ANALYSIS OF ECHOCARDIOGRAPHIC FINDINGS IN HIV POSITIVE PATIENTS WITH CARDIAC SYMPTOMS

Submitted to

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY,

CHENNAI, INDIA

Dissertation submitted in partial fulfillment

of requirements for

M.D. DEGREE IN GENERAL MEDICINE

BRANCH - 1

STANLEY MEDICAL COLLEGE

CHENNAI - 600 001.

APRIL - 2014

CERTIFICATE

This is to certify that **Dr. S.DHANRAJ,** Post -Graduate Student **(MAY 2011 TO APRIL 2014)** in the Department of General Medicine **STANLEY MEDICAL COLLEGE,** Chennai- 600 001, has done this dissertation on **"ANALYSIS OF ECHOCARDIOGRAPHIC FINDINGS IN HIV POSITIVE PATIENTS WITH CARDIAC SYMPTOMS AT GOVERNMENT STANLEY HOSPITAL, CHENNAI – 600 001".** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2014.

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DECLARATION

I solemnly declare that this dissertation entitled **"ANALYSIS OF ECHOCARDIOGRAPHIC FINDINGS IN HIV POSITIVE PATIENTS WITH CARDIAC SYMPTOMS"** was done by me at Stanley Medical College & Government Stanley Hospital, Chennai, during February to July 2013 under the guidance and supervision of **Prof.G.Sundaramurthy, M.D**. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This dissertation is submitted to the Tamil Nadu Dr. M. G. R. Medical University towards the partial fulfillment of the rules and regulation for the M. D. Degree examination in General Medicine.

Place: Chennai **Dr.DHANRAJ**

Date:

ACKNOWLEDGEMENT

At the outset I thank **Prof. S.GEETHALAKSHMI M.D.,** Dean, Stanley Medical College and Government Stanley Hospital, Chennai for permitting me to use hospital data for the study.

I would like to express my sincere gratitude to my beloved Professor and Director, Institute of Internal Medicine, **Prof. P.VIJAYARAGHAVAN, M.D**, for his guidance and encouragement for my study.

I am extremely grateful to my chief **Prof.G.SUNDARAMURTHY M.D.,** Professor of Internal Medicine, Stanley Medical College and Government Stanley Hospital, Chennai-3 for his support and encouragement throughout the study.

I would also like to thank **Dr. Baby Jhansi, Medical officer,** ART center, Stanley Medical College and Government Stanley Hospital, Chennai for her Support and guidance throughout the study.

I wish to thank **Dr.S.ASHOKKUMAR,M.D.,** and **DR.ILAVARASI MANIMEGALI, M.D.,** Assistant Professors of my unit, Department of Medicine, Stanley medical college Hospital for their valuable suggestions, encouragement and advice.

Sincerely thank the members of Institutional Ethical Committee, Stanley Medical College for approving my dissertation topic.

I thank all my Colleagues, House Surgeons, and Staff nurses and other para medical workers for their support.

My sincere thanks to all the patients who participated in this study.

DR.S.DHANRAJ

CONTENTS

ANALYSIS OF ECHOCARDIOGRAPHIC FINDINGS IN HIV POSITIVE PATIENTS WITH CARDIAC SYMPTOMS

KEY WORDS:

- ***HIV,PERICARDIAL EFFUSION***
- ***MYOCARDITIS,PULMONARY HYPERTENSION***

INSTITUTION: Govt. Stanley Medical College & Hospital, Chennai

BACKGROUND: Cardiac abnormalities are common in **HIV** positive patients. There is less documentation about its presentation.

AIM OF THE STUDY: To describe the pattern of cardiac abnormalities in **HIV** patients.

PATIENTS & METHODS: This was a hospital-based cross-sectional observational study. HIV positive patients, HIV patients on HARRT without cardiac, diabetic, hypertensive, renal and respiratory disease were selected for the study. They were subjected to routine investigations and then for Electrocardiogram, Chest x ray and Echocardiogram.
The results were analyzed and presented here.

ECHO Diagnosis	Males	Percentage	Females	Percentage
CAD	3	3	0	0
Cor pulmonale	5	5	1	5
Myocarditis	9	9	1	5
Pericardial effusion	4	4	2	10
Systolic Dysfunction	4	4	2	10
Diastolic Dysfunction	4	4	2	10
Normal	66	69	13	62
TOTAL	**95**	**100**	**21**	**100**

- 32% of the patients were confirmed with cardiac disease.
- 45.5% of the patients had CD4 count less than 200.
- 91% were on Anti Retroviral Therapy.
- 53.5% of the patients were in the WHO HIV clinical stage 3.
- Most common echocardiography diagnosis was myocarditis, pericardial effusion, systolic dysfunction and diastolic dysfunction each at 7% respectively.
- Smoking status correlated with HIV and coronary artery disease
- Low CD4 count correlated with HIV and myocarditis.

