DISSERTATION ON

A STUDY ON PREVALENCE OF CARDIAC ABNORMALITIES IN PATIENTS ATTENDING ART CENTRE, THANJAVUR MEDICAL COLLEGE AND HOSPITAL AND THEIR CORRELATION WITH STAGE OF INFECTION

Dissertation Submitted To

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In partial fulfillment of the

rules and regulations, for the award of the

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APRIL - 2017

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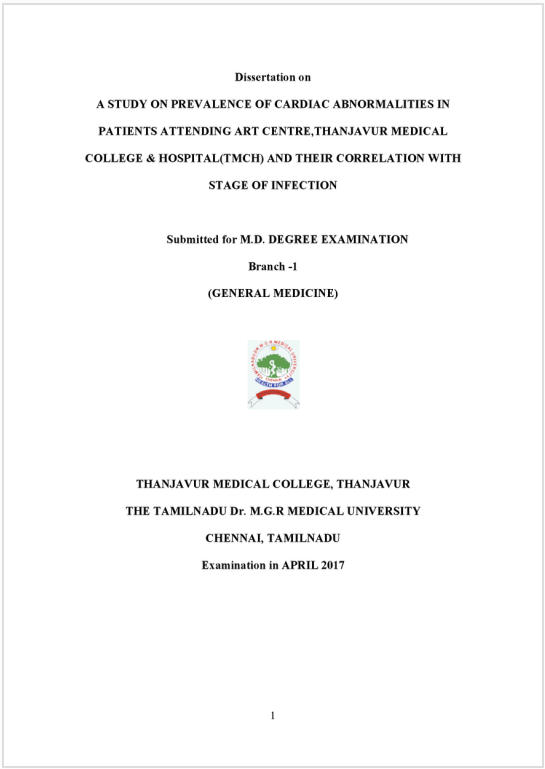

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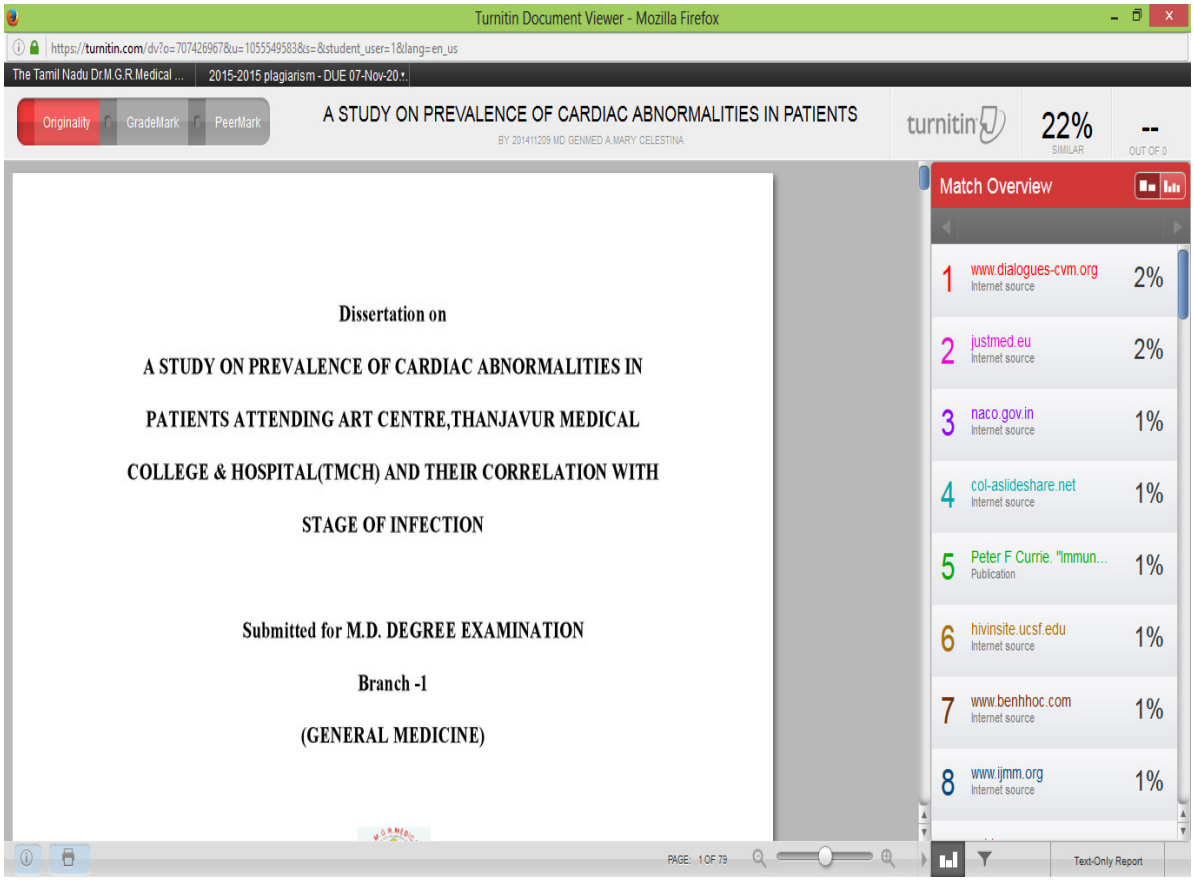
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CONTENTS

INTRODUCTION

Acquired Immuno Deficiency Syndrome was first documented in the United States in the year 1981 when the U.S Center for Disease Control and prevention (CDC) reported incidence of Pneumocystis jiroveci pneumonia in five previously healthy homosexual men. Within months, disease was recognized in injection drug users (IDUs), in people who received blood transfusions and patients with hemophilia[1].

Human Immuno Deficiency Virus was isolated from a patient who had lymphadenopathy in the year 1983 and in 1984 it was found clearly to be the causative organism of AIDS. India's first case of AIDS was reported in 1986 from Chennai[2].

HIV was isolated first at autopsy and later by non invasive techniques that cardiac manifestations can be caused by HIV infection. The reported frequency of cardiac abnormalities in PLHIV depends on the population under study and the definition of cardiac abnormality.

Prior to the advent of Anti-retroviral therapy (ART), clinically significant cardiac disease was present in almost all patient with HIV infection and was detected in most cases during postmortem. Cardiac abnormalities in AIDS patients appear to be more common than previously contemplated. When PLHIV were examined by echocardiography in late 1990, cardiac diseases were detected more often than could be expected from clinical symptoms and physical examination. Most conditions are clinically quiescent but some may have devastating and fatal outcomes. Pericardial effusion and Myocarditis are among the most commonly reported abnormalities though cardiomyopathy, endocarditis and coronary vasculopathy have also been reported.

It is expected that the risk of cardiac and cardiovascular disease will rise in the following years due to the cardiovascular risk profile and increased life expectancy of infected patients. Therefore diagnosis and therapy of HIV associated cardiovascular diseases should be an inherent part of current therapeutic concepts of HIV infection.

AIM OF THE STUDY

1. To study the prevalence of cardiac abnormalities in HIV infected individuals.

2. To correlate the cardiac abnormalities with stage of infection.

REVIEW OF LITERATURE

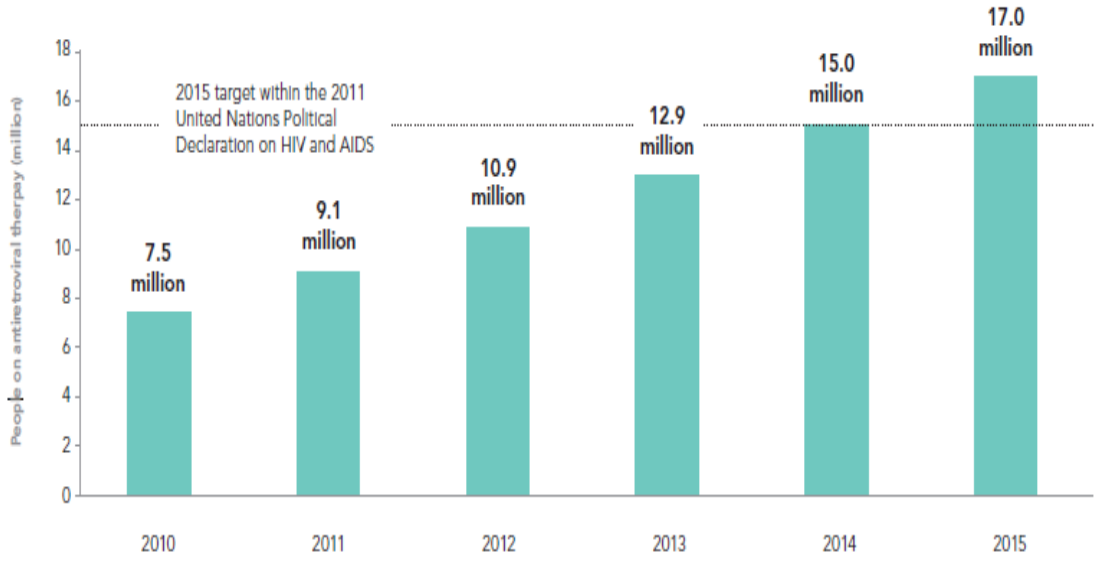
EPIDEMIOLOGY

GLOBAL SCENARIO OF HIV[3]

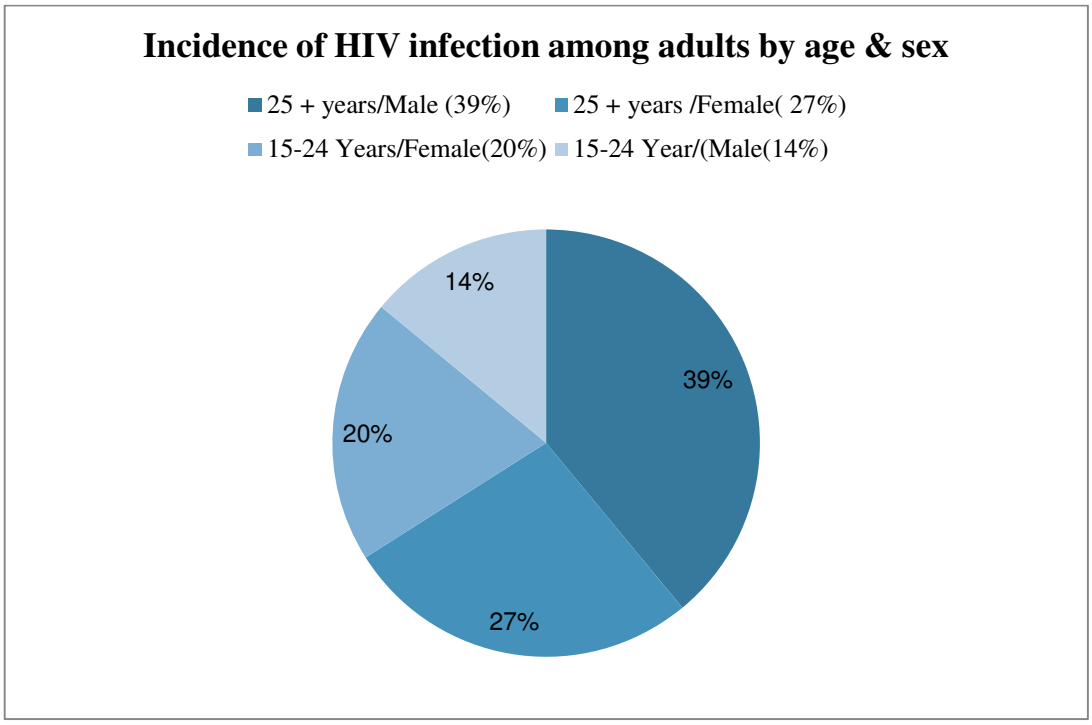
According to the UNAIDS report 2015, the HIV prevalence worldwide was 36.7 million (34 million-39.8 million) and the estimate of new HIV infections is 2.1 million(1.8 million-2.4 million).

The incidence of HIV infection among adults is remaining static in the recent era as compared with earlier data.

