	FARMER	6Y	+	-	-	-	-	-	78	3.5	8400	80	14	0	6	0	low	low	low	88	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	-	-	1	-	-	-	-	60%
125	IRANNA LAGASHETTY	3687/18	28	M	SHOPKEEPER	6Y	+	-	+	-	-	-	98	10.5	11100	86	8	0	6	0	low	low	low	114	NSR	-	-	+	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	-	55%
126	SHILPA GODDI	778/18	20	F	STUDENT	20Y	+	-	-	-	-	-	102	11.6	13100	80	14	0	6	0	157	33	112	62	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
127	SUBASH PATIL	42236/17	52	M	BUSINESS	5Y	-	-	-	-	-	-	197	8.6	7760	77.1	16	5.2	1.2	0.1	138	25	74	193	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	-	-	1	-	-	-	-	60%
128	ASHABEE IMMUWALI	40649/17	40	F	HOUSEWIFE	4Y	+	+	-	-	-	-	24	8.8	2310	61	33	5.2	1.3	0	138	25	74	193	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%

129	PRAKASH RATHOD	43181/17	33	M	SHOPKEEPER	7D	-	-	-	-	-	-	356	8.5	16580	84.4	7.6	6.8	0.8	0.4	150	28	95	133	ST	-	+	+	-	MD	-	-	-	-	-	-	-	-	1	1	-	-	-	-	-	55%
130	GURU HARAJAN	43096/17	40	M	DRIVER	6Y	+	-	-	-	-	-	106	12	12150	70	24	4	2	0	113	41	47	126	NSR	+	+	-	-	MD	-	-	-	-	-	-	-	-	1	2	-	-	-	-	-	55%
131	SHARANAMMA BAGALI	41428/17	36	F	HOUSEWIFE	2Y	+	-	-	-	-	-	430	12.6	9500	53	40	4	3	0	172	24	121	137	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
132	SACHIN HALLI	40814/17	18	M	STUDENT	18Y	+	-	-	-	-	+	133	6.1	8100	49	30	4	7	3	low	low	low	low	ST	-	-	-	-	MD	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
133	BHEMANNA KARPENTER	44225/17	53	M	BUSINESS	5Y	+	-	-	-	-	-	10	11	7120	80	14	4.9	1	0.1	117	30	58	144	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
134	RAJSHEKHAR PATIL	40104/17	45	M	FARMER	4Y	+	-	-	-	+	-	254	12.2	4250	72.3	22	5.6	0	0	184	42	127	74	NSR	-	-	+	-	-	-	-	-	+	-	+	+	-	+	1	-	1	-	-	-	-	40%
135	MAHADEVAPPA BIRADAR	43179/17	26	M	STUDENT	6Y	-	-	-	+	-	-	10	13.7	11900	67.7	22	5.5	4.8	0.3	97	25	47	121	ECTS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
136	SHIVAPUTRA	42813/17	45	M	DRIVER	5Y	+	-	-	-	-	-	231	14.1	8880	81.3	13	5.7	0.1	0.1	209	42	135	162	LVH	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
137	SIDDAPPA BAJANTRI	2706/18	60	M	FARMER	8Y	+	-	-	+	-	-	234	7.3	5340	75	18	6.2	0.6	0	110	21	61	134	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
138	MURGAYYA	2947/18	39	M	CARPENTER	2Y	+	-	-	-	-	+	286	16.5	9870	55.4	34	6.4	4.5	0.2	120	28	78	68	NSR	-	-	-	-	ML	-	-	-	-	-	-	-	-	1	1	-	-	-	-	-	60%
139	SIDDAPPA HONNUTAGI	2667/18	60	M	FARMER	10Y	+	+	-	-	-	+	198	16.8	9870	55.4	34	6.4	4.5	0.2	113	21	64	139	NSR	-	+	+	+	ML	-	-	-	-	-	-	-	-	1	1	-	-	-	-	-	60%
140	HANAMANT MARATHI	2294/18	48	M	FARMER	2Y	+	-	-	-	-	-	234	9.8	7250	77.2	19	4.1	0.1	0.1	143	26	67	254	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
141	DURGANATH SHASHTRI	42826/17	43	M	LABOUR	4Y	+	-	-	-	-	-	97	12.5	7580	78.9	8.3	10	2.4	0.1	192	57	95	199	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
142	SHEKHAR RABAKAVI	44726/17	60	M	G.EMPLOYEE	9Y	+	-	-	-	-	-	168	10.5	4400	72.8	20	7.7	0	0	low	low	low	90	NSR	-	+	+	+	ML	-	-	-	-	-	-	-	-	-	1	1	-	-	-	-	55%
143	SHARADA JGADAL	44152/17	35	F	HOUSEWIFE	5Y	+	-	-	-	-	-	107	11.5	3600	70.6	25	3.3	1.4	0	204	42	113	243	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
144	MALLIKARJUN KADAPATTI	43283/17	60	M	FARMER	12Y	+	-	-	-	-	-	98	10.5	19050	85.3	8.7	5.8	0.1	0.1	150	27	104	95	NSR	-	-	-	+	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	60%
145	SURESH KAJAPUR	40483/17	37	M	SHOPKEEPER	10D	-	-	-	-	-	-	394	12.5	7040	63.5	18	5.7	13	0.1	150	28	95	133	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
146	HANAMANT DUNDAPPA	2294/18	48	M	FARMER	2Y	-	-	-	-	-	-	234	9.8	7250	77.2	19	4.1	0.1	0.1	143	26	67	254	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
147	GOPAL PADAGATTI	7874/18	33	M	DRIVER	10D	-	-	-	-	+	+	158	11.9	7940	73.9	19	6.9	0.1	0.1	204	42	113	243	NSR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	60%
148	SIDDAPPA BAJANTRI	8750/18	50	M	FARMER	5Y	+	+	-	-	-	+	96	3.7	1200	72.8	19	8.6	0	0	145	30	28	436	NSR	-	+	+	-	ML	+	+	+	+	+	-	+	-	1	1	-	-	-	-	-	30%