INTRODUCTION

Acquired immunodeficiency syndrome is caused by human immunodeficiency virus which belongs to class of retroviridiae. AIDS has become the most dangerous pandemic which has plagued us in the last two decades. AIDS affects almost all systems in our body.

Due to increased discovery of new anti retroviral drugs against HIV the survival of the patients has considerably increased and hence the cardiovascular complications of AIDS is of more prevalent now[1].

The cardiovascular complications of HIV can be directly due to the virus itself, due to the associated risk factors like smoking and hyperlipidemias associated in HIV patients or due to the adverse effect the antiretroviral therapy.

Earlier heart diseases in HIV patients were mostly found in autopsy series. But with development of new diagnostic methods it is now possible to diagnose the cardiovascular complications of HIV in patients at a earlier time.

Of the diagnostic modalities for early detection of cardiovascular complications a simple but still a very effective tool is cardiac echocardiography.

The spectrum of cardiovascular conditions which can be diagnosed using imaging modalities include the following which are discussed in detail in later stage[2,3]

1. Pericarditis
2. Pericardial effusion
3. Endocarditis
4. Myocarditis
5. AIDS related malignancy like Kaposi sarcoma and lymphomas
6. Pulmonary hypertension
7. Cardiomyopathy
8. Coronary vascular diseases
9. Thrombosis, embolism, vasculitis and aneurysms.

Thus its now important to screen regularly for cardiac complications in HIV patients to Initiate early treatment and thus further improving the survival rate and increase the quality of life of HIV patients[4].

OBJECTIVE

1. TO STUDY PATTERN OF CARDIAC INVOLVEMENT IN HIV PATIENTS
2. TO KNOW INCIDENCE OF VARIOUS CARDIAC DISEASES IN OUR POPULATION
3. TO COMPARE RESULTS WITH AVAILABLE LITERATURE

MATERIALS AND METHODS

- **PLACE OF STUDY:**

  DEPARMENT OF MEDICINE, ART CLINIC,

  STANLEY MEDICAL COLLEGE HOSPITAL

- **DURATION**

  FEBRUARY 2013 TO JULY 2013

- **STUDY DESIGN**

  CROSS SECTIONAL STUDY

- **PATIENT SELECTION:**

  HIV POSITIVE PATIENTS

  HIV PATIENTS ON HAART.

- **EXCLUSION CRITERIA:**

1. KNOWN CORONARY ARTERY DISEASE PATIENTS AND OTHER KNOWN HEART DISEASE PATIENTS WITH HIV,
2. KNOWN HYPERTENSIVE AND DIABETIC PATIENTS
3. KNOWN COPD PATIENTS
4. RENAL FAILURE

- SAMPLE SIZE

  117 patients are studied .

METHODOLOGY

Patients from ART CLINIC, Medical ward in Stanley hospital from February to July 2013 who are positive for HIV antibodies were included in the study.

After getting informed consent from the patient, history and clinical examination was obtained from the patient followed by investigations.

Apart from all routine investigations like complete blood count, renal function test, lipid profile, liver function test, ultrasonogram of abdomen, chest x ray ,electrocardiogram,

Echocardiography was done for all these patients and analysis was done from the information from all the above.

REVIEW OF LITERATURE

From the time HIV was recognised as a potential global health problem with the capacity to transform into a major epidemic affecting millions of people globally spreading through all countries.

It was recognised that it could affect the heart and cause abnormalities in the heart. In the early times this was found by doing autopsy. The occurrence of heart abnormalities in HIV patients earlier was thought to be less.

Later with the advent of new scientific discoveries and development in imaging and diagnosing modalities it became more evident that HIV affects the heart.

And with the advent of echocardiography it became more apparent that HIV affected the heart more often and more early than it was earlier believed to be.

In most of the patients the cardiac complications due to HIV is not apparent and the patient might not have symptoms and clinical and physical examination of the patient may not yield significant results but it might cause complications like pericardial effusion and myocarditis which might have a grave consequence and might even result in death.