Prevalence of PLWHA on ART, global 2010 to 2015



Sources: Global AIDS Response Progress Reporting (GARPR) 2016; UNAIDS 2016 estimates.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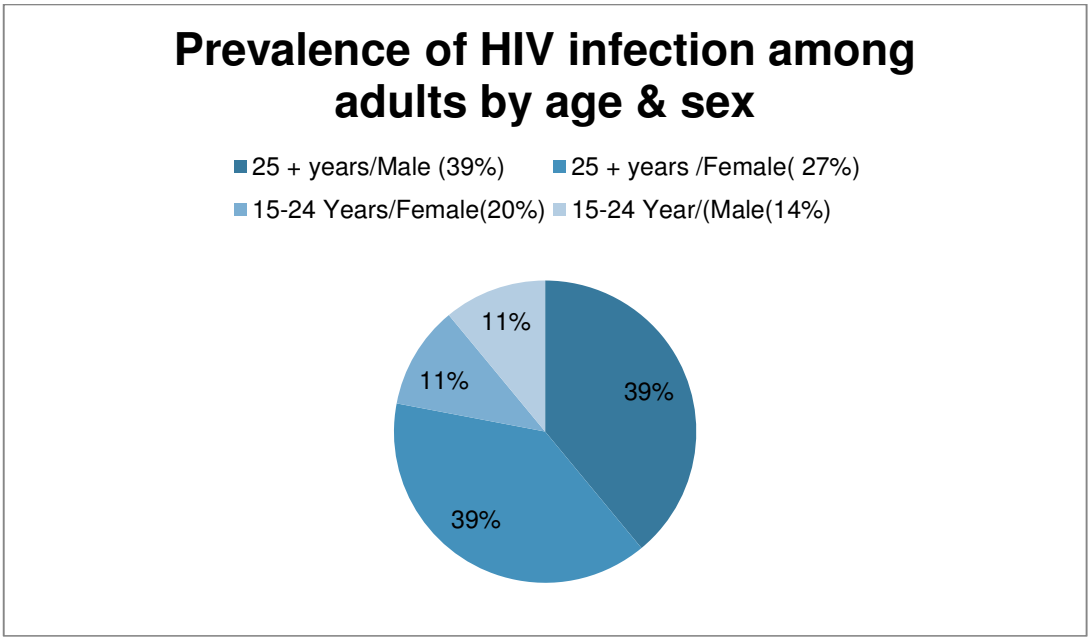
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11%
39%
39%

INDIAN SCENARIO OF HIV[4]

The India HIV Estimation 2015 report states that the National adult (15–49 years) HIV prevalence in India is 0.26% (0.22%–0.32%) in 2015. In 2015, the prevalence of HIV in adult males is 0.30% while in females it is 0.22%

Among the States/UTs, in 2015, Manipur has shown the highest prevalence of HIV (1.15%),whish is followed by Mizoram (0.80%), Nagaland (0.78%), Andhra Pradesh & Telangana (0.66%), Karnataka (0.45%), Gujarat (0.42%)

Goa (0.40%). The other states that have shown estimated adult HIV prevalence greater than the national prevalence (0.26%) are Maharashtra, Chandigarh, Tripura and Tamil Nadu , while Odisha, Bihar, Sikkim, Delhi, Rajasthan and West Bengal have shown an estimated adult HIV prevalence in the range of 0.21– 0.25%. All other States/UTs have levels of adult HIV prevalence below 0.20%.

The total number of People Living with HIV (PLHIV) in India is estimated at 21.17 lakhs (17.11lakhs–26.49 lakhs) in 2015 which has declined when compared with 22.26 lakhs (18.00 lakhs-27.85 lakhs) in 2007.The number of children (< 15 years) with HIV infection is estimated at 6.54%.

India is estimated to have around 86 (56–129) thousand new HIV infections in 2015,which has declined when compared with 66% in 2000 and 32% in 2007,the baseline year according to NACP-IV. Adults constituted 88% (75.9 thousand) of total new infections while children accounted for 12% (10.4 thousand) of total new infections were among adults (15+years). Telangana & Andhra Pradesh , Gujarat, Uttar Pradesh and Bihar currently account for 47% of total new infections among adults with each of these States contributing 7500 or more new infections in 2015.

SCENARIO IN TAMIL NADU

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

HUMAN IMMUNODEFICIENCY VIRUS [1]

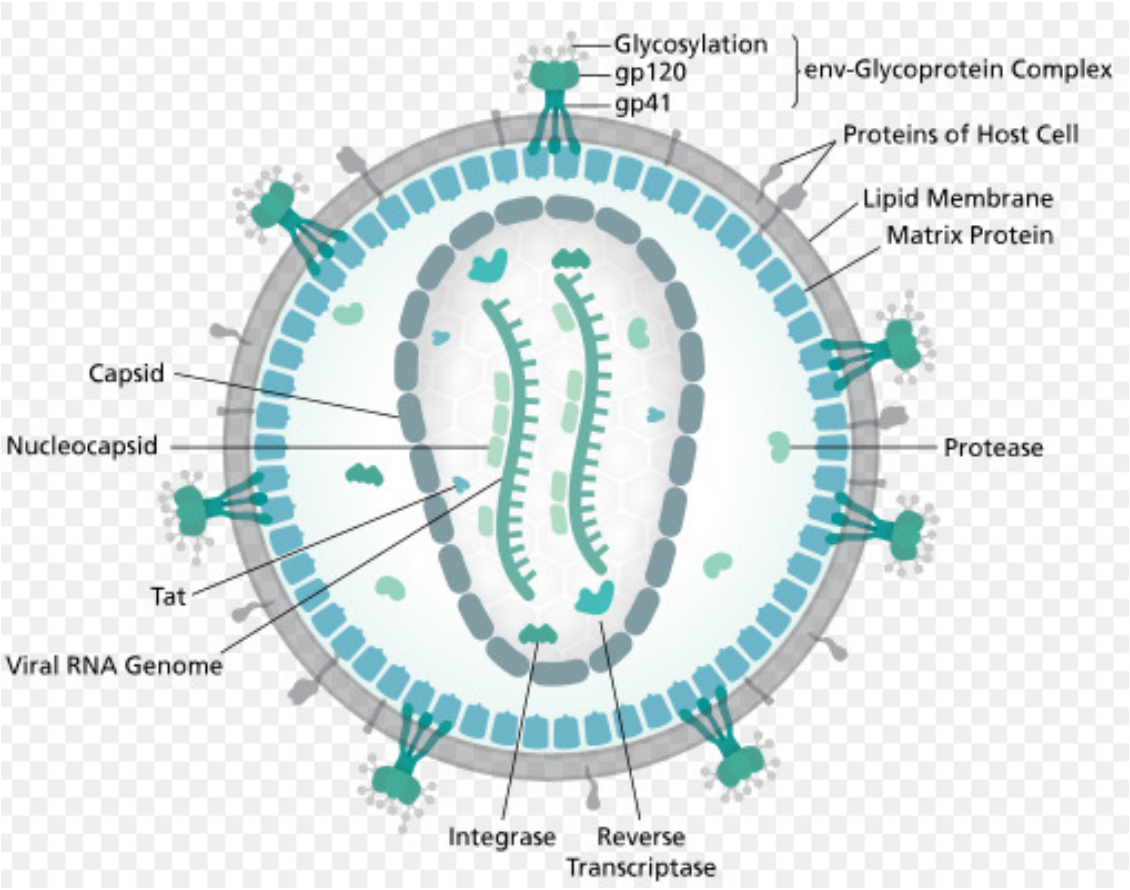
The origin of HIV is unclear. The most likely scenario is that HIV was introduced into human from another primate in Sub-Saharan Africa.

ETIOLOGIC AGENT

The etiologic agent of AIDS is Human Immuno Deficiency Virus (HIV) which belongs to the family of human retroviruses (Retroviridae) and the sub families of lentiviruses. There are two sub types of HIV namely HIV-1 and HIV 2. They are cytopathic viruses. HIV-2 is more similar to SIV (Simian Immuno Deficiency Virus) than HIV-1 and it is much less virulent usually not resulting in full blown AIDS, but still fatal.

MORPHOLOGY OF HIV

Electron microscopy shows that the HIV is a spherical enveloped virus, about 90 - 120 nm in size.Its basic structure constitutes an outer envelope, HIV matrix proteins and viral core. The outer envelope is made up of lipid bilayer in which the envelope proteins are embedded, namely glycoprotein 120(gp 120) and gp 41.The glycoprotein gp 120 is the main protein involved in the attachment of the virion to the host cell while gp 41 participates in cell fusion process.The core virus particle is composed of ribonucleoproteins. The virion contains two single stranded RNA surrounded by the capsule protein p24.The HIV matrix proteins are found between the HIV envelope and the viral core.

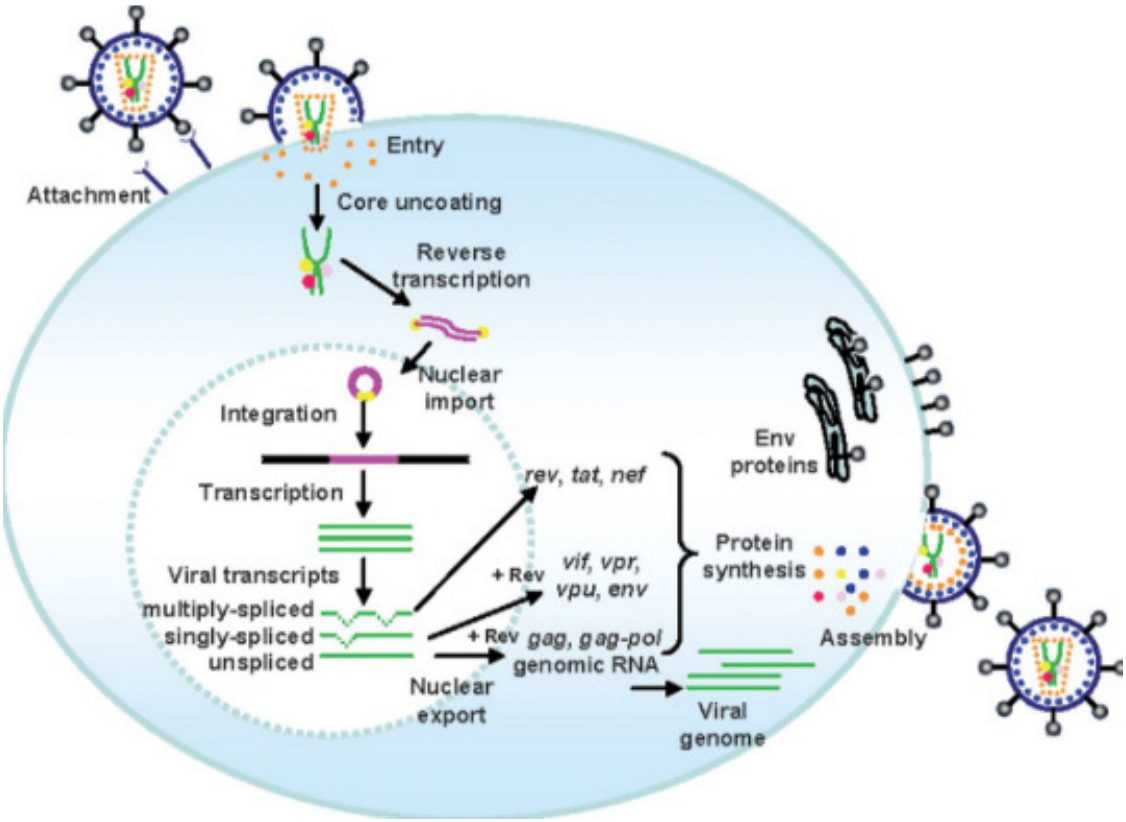


Structure Of HIV Virion

HIV GENOME

HIV-1 has the following genes. gag – encodes the proteins that form the core of virion. pol – encodes viral enzymes necessary for replication, reverse transcriptase, integrase and protease and env – encodes glycoprotein. It also contains atleast six other genes tat, rev, nef, vpr, vpu which code for proteins involved in the regulation of gene expression. The major difference between the genomes of HIV-1 and HIV-2 is the fact that HIV-2 lacks the vpu gene and has vpx gene which is not present in HIV-1.

LIFE CYCLE



ATTACHMENT AND ENTRY

The replication cycle of HIV begins with the high affinity binding of the gp 120 protein via a portion of its V1region near the N terminus to its receptor on the host cell surface, the CD4 molecule. It is also expressed on the surface of monocytes and dendrites / langerhans cells.The envelope

protein gp 120 binds to CD4 and undergoes a conformational change that facilitates binding to one of a group of co-receptors. CCR5 and CXCR4 constitute the two major co-receptors.

REVERSE TRANSCRIPTION AND INTEGRATION

Following binding of the envelop protein to the CD4 molecule, the virus is "uncoated" and the viral RNA is converted into complementary DNA (C-DNA) by virion associated reverse transcriptase enzyme. The C-DNA is transported to the host cell nucleus and eventually gets incorporated into the host cell chromosomes by virus specific integrase enzyme.

TRANSCRIPTION, TRANSLATION AND REPLICATION

The integrated DNA is transcribed into messenger RNA (mRNA) which comes out into cytoplasm and viral proteins are synthesized using protein synthesizing machinery and raw material from the host cell. Some of the viral proteins are synthesized as polyproteins that are eventually cleared by the proteinase enzyme.

MATURATION AND RELEASE

Newly synthesized progeny RNA and proteins are packaged together and the newly formed virus particles are released from the infected cell by the budding process.