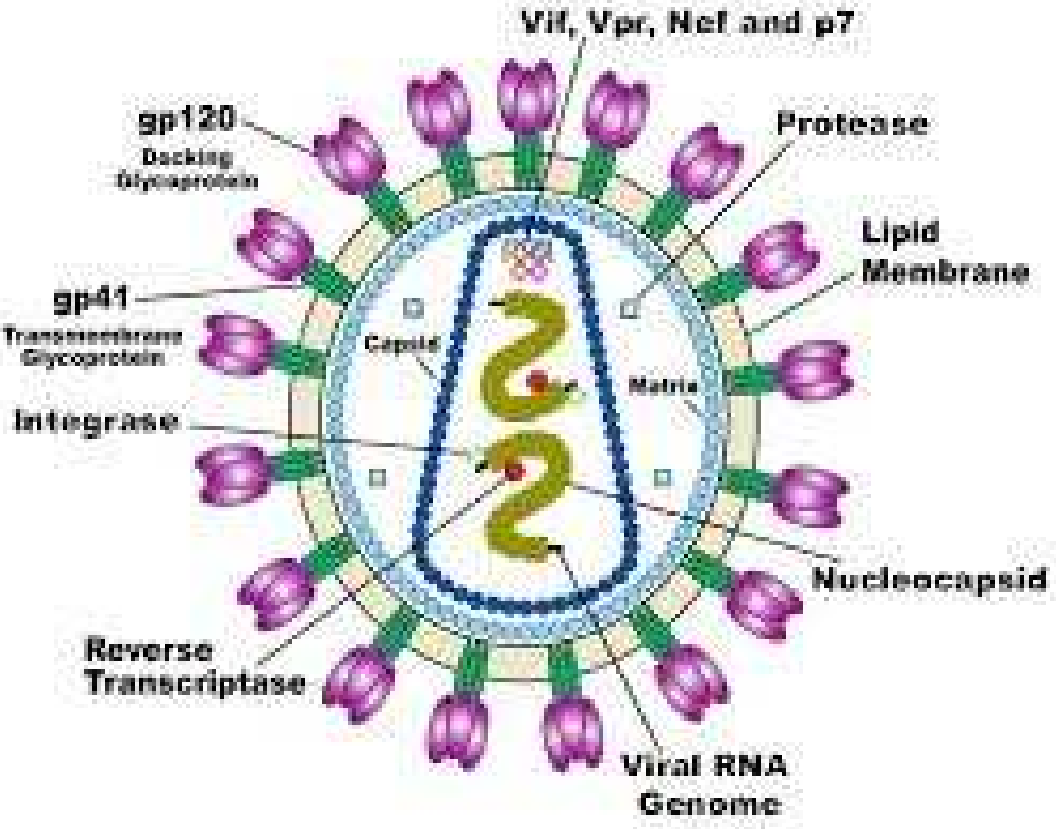
The common cardiac complications due to HIV are pericardial effusion and myocarditis and less common complications are cardiomyopathy, endocarditis and coronary artery disease[5,6].

With the advent of anti retroviral therapy and increased awareness about HIV, treated patients were having much longer life than before. Since due to this enhanced longevity chronic cardiac ailments which affect normal population affects HIV patient too.

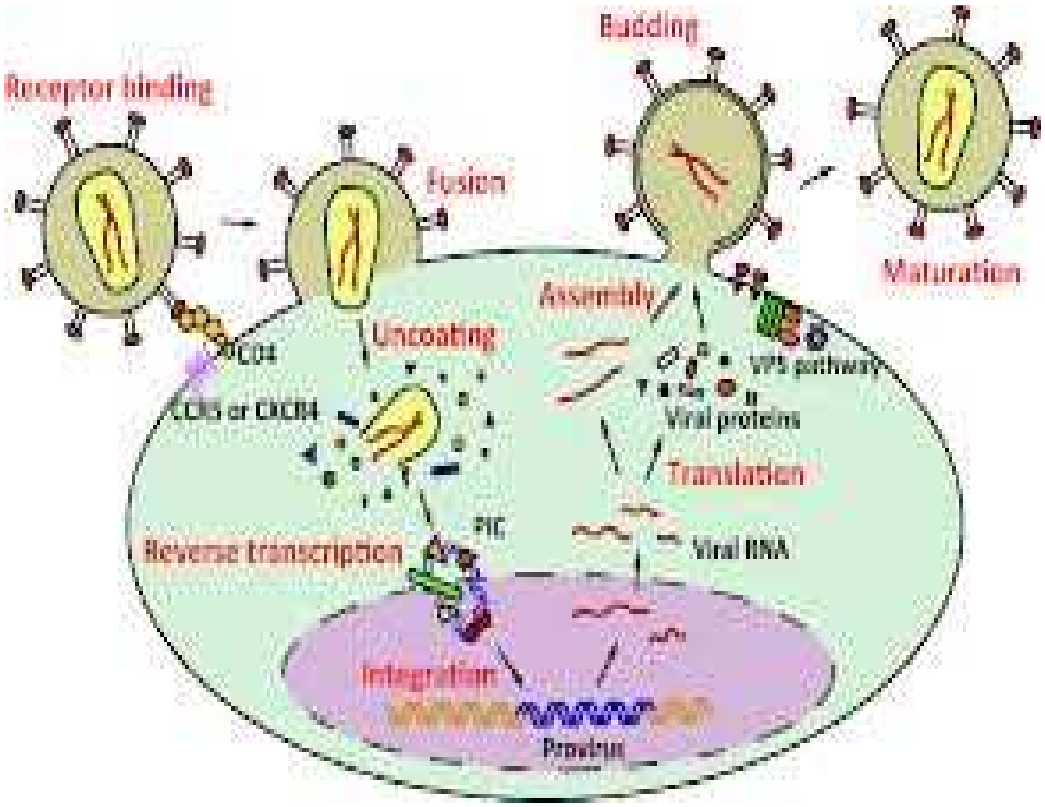
Hence there is increasing incidence of coronary heart disease among HIV patients.