PROGRESSION OF ILLNESS[1]

The median time from primary HIV infection to the development of AIDS in untreated individuals is approximately 10 years. Long term survivors are those who survive for >= 20 years after initial infection. It may be related to beneficial effect of ART and prophylaxis against opportunistic infections. Long term nonprogressors are those who have been infected with HIV for ≥ 10 years without decline in the CD4 count below the normal range and who are not on ART.

The reasons being

1. Mutant nef gene of HIV
2. Heterozygosity for CCR5- Δ32 deletion
3. Heterozygosity for CCR2-64I mutation
4. Homozygosity for SDF1-3'A mutation
5. Heterozygosity for the RANTES-28G mutation 11

CLASSIFICATION [1]

1993 Revised classification system for HIV infection and expanded AIDS surveillance case definition for adolescents and adults.

Table 1

CD4+ T cell Categories	Clinical categories		
	A	B	C
	Asymptomatic, Acute (primary) HIV or PGL	Symptomatic, Not A or C Conditions	AIDS-Indicator Conditions
≥ 500 /µl	A1	B1	C1
200 - 499 /µl	A2	B2	C2
< 200/ µl	A3	B3	C3

PGL – Progressive Generalized Lymphadenopathy HIV infected persons classified in A3, B3, C1, C2 and C3 are AIDS cases.

“ Category A:

One or more of the following conditions in adolescents or adults (> 13 years) with documented HIV infection. Conditions listed in categories B and C must not have occurred.

1. Asymptomatic HIV infection

2. Progressive Generalized Lymphadenopathy

3. Acute (primary) HIV infection with accompanying illness or history of acute HIV infection.

Category B:

Consists of symptomatic conditions in an HIV infected adolescent or adult that are not included among conditions listed in clinical category C and that meets one of the following criteria

1. The conditions are attributed to HIV infection or are indicative of a defect in cell mediated immunity (CMI).
2. Conditions are considered by physicians to have a clinical course or to require management that is complicated by HIV infection. Examples include, but are not limited to the following.

a. Bacillary angiomatosis

b. Candidiasis, oropharyngeal (thrush)

c. Candidiasis, vulvovaginal: persistent, frequent or poorly responsive to therapy

d. Cervical dysplasia (moderate or severe) / cervical carcinoma in situ

e. Constitutional symptoms, fever or diarrhea lasting > 1 month

f. Oral hairy leukoplakia

g. Herpes zoster involving atleast 2 distinct episodes or more than one dermatome

h. Idiopathic thrombocytopenic purpura

i. Listeriosis

j. Pelvic inflammatory disease, particularly complicated by tuboovarian abscess

k. Peripheral neuropathy

Category C:

Conditions listed in AIDS surveillance case definition

a. Candidiasis of bronchi, trachea or lungs

b. Candidiasis, esophageal

c. Cervical cancer, invasive

d. Coccidioidomycosis, disseminated or extrapulmonary

e. Cryptococcosis, extrapulmonary

f. Cryptosporidiosis, chronic intestinal (> 1 month's duration)

g. Cytomegalovirus disease (other than liver, spleen or nodes)

h. Cytomegalovirus retinitis (with loss of vision)

i. Encephalopathy, HIV related

j. Herpes simplex: chronic ulcers (> 1 month's duration) or bronchitis, pneumonia or esophagitis

k. Histoplasmosis, disseminated or extra pulmonary

l. Isosporiasis, chronic intestinal (> 1 month's duration)

m. Kaposi's sarcoma

n. Lymphoma, Burkitt's (or equivalent term)

o. Lymphoma, primary of brain

p. Mycobacterium avium complex or M. kansasii, disseminated or extrapulmonary

q. Mycobacterium tuberculosis, any site (pulmonary or extra pulmonary)

r. Mycobacterium, other species or unidentified species, disseminated

s. or extrapulmonary

t. Pneumocystis jiroveci pneumonia

u. Pneumonia, recurrent

v. Progressive multifocal leukoencephalopathy

w. Salmonella septicemia, recurrent

x. Toxoplasmosis of brain

y. Wasting syndrome due to HIV"

CLASSIFICATION OF HIV INFECTION (WHO CLINICAL STAGING SYSTEM) [7]

Clinical stage 1:

a. Asymptomatic

b. Persistent Generalized Lymphadenopathy

Clinical stage 2:

a. Weight loss <10% of body weight

b. Recurrent upper respiratory tract infections (e.g. bacterial sinusitis)

c. Herpes zoster

d. Angular cheilitis

e. Recurrent oral ulceration

f. Papular pruritic eruptions

g. Seborrhoeic dermatitis

h. Fungal nail infections

Clinical stage 3:

a. Weight loss > 10% of body weight

b. Unexplained chronic diarrhea > 1 month

c. Unexplained persistent fever (intermittent or constant) > 1 month

d. Persistent Oral Candidiasis (thrush)

e. Oral hairy leukoplakia

f. Pulmonary tuberculosis

g. Severe bacterial infections (e.g. pneumonia, pyomyositis)

h. Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis

i. Unexplained anemia (< 8 g / dl), neutropenia (< 0.5 X 109 / litre) and or chronic thrombocytopenia

Clinical stage 4:

a. HIV wasting syndrome

b. Pneumocystis jiroveci pneumonia

c. Recurrent severe bacterial pneumonia

d. Toxoplasmosis of the brain

e. Chronic Cryptosporidiosis

f. Chronic Isosporiasis

g. Cryptococcosis - extrapulmonary

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

i. HIV Encephalopathy

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

k. Disseminated endemic mycosis (Extrapulmonary Histoplasmosis, Coccidiomycosis)

l. Kaposi's sarcoma

m. Candidiasis - esophagus, trachea, bronchi or lungs

n. Disseminated non-tuberculous mycobacteria infection

o. Mycobacterium tuberculosis, extrapulmonary

p. Progressive multifocal leukoencephalopathy

q. Recurrent septicemia (including non typhoid salmonella septicemia)

r. Lymphoma (Cerebral or B cell Non-Hodgkin)

s. Invasive cervical carcinoma

t. Atypical disseminated leishmaniasis

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

WHO CASE DEFINITION FOR AIDS SURVEILLANCE IN ADULT WHERE HIV TESTING FACILITIES NOT AVAILABLE [7]

Case definition for AIDS is fulfilled if at least two major signs and one minor sign are present

Major signs:

a. Weight loss > 10% of body weight

b. Chronic diarrhea > 1 month

c. Prolonged fever > 1 month

Minor signs:

a. Persistent cough > 1 month

b. History of herpes zoster

c. Oropharyngeal candidiasis

d. Generalized lymphadenopathy

e. Chronic progressive herpes simplex infection

WHO GUIDELINES FOR INITIATION OF ART IN ADULTS AND ADOLESCENTS [7]

Table 2

Classification of HIV associated clinical disease	WHO clinical stage	CD4 test not available or pending	CD4 test available
Asymptomatic	1	Do not treat	Treat if CD4count <200
Mild symptoms	2	Do not treat	
Advanced symptoms	3	Treat	Consider treatment if CD4 <350 and initiate ART before CD4 drops below 200
Severe / Advanced symptoms	4	Treat	Treat irrespective of CD4 count

NACO GUIDELINES FOR INITIATION OF ART IN ADULTS AND ADOLESCENTS[6]

Table 3

CD4 count (cell /mm^3)	Actions
< 200	Treat irrespective of clinical stage
200 – 350	. Offer ART for symptomatic patients . Initiate treatment before CD4 drop below 200 cells/mm^3 for asymptomatic people *
>350	Defer treatment in asymptomatic persons

* If CD4 count is between 200- 250, this should be repeated in four weeks and treatment to be considered in asymptomatic patients.

British HIV association (BHIVA) suggests initiation of ART for asymptomatic HIV infected individuals having less than 200 CD4 T cell counts.70 International AIDS society recommends initiation of ART in asymptomatic individuals with CD4 count > 200 to 350 cells / µl and viral load 50000 - 100000 copies / ml.

In a study by Ramalingam et al, conducted in 2001 have shown that mean CD4 counts in south Indian population both normal and HIV infected individuals are lower than in western population and have proposed a modified classification based on CD4 cell count for south Indians. The

categories of CD4 count proposed were cell count > 300, 81 - 300, ≤ 80 cells / μL, instead of the ≥ 500, 201-499, ≤ 200 recommended by CDC(Centre for Disease Control and prevention).

Kannagai et al study conducted in 2008 has shown that majority of HIV infected individuals in South India with CD4 counts of 200 - 350 cells / μL had higher viral load than that suggested by International AIDS Society.

CARDIAC MANIFESTATIONS IN HIV INFECTED INDIVIDUALS

The reasons for the paucity of knowledge about the etiology of HIV associated cardiovascular diseases are

1. In the early years of AIDS epidemic, most patients died of infectious complications, before the manifestations of cardiovascular complications.

2. Because cardiomyocytes do not have CD4 receptors, the heart was thought to be unaffected by HIV infection.

3. Presence of cardiovascular risk factors like poor nutrition, alcohol and drugs that can lead on to cardiac disease in HIV infected individuals.

4. Cardiac disease remains relatively asymptomatic in early stages of HIV infection.

5. Heart disease can be overlooked in HIV-positive patients, because symptoms of breathlessness, fatigue and poor exercise intolerance are frequently ascribed to other conditions associated with HIV infection.

Around 40% of HIV infected patients are found to have cardiac disease at autopsy and 25% of patients with AIDS have lesions detectable by echocardiography. Many of these lesions are mild, and HIV related heart disease probably causes symptoms in less than 10% and death in less than 2% of all patients with HIV infection.

At the beginning of the epidemic, heart muscle disease was the dominant cardiac complication of HIV infection in developed world, and tuberculous pericarditis in Africa. The advent of HAART (highly active anti-retroviral therapy) has changed the pattern of disease in developed countries where premature coronary artery disease and other manifestations of atherosclerosis are now the most common cardiovascular disorder.

This is partly caused by HAART-induced metabolic problems, particularly insulin resistance and hyperlipidemia, but also reflects a high prevalence of conventional risk factors such as smoking.

Most of the cardiac problems associated with HIV are due to advanced immunodeficiency and are found mainly in developed countries due to poor accesss of anti-retroviral therapy.

CARDIAC MANIFESTATIONS OF HIV/AIDS[8]

Table 4

Pericardial effusion	. Idiopathic . Infections (viral, bacterial and fungal) . Neoplastic (Kaposi's sarcoma and Non Hodgkins Lymphoma)
Heart muscle disease	. Myocarditis (idiopathic / lymphocytic, infections, toxins) . Dilated cardiomyopathy & Left Ventricular dysfunction
Endocarditis	. Marantic (nonbacterial thrombotic endocarditis) . Infective
Tumors	. Kaposi's sarcoma . Lymphoma
Right ventricular dysfunction & Pulmonary hypertension	. Primary . Secondary (recurrent chest infections, thromboembolism)
Premature atherosclerosis and Coronary artery disease	. Protease inhibitors, chronic inflammation,
Autonomic dysfunction	. CNS disease, drugs, prolonged immunodeficiency, malnutrition
Arrythmias	. Drugs, autonomic dysfunction, acidosis, electrolyte abnormalities
Vasculitis	. Antibiotics and antivirals
Adverse drug effects	. Hyperlipidemia . Proarrythmia

CARDIOVASCULAR ASSESMENT OF THE HIV/AIDS PATIENT

Heart disease can be overlooked in HIV-positive patients, because symptoms of breathlessness, fatigue and poor exercise intolerance are frequently ascribed to other conditions associated with HIV infection. Echocardiographic assessment of HIV patients is extremely useful and can be used to identify those cardiac conditions that can be associated with poor outcome: pericardial effusion,left ventricular (LV) systolic dysfunction / heart muscle disease,intracardiac masses.

INDICATIONS FOR ECHOCARDIOGRAPHIC ASSESSMENT OF HIV POSITIVE PATIENTS [8]

1) Possible baseline assessment at the time of diagnosis of HIV infection

2) Base line assessment and 1 - 2 yearly monitoring of patient with

i) Clinical manifestations of possible cardiac involvement

- Unexplained dyspnea / hypoxia
- Third heart sound, inappropriate tachycardia
- Raised jugular venous pressure
- Peripheral edema / right heart failure
- Radiographic evidence of cardiomegaly

ii) Viral Coinfection

- Cytomegalovirus
- Epstein-Barr virus

- Coxsackie virus
- Adeno virus

iii) History of preexisting cardiac disease

- Left ventricular systolic dysfunction (any cause)
- Valvular heart disease
- Suspicion of infective endocarditis in intravenous drug users

iv) High-risk HIV patients with:

- Wasting and Encephalopathy
- CD4 count < 100 cells / mm3 or AIDS
- Potentially cardiotoxic medications
- Multiple hospitalizations

3) Possible 1-2 yearly monitoring of asymptomatic HIV positive patients

4) Frequent assessment of HIV positive patients with cardiovascular involvement (as guided by cardiologist)

HIV/AIDS AND THE PERICARDIUM

Pericardial effusion and pericarditis are the most common abnormalities found in early HIV/AIDS autopsy studies. Effusions are generally small and asymptomatic.

Pericardial effusion may be related to an opportunistic infection, metabolic abnormality or malignancy, but most often a clear etiology is not found. 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. Other causes can include uremia from HIV-associated nephropathy. Effusion markedly increases the mortality.

Screening echocardiography is recommended for HIV-infected individuals regardless of the stage of the disease. Patients should undergo pericardiocentesis if they have pericardial effusion and clinical signs (elevated jugular venous pressure, dyspnea, hypotension, persistent tachycardia, pulsus paradoxus) or Echocardiographic signs of tamponade (Continuous Wave Doppler evidence of respiratory variation in valvular flow, septal bounce, right ventricular collapse, a large effusion).

Patients with pericardial effusion without tamponade should be evaluated for treatable opportunistic infections, such as tuberculosis and for malignancy. Repeated echocardiography is recommended after one month or sooner if clinical symptoms direct.

HIV/AIDS AND THE MYOCARDIUM

MYOCARDITIS

Numerous pathologic studies have confirmed the presence of varying histologic patterns of lymphocytic myocarditis in HIV patients.15 As such, estimates of the prevalence of myocarditis in HIV/AIDS varies from 53% 16 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 including:

1) Primary HIV myocarditis

2) Secondary HIV myocarditis

3) Opportunistic infections

4) Autoimmunity

1) Primary HIV Infection of the Myocardium

HIV neither has been universally accepted nor unambiguously proven causative agent of myocarditis in AIDS. Although HIV can clearly infect monocytes/macrophages and myocardial interstitial cells, evidence proving that HIV can infect human cardiac myocytes which do not possess CD4 receptor is less clear.

HIV gene sequences have been detected by PCR in microdissected endomyocardial biopsies from HIV-positive patients some of whom had cardiac symptoms. HIV has also been shown gain entry into human fetal cardiac myocyte by ingestion through a specific crystallizable fragment (of immunoglobulin) (Fc) receptor, and it remains possible that this or other, unidentified mechanisms can promote HIV entry into the myocyte and facilitate a primary HIV myocarditis.

2) Secondary HIV Myocarditis

Interstitial lymphocytes and macrophages can form contact with myocytes causing focal loss of basement membrane through a local reaction.20 Proteolytic enzymes released through HIV replication in the interstitium could also damage myocytes. The HIV envelope glycoprotein group 120 can induce tumor necrosis factor-α (TNF-α) expression from macrophages and has been shown to enhance IL-1induced nitric oxide production in neonatal rat cardiac myocytes. Cytokine IL-6 which has some effect on immune response and viral replication in murine myocarditis, has been found in excess in small number of HIV-positive patients with biopsy proven myocarditis.

3) Myocardial Opportunistic Infections in HIV/AIDS

Autopsy has confirmed a variety of opportunistic infections of the myocardium in patients with AIDS. Infectious agents included Toxoplasma gondii, Cryptococcus, cytomegalovirus, Candida, Pneumocystis jiroveci, Microsporidium, Histoplama capsulatum, Atypical mycobacteria and Aspergillus organisms involving the myocardium.

4) Autoimmunity

Many autoimmune processes have been described in association with HIV/AIDS infection. HIV infection can itself trigger autoimmune phenomenon in susceptible patients. The presence of auto antibodies along with hypergammaglobulinemia and elevated circulating immune complexes suggests that yet unidentified autoimmune process can take place in HIV positive patients.

The symptoms of myocarditis are protean and include fatigue, dyspnea and pleuritic chest pain. The signs are unexplained tachycardia, third heart sound or a friction rub. ECG may show nonspecific conduction defects, repolarization abnormalities and ST-T wave changes, although these are not invariable.

Chest radiograph can be normal or suggest cardiac enlargement with pulmonary congestion. Echocardiography is usually non diagnostic but can show hyperdynamic LV function in HIV-positive children or occasionally LV dyskinesia in adult AIDS patients.

DILATED CARDIOMYOPATHY AND LEFT VENTRICULAR DYSFUNCTION IN HIV/AIDS

The prevalence of heart muscle disease appears to be approximately 4.4% for dilated cardiomyopathy and 6.4% for isolated LV dysfunction and the condition can cause symptoms in up to 5.5% of HIV/AIDS patients. 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 CD4 counts.

Mechanisms of Cardiomyopathy in HIV/AIDS

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

Drug Induced Heart Muscle Disease

Zidovudine and other Nucleoside Reverse Transcriptase Inhibitors (NRTIs) can be implicated in the development of some cases of heart muscle disease. In addition to inhibiting HIV reverse transcriptase, the drug causes a dose dependent reversible skeletal myopathy by altering mitochondrial DNA replication. Foscarnet in CMV infection associated with reversible congestive cardiac failure as has doxorubicin and interferon-α therapy in Kaposi's sarcoma. The effect of recreational drugs like cocaine use has been associated with myocarditis and a possibly reversible dilated cardiomyopathy in non-AIDS patients should be considered in the HIV population.

Nutritional Deficiencies and Cardiac Dysfunction in HIV/AIDS

HIV/AIDS patients with evidence LV systolic dysfunction should be assessed for micro nutrient deficiency, which is common in HIV infected individuals. Abnormally low levels of selenium and antioxidants have been demonstrated and oxidative stress can be an important mechanism for cellular damage in AIDS. Selenium deficiency is implicated in the pathogenesis of Keshan disease, a specific form of dilated cardiomyopathy in China, which can respond to dietary supplementation. In the same way, decreased selenium content has been demonstrated in the hearts of AIDS patients.

L- Carnitine deficiency has also been described in HIV patients, possibly in association with cardiac symptoms and in whom supplementation can be advantageous. Experimentally, Carnitine administration reversed myopathic changes induced by Zidovudine (AZT) in vitro, but the clinical effects have yet to be established.

The echocardiographic features of dilated cardiomyopathy is global LV systolic dysfunction with the consistent feature of reduced ejection fraction. LV dilatation can result in mitral valve distortion and lead to regurgitation. In AIDS patients, mitral regurgitation has also been described with infective endocarditis. Abnormalities of mitral flow, specifically reduced early mitral peak velocity (E) and other indices of diastolic dysfunction have been noted early in the course of HIV/AIDS with normal ejection fraction and in association with LV systolic dysfunction.

No randomized trials have been reported regarding the effectiveness of current heart failure therapies in people with HIV/AIDS.9 Common agents such as diuretics, aldosterone antagonists, and digoxin can improve well being. Angiotensin inhibitors can be poorly tolerated, possibly because many patients already have low systemic vascular resistance. Etarnacept, pentoxifylline have been used in severe heart failure with some success.

Intravenous immunoglobulin therapy has been used successfully in children with symptomatic HIV heart muscle disease and can be protective against the development of LV dysfunction in that group. The use of cardiac resynchronization therapy has not been described in HIV population, although case reports of successful use of LV assist devices and orthotopic heart transplant exist, although these are uncommon.

NONBACTERIAL THROMBOTIC ENDOCARDITIS

Marantic or nonbacterial thrombotic endocarditis (NBTE) is a condition in which friable clumps of platelets and red cells adhere to the cardiac valves. Unlike bacterial endocarditis these lesions are not infective and show no evidence of an inflammatory reaction.

The pathogenesis is not fully understood, but hypercoagulability, immune complex deposition, or specific vitamin deficiency can be important in conjunction with endothelial damage from intravenous catheters or injected particulate matters. Any heart valve can be affected and frequently multiple lesions are found on different levels.

Treatment should focus on reducing the underlying disease causing coagulation abnormalities, valvular endothelial damage or both. An anticoagulation risk benefit assessment must be made on individual basis.

INFECTIVE ENDOCARDITIS

The immunological abnormalities associated with HIV render patients susceptible to bacterial infections. The clinical presentation is same for both HIV positive and negative patients but runs a more fulminant course in later stages of AIDS. The most common valve involved is the tricuspid valve as with infective endocarditis in HIV negative intravenous drug users. Most frequently isolated organisms include staphylococcus aureus, salmonella species, streptococcus viridans but fungal endocarditis can occur in end-stage AIDS. Antimicrobial treatment may have to be widened. Operative indications include hemodynamic instability, failure to sterilize blood cultures after appropriate intravenous antibiotics and severe valvular destruction in patients with reasonable life expectancy after recovery from surgery.

CARDIAC TUMORS IN HIV/AIDS

Kaposi's sarcoma (KS) is the most common AIDS related neoplasia; there is often widespread and potentially fatal visceral involvement in HIV-positive individuals. The prevalence of cardiac Kaposi's sarcoma appears to have decreased significantly since early reports.

It's not usually associated with symptoms of cardiac dysfunction; but cases of fatal tamponade associated with the tumor have been reported, and heart failure without ventricular dilatation can occur in cases with extensive myocardial infarction.8 Kaposi's sarcoma can be treated with daunorubicin, doxorubicin or related anthracyclines. Liposomal encapsulated daunorubicin has an improved pharmacological profile and can therefore be preferred in patients with Kaposi's sarcoma and AIDS.

Non Hodgkin's Lymphoma (NHL) can involve the pericardium or myocardium. Cardiac lymphoma commonly gives rise to clinical symptoms of tamponade, heart failure, and conduction abnormalities or superior vena cava syndrome. Systemic chemotherapy with or without concomitant radiation or surgery has been beneficial in some patients, but overall the prognosis is poor.

RIGHT VENTRICULAR DYSFUNCTION

Isolated right ventricular dysfunction without pulmonary hypertension is of unknown significance and can be related to changes in pulmonary circulation. Therefore bronchopulmonary infections should be treated aggressively and intravenous drug use, which can result in micro vascular pulmonary emboli should be discouraged.

PULMONARY HYPERTENSION

Primary pulmonary hypertension is estimated to occur in 0.5% of hospitalized AIDS patients. Although pulmonary hypertension can be related to the action of viral proteins or the action of cytokines on the endothelial cell, characteristic pulmonary arteriopathy is found in HIVrelated pulmonary hypertension. Right heart catheterization can be worthwhile to determine if pulmonary hypertension can be reversed and has been used in HIV patients. HAART itself can be beneficial in terms of outcome in pulmonary hypertension but agents like bosentan, epoprostenol, treprostinil or sildenafil can improve feeling of well being without altering prognosis.

ACCELERATED ATHEROSCLEROSIS

Accelerated atherosclerosis has been observed in young HIVinfected adults and children without traditional coronary risk factors. Protease inhibitor therapy markedly alters lipid metabolism and can be associated with premature atherosclerotic disease. Chronic inflammatory states have also been associated with atherosclerotic disease. Atherosclerotic disease is believed to have multi factorial causes and is prone to plaque rupture, possibly related to the host environment.

Lipodystrophy should be recognized and treated because of an elevated 10 year cardiovascular risk. Risk stratification based on traditional risk factors plus diet, alcohol intake, physical exercise, hypertriglyceridemia, cocaine use, heroin use, thyroid disease, renal disease and hypogonadism should be considered for long term cardiac preventive care.

VASCULITIS

Most types of vasculitis have been reported in HIV-infected patients. Vasculitis should be suspected in patients with fever of unknown origin, unexplained multi system disease, arthritis or myositis, glomerulonephritis, peripheral neuropathy (especially mononeuritis multiplex) and unexplained gastrointestinal, cardiac or central nervous system ischemia. Immunomodulatory therapy, chiefly with systemic corticosteroid therapy has been successful.

AUTONOMIC DYSFUNCTION

Early signs of autonomic dysfunction include syncope and presyncope, diminished sweating, diarrhea, bladder dysfunction and impotence. Patients with HIV - associated nervous system disease have the greatest abnormalities in autonomic function.

DISORDERS OF RHYTHM

Sudden death and rhythm abnormalities are common in HIV infection and account for 20% of cardiac related death in this group of patients. These can be secondary to other cardiac pathology or be a consequence of some forms of treatment like pentamidine induced torsade de pointes ventricular tachycardia. HIV infection itself is associated with QT prolongation and torsades de pointes ventricular tachycardia. The incidence increases with progression to AIDS.

Hepatitis C is independently associated with increased QT duration and co infection with HIV nearly doubles the risk.56 Concomitant electrolyte abnormalities can be important in development of cardiac arrhythmia. 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 also been reported.

MATERIAL AND METHODS

Place of study- Department of Internal Medicine, Thanjavur Medical College and Hospital

Collaborating Departments- ART Centre, Department of Cardiology, Thanjavur Medical College and Hospital

Duration of the Study- Jan 2016 to June 2016

Type of study- Cross- sectional

STUDY POPULATION:

A total of 100 patients who were sero-positive and who fit the inclusion criteria were chosen. An attempt was made to find out whether the prevalence of cardiac abnormalities had any relation with the stage of infection.