As a physician it is our duty to be aware of the cardiac complications of HIV in our patients and anticipate them and take adequate measures to prevent development of risk factors, prevent progression of disease and finally to treat the cardiac ailment and try to prevent the complications and reducing the morbidity and mortality due to the disease[7,8,9].

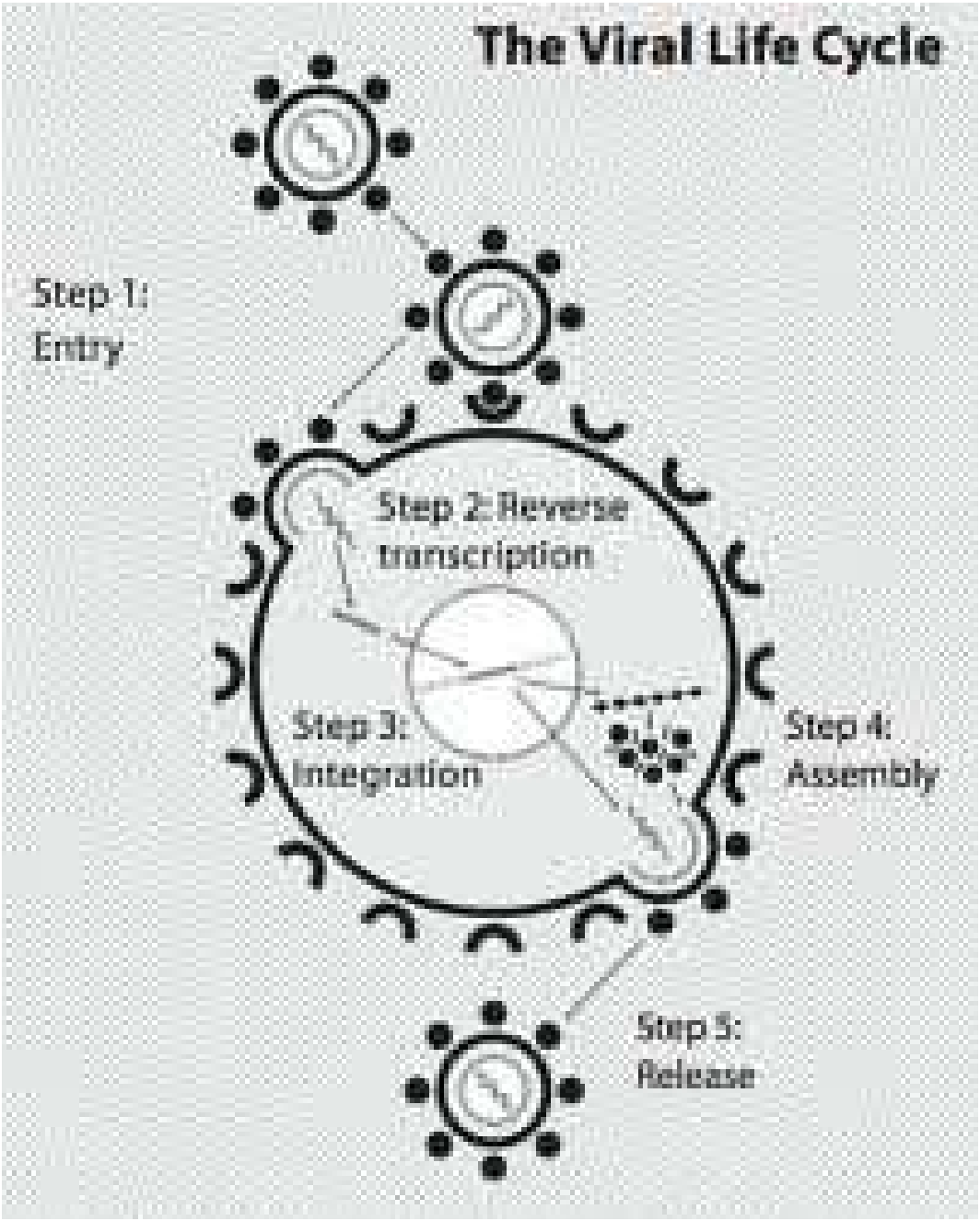
STRUCTURE OF HUMAN IMMUNODEFICIENCY VIRUS



LIFE CYCLE OF HUMAN IMMUNODEFICIENCY VIRUS



STAGES OF REPLICATION



RISK FACTORS FOR CARDIAC DISEASE IN HIV PATIENTS

1) DYSLIPIDEMIA

Often HIV infected patients suffer from dyslipidemias.

Their LDL , total cholesterol and triglycerides are often high and the level of HDL which is a good cholesterol serving as a scavenger of bad cholesterol is often low.

More over HIV patients average life span has increased considerably in recent times due to advances in medical science.

As a result of this HIV patients are increased risk of plaque formation in the coronaries and their chance of having a cardiac event is more high now than ever.

2) SMOKING

About two third of the HIV infected patients are found to be smokers. Smoking is an independent risk factor for coronary vascular event.

Smoking synchronises the risk of coronary vascular event with HIV infection.

3) CHRONIC INFLAMMATION

Its now believed that in HIV the arteries and veins are involved and are in a state of chronic inflammation.

As a result there is increased chance of plaque formation in the inflamed arteries and increased chance of myocardial vascular insufficiency.

4) DIABETES

Diabetic patients often suffer from dyslipidemias and macrovascular involvement in diabetes is often the major cause of silent myocardial infarction. Hence HIV patients with diabetes have an increased risk of adverse cardiac events.

5) CHRONIC KIDNEY DISEASE

HIV often affects the kidneys and is one of the important cause of focal segmental glomerulosclerois. Damage to the kidney might have an adverse effect on the blood pressure which in turn will affect the heart by increasing the afterload putting more strain on the heart. And when the kidney function deteriorates a volume overload state occurs increasing the preload thereby compounding the strain on the heart

PERICARDITIS AND PERICARDIAL EFFUSION

Pericardial effusion has an incidence rate of eleven percent in the time before the anti retroviral therapy was in use.

Pericardial effusion is one of the most common cardiac complications in HIV infected patients. Prevalence of pericardial effusion is higher among HIV patients when compared with that of non HIV patients[10,11].

Stage of HIV correlate with severity of effusion.

Most of effusions detected are smaller effusions and most of them are asymptomatic.

When HIV reaches end stage malignancies are found to be associated with pericardial effusions and also infections[13].

Pericarditis may lead to pericardial effusion which may result in cardiac tamponade causing diminished heart sounds , raised jugular venous pressure and hypotension.

It is so common that patients with pericardial effusion should be screened for HIV infection.

If pericardial effusion occurs in HIV patients it indicates grave prognosis and the chances of survival is very dim irrespective of the CD4 count.

The median age of survival of HIV patient with pericardial effusion was found to be 6 months.

However it is still unclear whether HIV is the direct cause of pericardial effusion.[14,15]

ETIOLOGY

In most cases the cause of pericardial effusion couldn't be ascertained. But many organisms have been found to be associated with pericardial effusion in HIV infected patients. Many of them are co infection and many are due to opportunistic organism which predominantly affect immune compromised individuals. They are

- Mycobacterium tuberculosis
- Mycobacterium avium complex
- Nocardia asteroids
- Chlamydia
- Staph aureus
- Listeria monocytogenes

- Rhodococcus
- Coxsackie
- Ebstein barr
- Herpes simplex
- Adenovirus
- Cytomegalovirus
- Toxoplasma gondii
- Histoplasma
- Cryptococcus

Other than infections, Neoplastic etiology includes, neoplasms like Kaposi sarcoma, lymphoma. Non infectious etiology includes, malnutrition, acute myocardial infarction, cirrhosis, uraemia, hypothyroidism connective tissue diseases, drugs, toxins, trauma and radiation are also found to be associated with the development of pericardial effusion in HIV patients[16.]

CLINICAL FEATURES

In many patients pericardial effusion might remain quiescent without any signs and symptoms. The earliest signs may be cough , chest pain and increase in temperature.