INCLUSION CRITERIA:

All patients diagnosed to have HIV infection/ AIDS after ELISA test being positive were included in the study.

EXCLUSION CRITERIA:

1. Diabetes
2. Hypertension
3. Congenital/ acquired heart disease
4. Dyslipidemia

RISK FACTOR ASSESSMENT QUESTIONNAIRE

All HIV infected individuals who were included in this study were subjected to a questionnaire to assess the risks of acquiring HIV, risk factors for cardiac disease and symptomatology of cardiac illness.

To assess the risk of acquiring HIV, history regarding their sexual exposures, use of intravenous drugs and history of blood transfusion were 40 asked. The individual's occupation, marital status, extramarital and premarital sexual exposures, history of past and present sexually transmitted infections were also noted.The HAART regimen the individual is currently taking and the duration of treatment was also noted.

To assess the risk factors for cardiac disease, questions regarding duration and amount of smoking and alcohol consumption were asked.

To assess the symptoms of cardiac disease questions regarding presence of chest pain, breathlessness, palpitation, pedal edema and fatigue were asked. Duration of each symptom was also noted.

CLINICAL EXAMINATION:

All patients were meticulously examined for the presence of anemia, cyanosis, clubbing, pedal edema, dyspnea, jaundice, generalized lymphadenopathy and skin and mucous membrane lesions. Respiratory rate, pulse rate, jugular venous pressure, blood pressure (both in supine and erect posture) were also recorded. A thorough clinical examination of the cardiovascular system, respiratory system, abdomen and central nervous system was done.

LABORATORY INVESTIGATIONS

All of them were subjected to the following investigations.

Complete blood count, blood urea and sugar, serum creatinine and electrolytes, liver function tests (serum bilirubin, alanine transamin ase and alkaline phosphatase) and Serum lipid profile were done for all patients.

A standard 12 lead resting electrocardiogram was taken for all individuals in this study.

CD4 Count Assay

The standard method for enumerating CD4 T cells uses a flow cytometer. Computer calculates the number of CD4 T cells by analyzing the size of the cell and which of the antibodies it has been tagged with. The overall process is called Fluorescence Activated Cell Sorting (FACS).

IMAGING

Chest Skiagram

An erect X-ray of the chest on deep inspiration in the posteroanterior view was taken for all patients.

Echocardiography

Two dimensional Echocardiography was done for all patients included in this study in Department of Cardiology, Thanjavur Medical College Hospital, Thanjavur.

Chi square test was applied for significance. “P” value less than 0.05 was considered as significant.

REFERENCE VALUE USED IN THIS STUDY

BMI (WHO criteria for Asian population)

Body Mass Index = Weight (kg) / Height in meter 2

Values 18.5 - 22.9 kg / m2 was taken as normal weight

< 18.5 kg / m2 was taken as underweight

23- 29.9 kg / m2 was taken as overweight

≥ 30 kg / m2 was taken as obesity

RESULTS

1. A total of 100 patients were studied of which 8 patients were excluded from the study. Their stage of infection was noted and divide accordingly into four groups.
2. There were 13 HIV seropositive patients in Stage I ,27 patients in Stage II,33 patients in Stage III and 19 patients in Stage IV.
3. The mean CD4 count of study population was 500.07± 334.37 .

   (Stage I- 690.38±464.62

   Stage II-614.67±304.60

   Stage III-346.63±252.68

   Stage IV-516.06±292.62)
4. Out of 92 patients,58 (63%) were males (Stage I — 10 Stage II — 16 Stage III — 17 Stage IV — 15) and 34 (37%) were females (Stage I — 3 Stage II — 11 Stage III — 16 Stage IV — 4)
5. Mean age of study group was 43±11 (Stage I — 40±11 Stage II — 44 ±11 Stage III — 42±12 Stage IV —44±12)
6. Unskilled labourers and house wives constituted the majority of the study population , about 34.8% and 22.8% respectively
7. Heterosexual route was the most common mode of transmission of HIV infection about 96.7%.

8. Mean duration of HIV infection was 3.53 ± 2.30 years.

(Stage I — 3.31±2.25 years Stage II — 3.71±2.34 years Stage III — 3.46±2.43 years Stage IV —3.55±2.22 years)

9. Smokers and alcoholics constituted 20.6% (19 patients) and 48.91% (45 patients) of the study population respectively.

10. Mean BMI was 19.18±1.85 kg / m^2

(Stage I —19.22±2.3 kg / m^2

Stage II — 19.33±1.85 kg / m^2

Stage III — 19.12±1.55 kg / m^2

Stage IV —19.03±2.12 kg / m^2.

11. Most patients were asymptomatic. Cardiac symptoms were found in 6 patients(6.52%).

(Stage I —0

Stage II — 1

Stage III —2

Stage IV —3)

12. Cardiac abnormalities either in the form of Electrocardiography or Echocardiography abnormality was found in 30 patients(32.60%)

(Stage I —1

Stage II —7

Stage III —11

Stage IV -11)

13. Twenty patients (21.73%)had Electrocardiographic abnormalities

(Stage I —1

Stage II -3

Stage III —8

Stage IV —8)

14. Echocardiography abnormality was seen in 18 (19.56%) patients.

(Stage I —0

Stage II —4

Stage III —5

Stage IV —9)

15. Out of the 92 patients, 83 patients were on ZLN regimen and 9 patients were on TLE regimen.

16. Significant correlation was found between Stage of infection,CD4 count and duration of HIV infection with cardiac abnormalities.

17. There was no significant correlation between age, sex, BMI, cardiac symptoms, type of HAART regimen, smoking and alcohol with cardiac abnormalities

STAGE OF INFECTION:

TABLE 5

STAGE OF INFECTION	NO. OF PATIENTS	Percentage (100%)
I	13	14.1
II	27	29.3
III	33	35.9
IV	19	20.7

Stage I included 14.1% of patients

Stage II included 29.3 % of patients

Stage III and Stage IV included 35.9% and 20.7% of patients respectively.

CARDIAC ABNORMALITIES:

TABLE 6

CARDIAC ABNORMALITIES	NUMBER OF PATIENTS
PRESENT	30(32.60%)
ABSENT	62(67.40%)
TOTAL	92

Cardiac abnormalities either in the form of electrocardiography or echocardiography abnormality were found in 30 patients out of the total 92 patients.The prevalence of cardiac abnormalities was 32.60%.

CD 4 COUNTS:

TABLE 7

STAGE OF INFECTION	MEAN CD4 COUNT cells / mm3
I	690.38±464.62
II	614.67±304.60
III	346.63±252.68
IV	516.06±292.62

The mean CD4 count of study population was 500.07± 334.37.

STAGE OF INFECTION AND CARDIAC ABNORMALITIES:

TABLE 8:

STAGE OF INFECTION	CARDIAC ABNORMALITIES		TOTAL
	PRESENT	ABSENT	
I	1	12	13
II	7	20	27
III	11	22	33
IV	11	8	19
TOTAL	30	62	92

There was a statistically significant correlation observed between Stage of infection and cardiac abnormalities. Prevalence of cardiac abnormalities increased with stage of infection. P value was 0.02(<0.05)

AGE DISTRIBUTION IN RELATION TO CARDIAC

ABNORMALITIES AND STAGE OF INFECTION:

TABLE 9

STAGE OF INFECTION	MEAN AGE
I	40±11
II	44 ±11
III	42±12
IV	44±12

Mean age in study population was 43±11 years.

Mean age in Stage I was 40 ±11.

Mean age in Stage II was 44±11years.

Mean age in Stage III was 42± 12 YEARS.

Mean age in Stage IV was 44±12 YEARS

TABLE 10

AGE IN YEARS	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total
20-25	1 (7.7%)	0	1 (7.7%)	0	0	0	0	1 (3.0%)	1 (3.0%)	0	1 (5.2%)	1 (5.2%)
26-30	0	1 (7.7%)	1 (7.7%)	2 (7.4%)	1 (3.7%)	3 (11.1%)	4	3 (9.0%)	7 (21.2%)	2 (10.5%)	1 (5.2%)	3 (15.8%)
31-35	0	2 (15.4%)	2 (15.4%)	1 (3.7%)	3 (11.1%)	4 (14.8%)	1 (3.0%)	3 (9.0%)	4 (12.1%)	0	2 (10.5%)	2 (10.5%)
36-40	0	4 (30.8 %)	4 (30.8 %)	2 (7.4%)	4 (14.8%)	6 (22.2%)	1 (3.0%)	2 (6.0%)	3 (9.0%)	2 (10.5%)	0	2 (10.5%)
≥41	0	5 (38.5%)	5 (38.5%)	2 (7.4%)	12(44.4)	14 (51.9%)	5 (15.1%)	13 (13.4%)	18 (54.6%)	7 (21.2%)	4 (21.0%)	11 (57.9%)
TOTAL	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11 (33.3%)	22 (66.7%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

There was no statistically significant correlation noted between age and cardiac abnormalities ($P > 0.05$).

SEX DISTRIBUTION IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 11:

SEX	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total
MALE	1 (7.7 %)	9 (69.2%)	10 (76.9%)	6 (22.2%)	10 (37.0%)	16 (59.2%)	3 (9.0%)	14 (42.4	17 (51.5%)	8 (42.1%)	7 (36.8%)	15 (78.9%)
FEMALE	0	3 (23.0%)	3 (23.0%)	1 (3.7%)	10 (37.0%)	11 (40.7%)	8 (24.2%)	8 (24.2%)	16 (48.5%)	3 (15.8%)	1 (5.2%)	4 (21.0%)
TOTAL	1 (7.7 %)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11(33.3%)	22 (66.7%)	33 (100%)	11 (33.3%)	8 (42.1%)	19 (100%)

There was no statistically significant correla tion noted between sex and cardiac abnormalities($P > 0.05$)

OCCUPATION:

TABLE 12:

OCCUPATION	TOTAL
House wife	21(22.8%)
Lorry driver	4(4.3%)
Office worker	18(19.6%)
Sex worker	1(1.1%)
Skilled labourer	16(17.4%)
Unskilled labourer	32(34.8%)

ROUTE OF TRANSMISSION:

TABLE 13

ROUTE OF TRANSMISSION	TOTAL	Percentage (100%)
Heterosexual	89	96.7
Homosexual	2	2.2
IV Drug user	1	1.1
Bisexual	0	0
Blood transfusion	0	0

BMI IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 14:

STAGE OF INFECTION	MEAN BMI
I	19.22±2.3
II	19.33±1.85
III	19.12±1.55
IV	19.03±2.12

Mean BMI in study group was 19.18±1.84 kg / $m^{2.}$

Mean BMI in Stage I ,Stage II, Stage III and Stage IV was 19.22±2.3 kg / $m^{2.}$,19.33±1.85 kg / $m^{2.}$,19.12±1.55 kg / $m^{2.}$and 19.03±2.12 kg / m^{2} respectively

TABLE 15

BMI	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total	Present	Absent	Total
UNDER WEIGHT	0	6 (46.2%)	6 (46.2%)	2 (7.4%)	8 (29.6%)	10 (37.0%)	3 (9.0%)	7 (21.2%)	10 (30.3%)	5 (26.3%)	5 (26.3%)	10 (52.6%)
NORMAL	1 (7.7%)	5 (38.5%)	6 (46.2%)	4 (14.8%)	12 (44.4%)	16 (59.2%)	7 (21.1%)	15 (45.6%)	22 (66.7%)	6 (31.6%)	2 (15.8%)	8 (42.1%)
OVER WEIGHT	0	1 (7.7%)	1 (7.7%)	1 (3.7%)	0	1 (3.7%)	1 (9.0%)	0	1 (9.0%)	0	1 (5.2%)	1 (5.2%)
OBESE	0	0	0	0	0	0	0	0	0	0	0	0
TOTAL	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11(33.3)	22 (66.7%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

There was statistically no significant correlation noted between BMI and cardiac abnormalities ($P > 0.05$).

CD4 COUNT IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 16

CD4 COUNT cells/cu.mm	STAGE I			STAGE II(			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
< 200	0	1 (7.7%)	1 (7.7%)	1 (3.7%)	1 (3.7%)	2 (7.4%)	7 (21.3%)	6 (18.1%)	13 (39.3%)	4 (21.0%)	0	4 (21.4%)
>=200	1 (7.7%)	11 (84.6%)	12 (92.3%)	6 (22.2%)	19 (70.3%)	25 (92.5%)	4 (12.1)	16 (48.4%)	20 (60.6%)	7 (36.8%)	8 (42.1%)	15 (78.9%)
TOTAL	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11 (33.3%)	22 (66.7%)	33 (100%)	11 (58.9%)	8 (42.1%)	19 (100%)

P value for stage I was 0.76 — statistically not significant

P value for Stage II was 0.41 — statistically not significant

P value for Stage III was 0.04 — statistically significant

P value for Stage Iv was 0.04 — statistical significant

There was a statistically significant correlation between cd 4 count and cardiac abnormalities in Stage III and Stage IV in contrast to Stage I and Stage II

DURATION OF HIV INFECTION IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION

Table 17

STAGE OF INFECTION	MEAN DURATION
I	3.52±3.14
II	3.34±2.22
III	3.41±2.05
IV	3.66 ±2.36

Mean duration of HIV infection in study population was 3.53 ± 2.30 years.