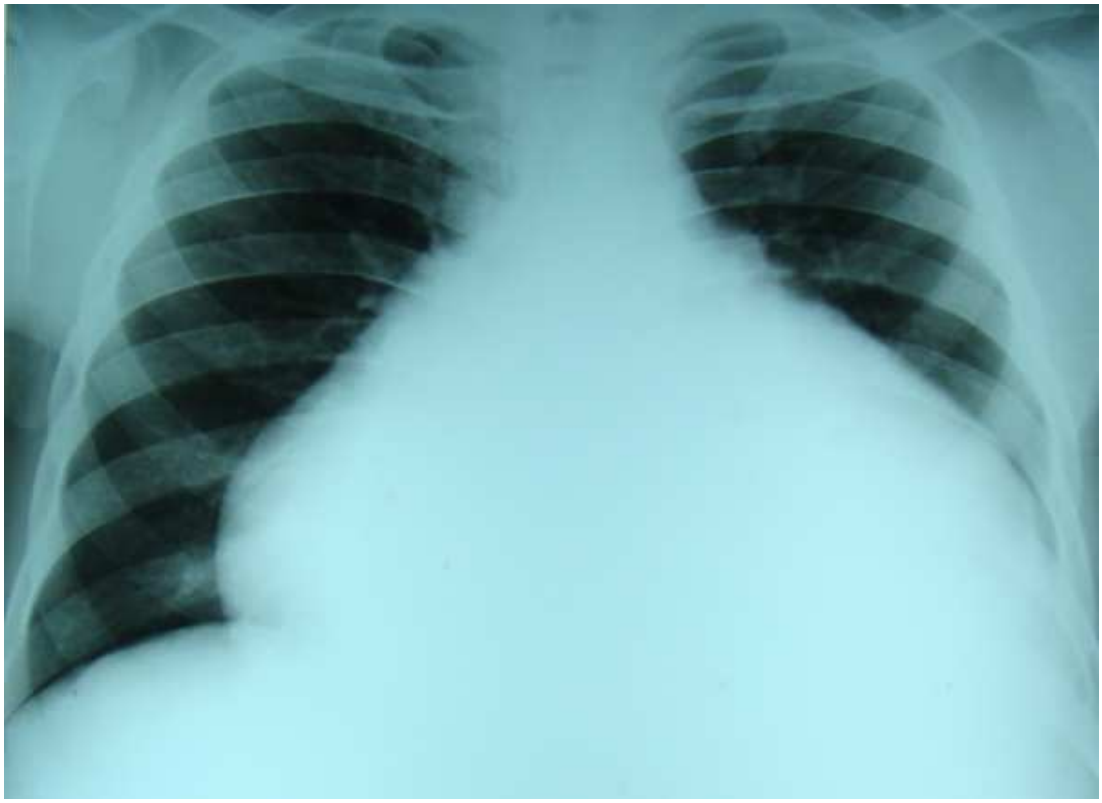
However in some patients the course may be severe and it might present with severe hypotension , shock and cardiac arrest.

Sometime even at lower volume some HIV patients may go for shock which seen in thin cachectic patients .Mechanism behind this 'low volume tamponade' is when the patient is severely dehydrated , there is severe volume loss and hence there is reduced right ventricular inflow and hence reduced filling pressure of right ventricle, in such a picture even mild pericardial effusion can precipitate tamponade[17,18].

In some patients there is spontaneous resorption of pericardial fluid. However the prognosis in HIV patients who develop pericardial effusion with or without symptom is poor with increased mortality within 6 months.

Thus pericardial effusion is a marker for advanced disease and poor prognosis in HIV patients[19].