Mean duration of HIV infection in Stage I was 3.31±2.25 years.

Mean duration of HIV infection in Stage II was 3.71±2.34 years.

Mean duration of HIV infection in Stage III was 3.46±2.43 years

Mean duration of HIV infection in Stage IV was 3.55±2.22 years

TABLE 18

DURATION OF HIV	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
< 1 year	0	2 (15.4%)	2 (15.4%)	0	3 (11.1%)	3 (11.1%)	1 (3.0%)	6 (18.1%)	7 (21.2%)	0	3 (15.8%)	3 (15.8%)
1- 3 years	0	5 (38.5%)	5 (38.5%)	0	7 (25.9%)	7 (25.9%)	2 (6.0%)	8 (24.2%)	10 (30.3%)	3 (15.8%)	3 (15.8%)	6 (31.6%)
>3 years	1 (7.7%)	5 (38.5%)	6 (46.1%)	7 (25.9%)	10 (37.0%)	17 (62.9%)	8 (24.2%)	8 (24.2%)	16 (48.5%)	8 (42.1%)	2 (10.5%)	10 (52.6%)
TOTAL	1 (7.7%)	12	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11	22 (66.7%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

P value for stage I was 0.53 — statistically not significant

P value for Stage II was 0.04— statistically significant

P value for Stage III was 0.01— statistically significant

P value for Stage Iv was 0.04 — statistical significant

There was a statistically significant correlation between duration of infection and cardiac abnormalities in Stage II, Stage III and Stage IV in contrast to Stage I

SMOKING IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 19

CARDIAC SYMPTOMS	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
PRESENT	0	0	0	0	1 (3.7%)	1 (3.7%)	0	2 (12.1%)	2 (12.1%)	2 (10.5%)	1 (5.2%)	3 (15.8%)
ABSENT	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	19 (70.3%)	26 (96.2%)	11 (33.3%)	20 (60.6%)	31 (93.9%)	9 (47%.3%)	7 (36.8%)	16 (84.2%)
TOTAL	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11 (33.3%)	20 (60.6%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

There was no statistically significant correlation noted between smoking and cardiac abnormalities(p value>0.05)

ALCOHOL IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 20

ALCOHOL	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
PRESENT	1 (7.7%)	7 (53.8%)	8 (61.5%)	6 (22.2%)	9 (33.3%)	15 (55.6%)	2 (6.0%)	9 (27.3%)	11 (33.3%)	7 (36.8%)	4 (21.0%)	11 (57.9%)
ABSENT	0	5 (38.4%)	5 (38.4%)	1 (3.7%)	11 (40.7%)	12 (44.4%)	9 (27.2)	13 (39.4%)	22 (66.7%)	4 (21.0%)	4 (21.0%)	8 (42.1%)
TOTAL	1 (7.7%)	12 (92.3%)	13 (100%)	7 (25.9%)	20 (74.0%)	27 (100%)	11 (33.3%)	22 (66.7%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

There was no statistically significant correlation noted between alcohol and cardiac abnormalities ($P > 0.05$).

CARDIAC SYMPTOMS IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

TABLE 21

ALCOHOL	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
PRESENT	1 (7.7%)	7 (53.8%)	8 (61.5%)	6 (22.2%)	9 (33.3%)	15 (55.6%)	2 (6.0%)	9 (27.2%)	11 (33.3%)	7 (36.8%)	4%) (21.0%)	11 (57.9%)
ABSENT	0	5 (38.5%)	5 (38.5%	1 (3.7%)	11 (40.7%)	12 (44.4%)	9 (27.2%)	13 (39.3%)	22 (66.7%)	4 (21.0%)	4 (21.0%)	8 (42.1%)
TOTAL	1 (7.7%)	12 (92.3%)	13 (100)	7 (25.9%)	20 (74.0%)	27 (100%)	11 (33.3%)	22 (66.7%)	33 (100%)	11 (57.9%)	8 (42.1%)	19 (100%)

There was no statistically significant correlation found between cardiac symptoms and cardiac abnormalities (p value > 0.05).

ANALYSIS OF ELECTROCARDIOGRAM:

TABLE 22

ECG Abnormalities	**STAGE I**	**STAGE II**	**STAGE III**	**STAGE IV**	**TOTAL**
Sinus tachycardia	1 (5%)	0	0	1 (5%)	2
Conduction abnormalities	0	1 (5%)	1 (5%)	0	2
Atrial ectopic	0	1 (5%)	1 (5%)	1 (5%)	3
Ventricular ectopic	0	0	1 (5%)	0	1
Poor progression of R wave	0	0	3 (15%)	2 (10%)	5
Low voltage	0	0	0	3 (15%)	3
ST/ T wave abnormality	0	1 (5%)	2 (10%)	1 (5%)	4
Total	**1**	**3**	**8**	**8**	**20**

Out of 92 patients,20 patients had ECG abnormalities.

ELECTROCARDIOGRAPHIC CHANGES IN RELATION TO STAGE OF INFECTION:

TABLE 23

ELECTRO CARDIOGRAPHY	STAGE I	STAGE II	STAGE III	STAGE IV	TOTAL
NORMAL	12	24	25	11	72
ABNORMAL	1	3	8	8	20
TOTAL	13	27	33	19	92

There was statistically significant correlation noted regarding electrocardiographic abnormalities (P value was 0.04) . Prevalence of electrocardiographic abnormalities increased with stage of infection.

ECHOCARDIOGRAPHIC CHANGES IN RELATION TO STAGE OF INFECTION:

Table 24

ECHOCARDIOGRAPHY	STAGE I	STAGE II	STAGE III	STAGE IV	TOTAL
Pericardial Effusion	0	0	0	1 (5.6%)	1
Dilated Cardiomyopathy	0	0	2 (11.1%)	1 (5.6%)	3
Hypokinesia and diasto;ic dysfunction	0	0	2 (11.1%)	2 (11.1%)	4
Infective Endocarditis	0	0	0	1 (5.6%)	1
diastolic dysfunction	0	2 (11.1%)	0	4 (22.2%)	8
PHT	0	0	1 (5.6%)	0	1
TOTAL	0	2	5	9	18

Out of 92 patients ,16 patients had echocardiographic findings

There was a statistically significant difference noted regarding echocardiographic abnormalities (P value was 0.00).Prevalence of echocardiographic abnormalities increased with stage of infection

TYPE OF HAART REGIMEN IN RELATION TO CARDIAC ABNORMALITIES AND STAGE OF INFECTION:

HAART REGIMEN	STAGE I			STAGE II			STAGE III			STAGE IV		
	CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES			CARDIAC ABNORMALITIES		
	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL	PRESENT	ABSENT	TOTAL
ZLN	0	13	13	7	20	27	8	19	27	11	6	17
TLE	0	0	0	0	0	0	3	3	6	0	2	2
TOTAL	0	13	13	7	20	27	11	21	33	11	8	19

Of the 92 patients,8 patients were on TLE regimen while the remaining 84 patients were on ZLN regimen.Of the 84 patients on ZLN 7 patients in Stage II,8 patients in Stage III and 11 patients in Stage IV had cardiac abnormalities.Three patients out of the 8 patients on TLE regimen had cardiac abnormalities either in the form of ECG or echocardiographic findings.P value was more than 0.05 and there was no statistically significant correlation noted between the type of regimen and cardiac abnormalities.

DISCUSSION

Cardiovascular manifestations of HIV infection have not attracted much attention in the Indian sub continent. This is partly because of the clinical picture of HIV infection still dominated by opportunistic infections and symptoms of breathlessness, fatigue and poor exercise intolerance are frequently ascribed to other conditions associated with HIV infection. With the greater access to Anti-retroviral medications more patients may live longer enough to present with end organ disorders. Our study throws light into various unsuspected cardiac abnormalities in various groups of HIV infected patients and its relationship to CD4 count.

A study conducted by Kannagai et al[57] at CMC Vellore in 2008 has shown that majority of the HIV infected individuals in South India with CD4 counts of 200 - 350 cells / mm^3 had higher viral load than that suggested by International AIDS Society.

AGE DISTRIBUTION

Our study population consisted of 100 patients. The mean age of our study group was Mean age in study population was 43±11 years. Mean age in Stage I, Stage II, Stage III and Stage IV was 40±11 years, 44 ±11 years, 42±12 and 44±12 respectively with the age group ranging from 22 to75

years.. In a study conducted by Joshi et al at Mumbai,[59] the age group ranged from 17 to 52 years with the mean age of 29.8 years.

Correlation between age, stage of infection,and cardiac abnormalities was attempted. In Stage I, only one patient had cardiac abnormality and presented in the 20 - 25 years age group.. In Stage II, out of 7 patients with cardiac abnormalities 0 patient was in 20 - 25 years age group,two patients each in 26 -30 years group 36 - 40years age group and ≥41age group and 1 patient in 31 - 35 years of age group . In Stage III, out of 11 patients with cardiac abnormalities 0 patient was in 20 - 25 years age group, 4 patients were 26 - 30 years age group, 1 patient was in 31 - 35 years age group, 1 patient was in 36 - 40 years age group and 5 patients were in more than 40 years of age group.In Stage IV, out of 7 patients with cardiac abnormalities 0 patient was in 20 - 25 years age group, 2 patients were 26 - 30 years age group, 0 patient was in 31 - 35 years age group,2 patients was in 36 - 40 years age group and 7 patients were in more than 40 years of age group.

P values of all the groups was > 0.05 which was statistically insignificant. There was no correlation between age and cardiac abnormalities in this study similar to the study conducted by Caggese et al.[60]

GENDER DISTRIBUTION

The gender distribution demonstrated a majority of 58 (63%) of males compared to 34 (37%) of females. In a study conducted by Joshi et al[59], male and female ratio was 5.7:1 (63 males and 11 females). In P Kannan et al[61] study males were 120 and females were 80. In El Hattoui et al[62] study males and females were 88 and 70 respectively.

Sex distribution in relation to cardiac abnormalities and stage of infection was seen. In Stage I, 10 males and 3 females had cardiac abnormalities. In Stage II, 16 males and 11 females had cardiac abnormalities.In Stage III, 17 males and 16 females had cardiac abnormalities.In Stage IV, 15 males and 4 females had cardiac abnormalities. Sex did not play a significant role in cardiac abnormalities in HIV patients.

OCCUPATION

In our study group, unskilled labourers and house wives constituted majority of the population about 34.8% and 22.8% respectively. Skilled labourers were 16(17.4%), 4.3% was lorry driver, 19.6% was office workers and 1.1% was commercial sex worker.

ROUTE OF TRANSMISSION

Most of the study population had heterosexual behaviour (96.7). Remaining were homosexual (2.2%), IV drug users (1.1%). In a study by Joshi et al,[59] among 74 patients 58.1% were heterosexuals, 4.05% blood transfusion related, 2.7% IV drug users, 1.35% bisexuals and 20.27% had multiple risk factors.

CD 4 COUNT

In our study, 21.73% of patients had CD4 count <200 and 78.26% of patients had CD 4 count >200. In Stage I one patient had cardiac abnormality and had CD4 count >200,in Stage II cardiac abnormalities were present in 1 patient with CD 4 count < 200 and 6 patients with CD 4 count > 200, in Stage III cardiac abnormalities were present in 7 patients with CD 4 count < 200 and 4 patients with CD 4 count > 200, in Stage IV patients with cardiac abnormalities 4 patients had CD 4 count < 200 and 7 patients had CD 4 count > 200 .

RISK FACTORS

In our study group, 20.65% were smokers and 48.91% were alcoholic.

In Stage I, 38.4% were smokers. In Stage II 11.11% were smokers. In Stage III,18.18% were smokers. In Stage IV 26.31% were smokers. P values for all the four stages were statistically insignificant.

In Stage I,61.53% were alcoholics. In Stage II,55.55% were alcoholics.In Stage III,33.33% were alcoholics.In Stage IV,57.89% were alcoholics.In a study by Smith CJ et al,[63] among 394 patients 45% were smokers and 7% were alcoholics. There was no significant correlation between smoking and alcohol with cardiac abnormalities in our study. This finding correlated well with the study by Caggese et al.[60]

DURATION OF HIV INFECTION

In this study 30.43% of patients had duration of illness between 1 - 3 years. 16.30% had less than a year and 53.26% had duration of > 3 years. The duration of illness ranged from 2 months to 8.75years. Mean duration of illness in study population was 3.53± 2.31 years.

Mean duration of illness in Stage I,Stage II,Stage III,Stage IV were 3.31±2.25 years , 3.71±2.34 years, 3.46±2.43 years and 3.55±2.22 years respectively. In a study by P Kannan et al,[61] the duration of illness ranged from 6 months to 7 years.

In Stage I, the duration was more than 3 years for the one patient who had cardiac abnormality . P value was 0.53, statistically insignificant. In Stage II, among 7 patients with cardiac abnormalities all the 7 patients had a duration of > 3 years. P value was 0.04, statistically significant. In Stage III, among 11 patients with cardiac abnormalities 8 patients had a duration of > 3 years, 2 patient had a duration of 1 -3 years and 1 patient had a duration of less than a year.In Stage IV, among 11 patients with cardiac abnormalities 8 patients had a duration of > 3 years, and 3 patients had a duration of 1 -3 years.There was a significant correlation between duration of HIV infection and cardiac abnormalities in Stage II ,III and IV.

SYMPTOMS

Most patients were asymptomatic. Only 6 patients had cardiac symptoms (Stage I - 0, Stage II - 1, Stage III - 2 ,Stage IV-3). Among 26 patients with cardiac abnormalities 6 patients had symptoms. The symptoms were dyspnea (10.7%),palpitation (1.3%) and chest pain (0.7%).

There was no significant correlation was found between cardiac abnormalities and cardiac symptoms. P values for all the stages were >0.05. In a study by Cardosa JS et al, [64] 7.3% (10 / 137) of patients were symptomatic. In a study by Ewig S et al, [65] nine out of 14 patients (64%) with cardiac abnormalities had symptoms.

BMI

The mean BMI of our study group was 19.18±1.84 kg / m^2. The mean BMI of Stage I,Stage II,Stage III, and Stage IV were 19.22±2.3 kg /m^2, 19.33±1.85 kg / m^2.,19.12±1.55 kg / m^2.and 19.03±2.12 respectively.

In Stage I, one patient who had cardiac abnormality had normal BMI.. In Stage II, out of 7 patients with cardiac abnormalities, 4 patients had normal BMI,two patients were underweight and one patient was overweight. In Stage III, out of 11 patients with cardiac abnormalities, 7 patients had normal BMI,three patients were underweight and one patient was overweight.In Stage IV, out of 7 patients with cardiac abnormalities,4 patients had normal BMI and three patients were underweight. P values for all the stages were more than 0.05 and statistically insignificant. There was no significant correlation between BMI and cardiac abnormalities.

ELECTROCARDIOGRAPHIC ABNORMALITIES

Electrocardiographic abnormalities were seen in 20 patients (21.73%). Twelve Patients had ECG abnormalities without echocardiographic abnormalities. Ten patients had normal ECG inspite of echocardiographic abnormality. In Stage I,one patient had ecg abnormality. In Stage II, 3 patients had ECG abnormalities.In Stage III and IV, 8 patients had cardiac abnormalities respectively. The ECG abnormalities observed were poor progression of R wave (25%), ST-T changes (20%), atrial ectopic (15%),low voltage complexes(15%),conduction abnormality (10%), sinus tachycardia (10%), and ventricular ectopic (5%).There was a significant correlation between CD4 count and ECG abnormalities (P value was 0.04). In a study by Herdy GV et al, [67] out of 50 patients 18 patients had sinus tachycardia, 10 patients had ST-T changes, 5 patients had low voltage complexes, 5 patients had ST segment elevation and 3 patients had extra systole. In Mirri A et al [68] study, ECG abnormalities unrelated to echocardiographic abnormalities or clinical problems were seen in 11 patients. In Joshi et al [59] study, among 74 patients, 20.27% had ECG abnormalities.

ECHOCARDIOGRAPHIC ABNORMALITIES

Prevalence of cardiac abnormalities by echocardiography in our study was 19.56%. (22.22% in Stage II,27.77% in Stage III and 50% in Stage IV) Echocardiographic findings were diastolic dysfunction (44.44%),hypokinesia and diastolic dysfunction(22.22%) , dilated cardiomyopathy (16.66%), pericardial effusion (5.55%),pulmonary hypertension(5.55%)and infective endocarditis (5.55%). P value was 0.000, statistically significant.

There was a significant correlation CD4 count and echocardiographic abnormalities. In a study by Joshi et al,[59] among 74 patients 10.6% had dilated cardiomyopathy, 8.5% had pericardial effusion, 4.2% had vegetations, 2.1% had constrictive pericarditis and 10.6% had incidental valvular, left ventricular hypertrophy, ischemic heart disease. In a study by Mishra et al[69] at AIIMS, 36.7% had diastolic dysfunction and 23.3% had systolic dysfunction. In P Kannan et al[61] study, out of 200 patients, 28 patients had left ventricular dysfunction, 20 patients had pericardial effusion, 6 patients had pulmonary hypertension and one patient had dilated cardiomyopathy. In Mirri A et al[68] study, 17% had echocardiographic abnormalities.

CARDIAC ABNORMALITIES

In our study among 100 patients, 30 patients (32.60%) had cardiac abnormalities either in the form of ECG or Echocardiography abnormality. It is observed that 1 patient out of 13 patients (7.69%) in Stage I ,7 patients out of 27 (25.92%) patients in Stage II, 11 patients out of 33 (33.33%) patients in Stage III and 11 patients out of 27 (40.74%) patients in Stage IV had cardiac abnormalities. There was a statistically significant correlation between cardiac abnormalities and CD4 count (P value was 0.02). As the stage of infection increases, the cardiac abnormalities increase proportionally. Cardiac abnormalities are directly proportional to the Stage of infection.

SUMMARY

The present study aimed at estimating the prevalence of cardiac abnormalities in HIV seropositive patients and also to find out its correlation with stage of infection. With rigid criteria 100 HIV seropositive cases were selected. There were 58 males and 34 females in the study group.

Prevalence of cardiac abnormalities was 32.60% in our study. Diastolic dysfunction was the most common echocardiographic abnormality. Poor progression of R waves was the most common electrocardiographic abnormality.

Cardiac abnormalities were specifically correlated with Stage of infection. Present study recommends screening for cardiac abnormalities in HIV patients to identify early cardiac involvement and minimize cardiac complications by early intervention.

People with cardiac abnormalities did not necessarily have cardiac symptoms and the frequency of cardiac symptoms did not correlate with the stage of infection in this study. Cardiac symptoms in HIV infected individuals are likely to be attributed to other concurrent illnesses and the cardiac abnormalities remain undiagnosed further contriburting to the morbidity and mortality of HIv patients.

Cardiac abnormalities correlated with the increase in the duration of infection and the decline in CD 4 count.

CONCLUSION

1. The determination of Incidence and Prevalence of cardiac abnormalities in HIV infected individuals using non invasive tests is quite feasible and should be done in all patients registering in ART centre.

2. There was a direct correlation between stage of infection and cardiac abnormalities.

3. There was a significant correlation between duration of HIV infection and cardiac abnormalities. Prevalence of cardiac abnormalities was found to be more with increase in the duration of HIV infection.

4. Most of the patients were asymptomatic.Correlation was not found between cardiac symptoms and cardiac abnormalities.

5. Heterosexual route was the most common route of transmission of HIV.

6. There was no significant correlation between age, sex, BMI, Type of HAART regimen,smoking and alcohol with cardiac abnormalities**.**

7. The patients included in this study were on ART and correlation was not found between the type of HAART regimen and the cardiac abnormalities. Majority of patients were on ZLN regimen and the remaining were on TLE regimen.

LIMITATIONS

1. The mean duration of the disease in our patients was less. This could be responsible for decreased incidence of cardiac abnormalities in our patients.
2. Follow up study was not done. So the incidence of cardiac abnormalities in patients with previous normal echocardiography as well as the natural history of those who had Cardiac abnormality could not be studied.
3. Since the critically ill patients were not included in our study the entire spectrum of cardiac abnormalities could not be established.
5. Viral load could not be estimated due to constraints.
6. Histopathologic studies like pericardial biopsy, endomyocardial biopsy and cytological study of pericardial fluid to determine the etiology of pericardial effusion and cardiomyopathy were not done.

RECOMMENDATIONS

1. A baseline Echocardiographic study for all patients with HIV infection at first visit to be done.

2. Echocardiographic follow up should be done to determine the evaluation of cardiac abnormalities and their reversibility with or without treatment.

3. Histopathologic studies like pericardial biopsy, endomyocardial biopsy and cytological study of pericardial fluid to be done to determine the etiology of pericardial effusion and cardiomyopathy.

4. Autopsy studies should be performed in patients dying of HIV associated illness and this will throw light on exact incidence of cardiac abnormalities in HIV infected patients.

ABBREVIATION

AIDS - Acquired Immuno Deficiency Syndrome ANC - Antenatal clinic

ART - Anti Retro Viral Therapy

ATT - Anti Tuberculous Therapy

BMI - Body Mass Index

CD - Cluster Differentiation

CNS - Central Nervous System

CVS - Cardiovascular system

DM - Diabetes Mellitus

DNA - Deoxyribonucleic acid

ECG - Electrocardiography

Echo - Echocardiography

FSW - Female Sex Worker

HAART - Highly Active Anti Retro Viral Therapy HIV - Human

Immunodeficiency Virus

HT - Hypertension

IDU - Intravenous Drug Users

JVP - Jugular venous pressure

LV - Left Ventricle

MSM - Men having sex with men

NACO - National AIDS Control Organization

NACP - National AIDS Control Programme

NHL - Non Hodgkin's Lymphoma OH - Orthostatic Hypotension RNA - Ribonucleic acid

RS - Respiratory system

STD - Sexually Transmitted Disease WHO - World Health Organization Kg - Kilo gram

µL - Micro Litre

mm^3 - Cubic millimetre m^2 - Metre square

> - More than

≥ - More than or Equal

≤ - Less than or Equal

< - Less than

% - Percentage

± - Plus or minus & - And

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PROFORMA

Name	Hospital No:	Serial No:
Age		
Sex	Height	WHO Stage:
Occupation	Weight	Education :
Address	BMI	

Marital status

Sexual exposure

Other risk factors

Partners HIV status

Duration of HIV infection

Age at first sexual exposure

Recent extra marital exposure

Previous STD's

Present STD's

Smoking

Alcohol

DM

HT

Previous cardiac illness

H/O ATT

PRESENTING COMPLAINTS

Chest pain
Breathlessness
Palpitation
Pedal edema
Fatigue
Postural giddiness

EXAMINATION

Pallor
Jaundice
Cyanosis
Clubbing
Pedal edema
Lymphadenopathy

BP: Supine - Standing -
Pulse rate Respiratory Rate Temperature
JVP
Opportunistic infections

CVS
RS
ABDOMEN
CNS

INVESTIGATIONS

HEMOGRAM CD4 count -

Hb%
Total count
Differential count P - , L - ,E - ,M - ,B - .
ESR
PCV
Platelet count

RENAL FUNCTION TEST

Blood urea
Blood sugar
Serum creatinine
Serum electrolytes

LIPID PROFILE

Total Cholesterol
Triglycerides
HDL
LDL
VLDL

LIVER FUNCTION TEST

Serum Bilirubin
SGOT
SGPT
Sr. Alkaline Phosphatase
Total Protein
Albumin
Globulin