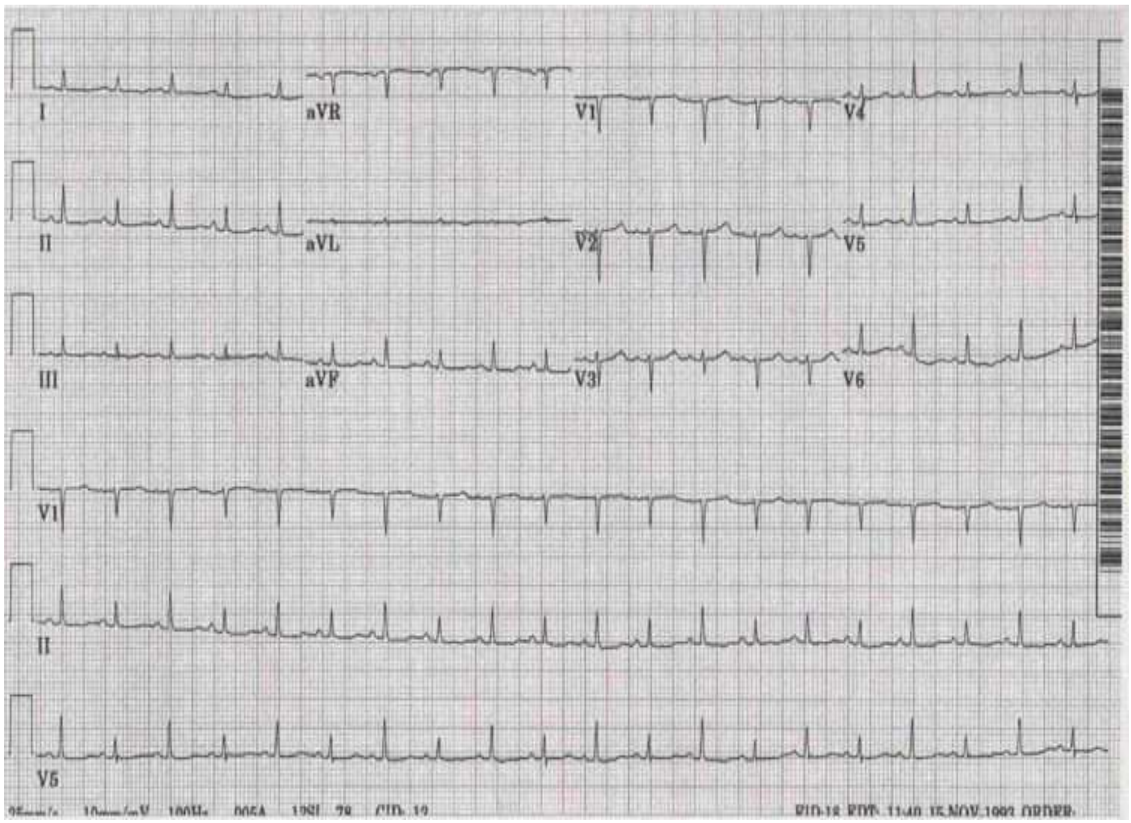
Below is chest x ray of pericardial effusion(gross enlargement of cardiac borders to pericardial effusion)



DIAGNOSIS

Electrocardiography shows low voltage complexes, electrical alternans, T wave inversion and p wave changes,

Shown below was ECG of pericardial effusion showing electrical alternans and low voltage complexes



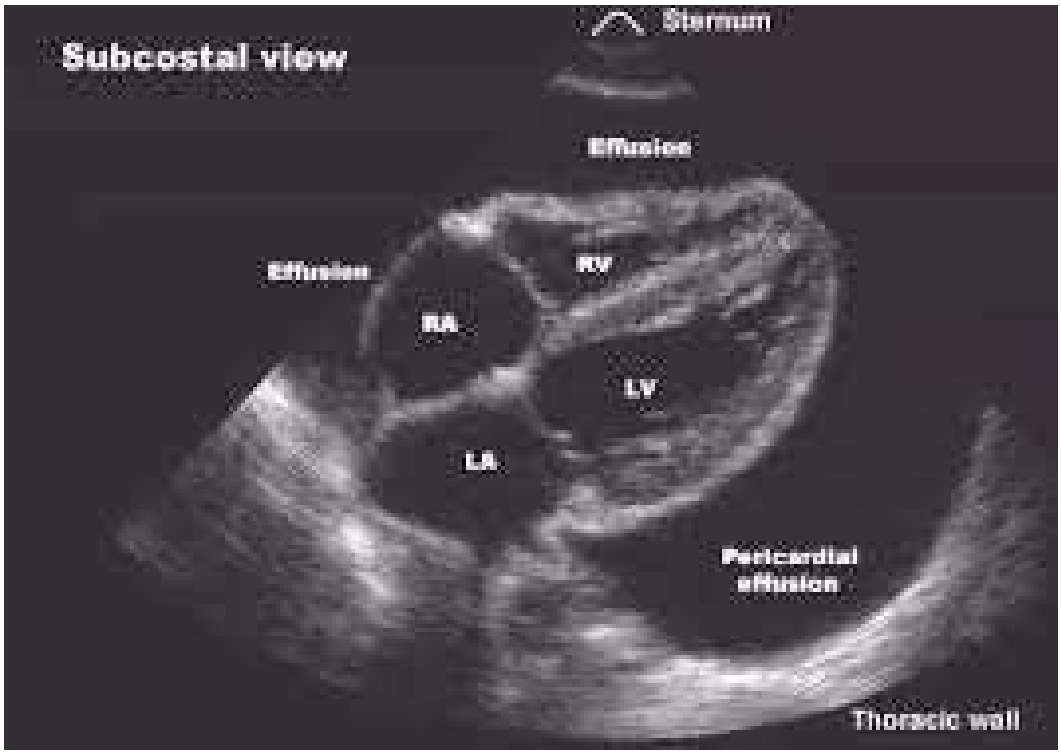
Echocardiography is the most easy and reliable way to detect pericardial effusion.