ECG

Chest X ray

Echocardiography

NAME	AGE & SEX	OCCUPATION	OPPORTUNISTIC INFECTION	MODE OF TRANSMISSION	BMI	SMOKER	ALCOHOLIC	DURATION	HAART REGIMEN	CD4 COUNT - /cumm	STAGE OF INFECTION	CARDIAC S	ECG	ECHO
Sekar	48/M	Unskilled labourer	PTB	Heterosexual	18.19	Yes	Yes	0.66	TLE	202	IV	Yes	Normal	Normal
Sivnandha	35/M	Unskilled labourer	NO	Heterosexual	19.34	No	Yes	1	ZLN	236	IV	No	Normal	Normal
Arokyanan	49/M	Lorry driver	PTB	Heterosexual	24	No	No	2.5	ZLN	402	IV	No	Normal	Normal
Gunasekar	51/M	Skilled labourer	No	Heterosexual	17.2	No	No	1.25	ZLN	1073	II	No	Normal	Normal
Vasuki	30/F	House wife	No	Heterosexual	23.42	No	No	2	TLE	200	III	No	Normal	Hypokinesia & Diastolic dysfunc
Palanivel	30/M	Office worker	No	Heterosexual	16.84	No	Yes	3.08	ZLN	479	II	No	Normal	Diastolic dysfunction
Vijaya	40/F	House wife	No	Heterosexual	24.4	No	No	4.08	ZLN	224	II	No	Atrial ectopic	Normal
Isak	50/M	Unskilled labourer	No	Heterosexual	21.86	Yes	Yes	8.25	ZLN	450	III	No	Normal	Normal
Elakkiya	28/F	Sex worker	No	Heterosexual	22	No	No	6.33	ZLN	694	IV	No	ST,T wave changes	Dilated cardimyopathy
Kannadasa	40/M	Unskilled labourer	No	Heterosexual	20.46	No	Yes	5.16	ZLN	124	II	No	Normal	Diastolic Dysfunction
Chinnatha	60/F	House wife	No	Heterosexual	16.75	No	No	2	ZLN	189	IV	Yes	Normal	Pericardial effusion
Selvi	42/F	Office worker	NO	Heterosexual	19.45	No	No	0.41	ZLN	159	III	No	Normal	mild PHT
Ramesh	47/M	Lorry driver	Herpes zoster	IV Drug user	20.23	No	Yes	0.75	ZLN	424	IV	No	Normal	Normal
Rengasamy	33/M	Unskilled labourer	No	Heterosexual	26.2	No	No	1.33	ZLN	381	I	No	Normal	Normal
Ezhilmathi	40/F	Office worker	No	Heterosexual	17.43	No	No	3.25	ZLN	396	II	No	Normal	Normal
Sumathy	31/F	Office worker	No	Heterosexual	19.76	No	No	4	ZLN	479	II	No	Normal	Normal
Jeeva	40/F	House wife	No	Heterosexual	22.76	No	No	2	ZLN	170	IV	Yes	Low voltage complexes	Infective endocarditis
Chitra	29/F	Skilled labourer	No	Heterosexual	20.2	No	No	3.16	ZLN	189	II	No	Normal	Normal
Indra	49/F	House wife	oral candidiasis	Heterosexual	18.34	No	No	1	ZLN	620	II	No	Normal	Normal
Rosammal	70/F	House wife	PTB	Heterosexual	17.11	No	No	4.41	TLE	278	III	No	Poor progrssion of R wave	Hypokinesia & Diastolic dysfunc
Ammakani	33/F	House wife	Herpes zoster	Heterosexual	18.54	No	No	1	ZLN	1311	II	No	Normal	Normal
Vasantha	45/F	Office worker	No	Heterosexual	20.87	No	No	4.08	ZLN	709	II	No	Normal	Normal
Radhakrish	59/M	Office worker	No	Heterosexual	16.77	Yes	Yes	3.25	ZLN	804	IV	No	Normal	Diastolic dysfunction
Premalath	46/F	House wife	oral candidiasis	Heterosexual	19.88	No	No	3	ZLN	466	II	No	Normal	Normal
Nadesan	63/M	Unskilled labourer	No	Heterosexual	18.37	No	Yes	5.16	ZLN	425	II	No	Normal	Normal
Selvi	39/F	Office worker	No	Heterosexual	17.95	No	No	0.16	ZLN	390	III	No	Normal	Normal
Chinnatha	38/M	Unskilled labourer	No	Heterosexual	18.72	No	No	2.41	ZLN	252	I	No	Normal	Normal
Segappi	40/F	House wife	No	Heterosexual	21.53	No	No	6.25	ZLN	120	III	No	ST,T wave changes	Normal
Krishnaver	32/F	Unskilled labourer	No	Heterosexual	16.83	No	No	5.16	ZLN	345	III	No	Normal	Normal
Govindasa	44/M	Unskilled labourer	No	Heterosexual	18.37	Yes	Yes	5.25	ZLN	166	IV	No	Low voltage complexes	Diastolic dysfunction
Krishnasar	35/M	office worker	No	Heterosexual	22.44	No	Yes	0.58	ZLN	940	II	No	Normal	Normal
Shanthi	32/F	skilled labourer	No	Heterosexual	18.1	No	No	3.58	ZLN	1091	I	No	Normal	Normal
Bangaru	44/M	unskilled labourer	No	Heterosexual	19.6	No	Yes	5.08	ZLN	259	III	No	Normal	NORMAL
Venmathi	30/F	office worker	No	Heterosexual	16.53	No	No	6.75	ZLN	552	III	No	Normal	Normal
Dhanapal	34/M	unskilled labourer	No	Heterosexual	19.65	Yes	Yes	4	ZLN	778	II	No	Normal	Diastolic dysfunction
Anbumani	40/M	unskilled labourer	PTB	Heterosexual	18.98	No	Yes	0.75	TLE	285	III	No	Normal	Normal
Saraswati	40/F	house wife	No	Heterosexual	19.29	No	No	7.16	ZLN	1100	I	No	Normal	Normal
Mani	38/M	Skilled labourer	No	Heterosexual	17.9	No	Yes	5	ZLN	445	IV	No	Poor progression of R wave	Diastolic Dysfunction
Mala	40/F	house wife	Herpes zoster	Heterosexual	21.78	No	No	1.25	ZLN	254	II	Yes	Normal	Normal
Raja	28/M	Office worker	No	Heterosexual	17.53	No	Yes	6	ZLN	965	II	No	RBBB	Normal
Mary	30/F	House wife	No	Heterosexual	16.99	No	No	0.91	ZLN	623	III	No	Normal	Normal
Saralmary	45/F	Unskilled labourer	oral candidiasis	Heterosexual	20.31	No	No	4.75	ZLN	521	III	No	Normal	Normal
Rajendren	48/M	Unskilled labourer	No	Heterosexual	20.55	Yes	Yes	2.5	ZLN	947	III	No	Normal	Normal
Pitchai	58/M	Lorry driver	No	Heterosexual	19.24	No	Yes	3	ZLN	120	IV	No	Poor progression of R wave	Normal
Perumal	56/M	Skilled labourer	No	Heterosexual	19.71	Yes	No	6.16	ZLN	458	III	No	Normal	Normal
Sarala	36/F	House wife	No	Heterosexual	18.56	No	No	0.66	ZLN	987	II	No	Normal	Normal
Manikanda	42/M	Unskilled labourer	PTB	Heterosexual	19.77	No	Yes	3.33	ZLN	759	III	No	Normal	Normal
Rajesh	50/M	Unskilled labourer	No	Heterosexual	20.31	Yes	Yes	5.58	ZLN	954	IV	No	Normal	Diastolic Dysfunction
Megala	45/F	House wife	No	Heterosexual	19.69	No	No	2	ZLN	854	III	No	Normal	Normal
Saranya	28/F	House wife	No	Heterosexual	20.06	No	No	6.16	ZLN	114	III	No	LBBB	Normal
Veerasamy	75/M	Unskilled labourer	No	Heterosexual	21.6	No	Yes	8.75	ZLN	741	II	No	Normal	Normal
Aravind	45/M	Skilled labourer	oral candidiasis	Heterosexual	18.24	No	Yes	4.33	ZLN	254	I	No	Normal	Normal
Ayyapan	46/M	Skilled labourer	No	Heterosexual	19.52	Yes	No	0.41	ZLN	102	III	No	Normal	Normal
Munusamy	58/M	Unskilled labourer	Herpes zoster	Heterosexual	16.82	No	Yes	7.91	TLE	354	III	No	Normal	Normal
Vendhan	45/M	Unskilled labourer	No	Heterosexual	19.82	No	Yes	8.75	ZLN	258	II	No	Normal	Normal
Santhanan	28/M	Unskilled labourer	No	Homosexual	17.62	No	Yes	2.25	ZLN	325	I	No	Normal	Normal
Arokiasam	40/M	Skilled labourer	No	Heterosexual	19.5	Yes	Yes	4.25	ZLN	654	II	No	Normal	Normal
Muniyamm	62/F	House wife	No	Heterosexual	19.72	No	No	7.25	ZLN	159	III	No	Poor progression of R wave	Normal
Latha	45/F	House wife	PTB	Heterosexual	19.65	No	No	2	ZLN	257	III	No	Ventricular ectopic	Normal
Malar	29/F	House wife	No	Heterosexual	18.72	No	No	0.25	ZLN	759	III	No	Normal	Normal
Balasubrar	56/M	Skilled labourer	No	Heterosexual	19.05	No	No	3	ZLN	145	III	No	Normal	Normal
Amutha	52/F	House wife	No	Heterosexual	18.28	No	No	3	ZLN	857	III	No	Normal	Normal
Dhandapar	56/M	Lorry driver	No	Heterosexual	17.53	No	Yes	6.58	ZLN	965	II	No	Normal	Dilated cardiomyopathy
Mohan	22/M	Skilled labourer	Herpes zoster	Heterosexual	20.63	Yes	Yes	4.33	TLE	1452	I	No	Sinus tachycardia	Normal
Shankar	36/M	Skilled labourer	No	Heterosexual	18.48	No	Yes	1	ZLN	125	I	No	Normal	Normal
Muthulaks	55/F	House wife	No	Heterosexual	19.57	No	No	0.42	ZLN	985	I	No	Normal	Normal
Chinnapon	56/F	Unskilled labourer	No	Heterosexual	19.04	No	No	2.83	ZLN	745	II	No	Normal	Normal
Umarani	35/F	House wife	PTB	Heterosexual	17.38	No	No	6.25	ZLN	321	IV	No	Normal	Normal
Seetha	29/F	Unskilled labourer	No	Heterosexual	19.29	No	No	3.91	TLE	154	III	No	Poor progression of R wave	Dilated cardiomyopathy
Kumaresar	64/M	Unskilled labourer	No	Heterosexual	18.2	Yes	Yes	6.83	ZLN	652	IV	No	Low voltage complexes	Hypokinesia & Diastolic dysfunc
Ponnusamy	44/M	Unskilled labourer	No	Heterosexual	18.35	No	No	0.5	ZLN	147	III	No	Normal	Normal
Sivakumar	48/M	Office worker	PTB	Heterosexual	19.5	No	Yes	5.58	ZLN	841	IV	No	Atrial ectopic	Normal
Abdul Lati	22/M	Office worker	PTB	Heterosexual	18.66	No	Yes	1.83	TLE	64	III	Yes	Normal	Normal
Peter	46/M	Skilled labourer	NO	Heterosexual	16.78	Yes	Yes	3	ZLN	255	I	No	Normal	Normal
Gopinath	23/M	Office worker	No	Heterosexual	19.64	No	No	0.25	ZLN	987	IV	No	Normal	Normal
Prabakara	55/M	Unskilled labourer	No	Homosexual	19.57	No	Yes	2	ZLN	485	II	No	Normal	Normal
Ganesh	41/M	Unskilled labourer	No	Heterosexual	18.24	No	Yes	4	ZLN	652	II	No	Normal	Normal
Anand	33/M	Skilled labourer	No	Heterosexual	17.3	Yes	No	2	ZLN	145	III	No	Normal	Normal
Kalyana su	26/M	Office worker	Herpes zoster	Heterosexual	19.29	No	Yes	3.75	ZLN	254	III	No	Atrial ectopic	Normal
Arivumani	55/M	Office worker	No	Heterosexual	18.3	Yes	Yes	6.33	ZLN	1256	I	No	Normal	Normal
Paulraj	36/M	Unskilled labourer	PTB	Heterosexual	18.54	Yes	Yes	0.66	ZLN	1021	I	No	Normal	Normal
Arumugam	52/M	Unskilled labourer	No	Heterosexual	18.64	No	No	1.25	ZLN	144	III	Yes	Normal	Normal
Valluvan	32/M	Unskilled labourer	No	Heterosexual	20.61	No	Yes	2.25	ZLN	285	III	No	Normal	Normal
Karthikeya	55/M	Skilled labourer	No	Heterosexual	19.45	Yes	Yes	6.16	ZLN	478	I	No	Normal	Normal
Aasaitham	29/M	Unskilled labourer	No	Heterosexual	16.24	No	Yes	5.75	TLE	285	IV	No	Normal	Normal
Senthil ku	48/M	Unskilled labourer	No	Heterosexual	16.87	No	No	1.33	ZLN	547	IV	No	Normal	Normal
Harish	54/M	Office worker	No	Heterosexual	18.49	Yes	Yes	0.42	ZLN	874	II	No	Normal	Normal
Dhinakara	44/M	Skilled labourer	No	Heterosexual	16.05	No	Yes	6.75	ZLN	345	II	No	ST,T wave changes	Normal
Ponnilavar	57/M	Skilled labourer	No	Heterosexual	19.82	No	Yes	5.25	ZLN	458	II	No	Normal	Normal
Arulkumar	26/M	Office worker	PTB	Heterosexual	17.93	No	No	4.25	ZLN	658	IV	No	Sinus tachycardia	Hypokinesia & Diastolic dysfunc
Vijayan	31/M	Office worker	No	Heterosexual	19.72	Yes	Yes	3.66	ZLN	154	III	No	Normal	Diastolic Dysfunction
John	47/M	Unskilled labourer	No	Heterosexual	17.24	No	Yes	6.5	ZLN	145	III	No	ST,T wave changes	Normal
					19.18097									