Pericardial effusion is diagnosed when the space between parietal and visceral pericardium gets persisted throughout the cardiac cycle during recording.

The characteristic findings found in echo for moderate to severe effusions are compression of the right atrium and the collapse of the right ventricle during diastole.

In order to find the etiology, pericardiocentesis can be performed and the sample can be sent for culture , polymerase chain reaction test, immuno fluorescent test and other tests to find out the etiological agent[20].

Below is echo image of pericardial effusion



MANAGEMENT

Institution of highly active anti retroviral therapy results in reducing the viral load and reducing the immuno compromised level thereby decreasing the chance of occurrence of opportunistic infections and

thus has been found to delay the onset of pericardial effusion in HIV infected patients.

NSAID's can be given which provides some relief.

Initial evaluation always centers around to see for all causes other than HIV

Which includes etiologies mentioned above.

Attention should be paid especially to curable conditions like tuberculosis, also every attempt should be made should be made to find etiological diagnosis.

Pericardiocentesis can done as diagnostic procedure and pericardial fluid can be sent for analysis, that includes biochemical,microbiological and pathological analysis.

For those patients who come in state of pericardial tamponade showing signs of

“Becks Triad”(distant heart sounds,distended neck veins,low blood pressure”

patient should be hospitalised and urgent pericardiocentesis should be done.

Pericardiectomy can be done for patients with recurrent effusions as in the case of neoplastic and other chronic pericarditis[21-24].

ENDOCARDITIS

Endocarditis is inflammation of lining of heart valves which is often caused by infection**.**

People who develop the condition have already existing heart problems and are over 50 years old, but can occur at any age, including children.

While not common, this can be a serious disease. Men are twice likely to be affected as women.

TYPES:

There are two types of endocarditis: *infective* & *non-infective*.

With effective treatment, the majority of people with infective endocarditis will survive. Non-infective endocarditis is difficult to treat.

Both thrombotic endocarditis due to non bacterial cause as well as infective endocarditis has been reported in HIV infected patients.

HIV patients who are intravenous drug abusers are at highest risk of developing endocarditis. Right sided valves are mostly involved in HIV patients who are drug abusers. The mortality is more in patients with advanced disease[25].

CAUSES OF ENDOCARDITIS

The most common organism is staphylococcus aureus followed by streptococcus viridans. The other organisms causing endocarditis are

- Staphylococcus epidermis,
- Haemophilus influenza,
- Streptococcus pneumonia
- HACEK group of organisms,
- Salmonella,
- Mycobacterium avium complex,
- Methicillin resistant staphylococcus aures
- Fungi like candida, aspergillus, Cryptococcus.

Following conditions elevate the risk both in HIV and non HIV patients:

- A history of rheumatic fever/rheumatic heart disease
- A congenital heart defect
- Prosthetic heart valves
- History of intravenous drug use
- Mitral valve prolapse (MVP)
- Diabetes
- Pregnancy

- Endocarditis develops in endocardium, the inner tissue of heart**.** It starts if this tissue has been damaged, injured, or infected.Just as a cut on the skin causes a scab , damage to the endocardium leads to formation of blood and tissue clot (*thrombus*).

In infective endocarditis, the clots may be caused by HIV infection, inflaming and damaging the heart cells.

SYMPTOMS

The symptoms of endocarditis can be similar to routine infective endocarditis picture and infact it is more and may include:

- high temperature (fever) above 38C (101.4F)
- chills
- headache,
- joint pain and
- muscle pain
- A new or changed heart murmur - not normal heart sounds made by blood rushing through heart,
  - Fatigue, Joint and muscle aches,
  - Night sweats,
  - Shortness of breath ,
  - Paleness, Persistent cough,

- Swelling of feet, legs or abdomen,
- Unexplained weight loss,
- Blood in urine (either visible or found in a doctor's viewing of your urine under a microscope)(hematuria),

In patients with HIV wasting syndrome, marantic endocarditis can occur which mainly affects left sided valves and it consist of friable vegetations composed of platelets, inflammatory cells encompassed within a mesh made up of fibrin.

Systemic embolization from this vegetation is a rare cause of death in HIV patients.

TESTS AND DIAGNOSIS :

Blood Tests:

Blood culture to identify bacteria in blood. Blood tests helps doctor identify conditions, including anaemia.

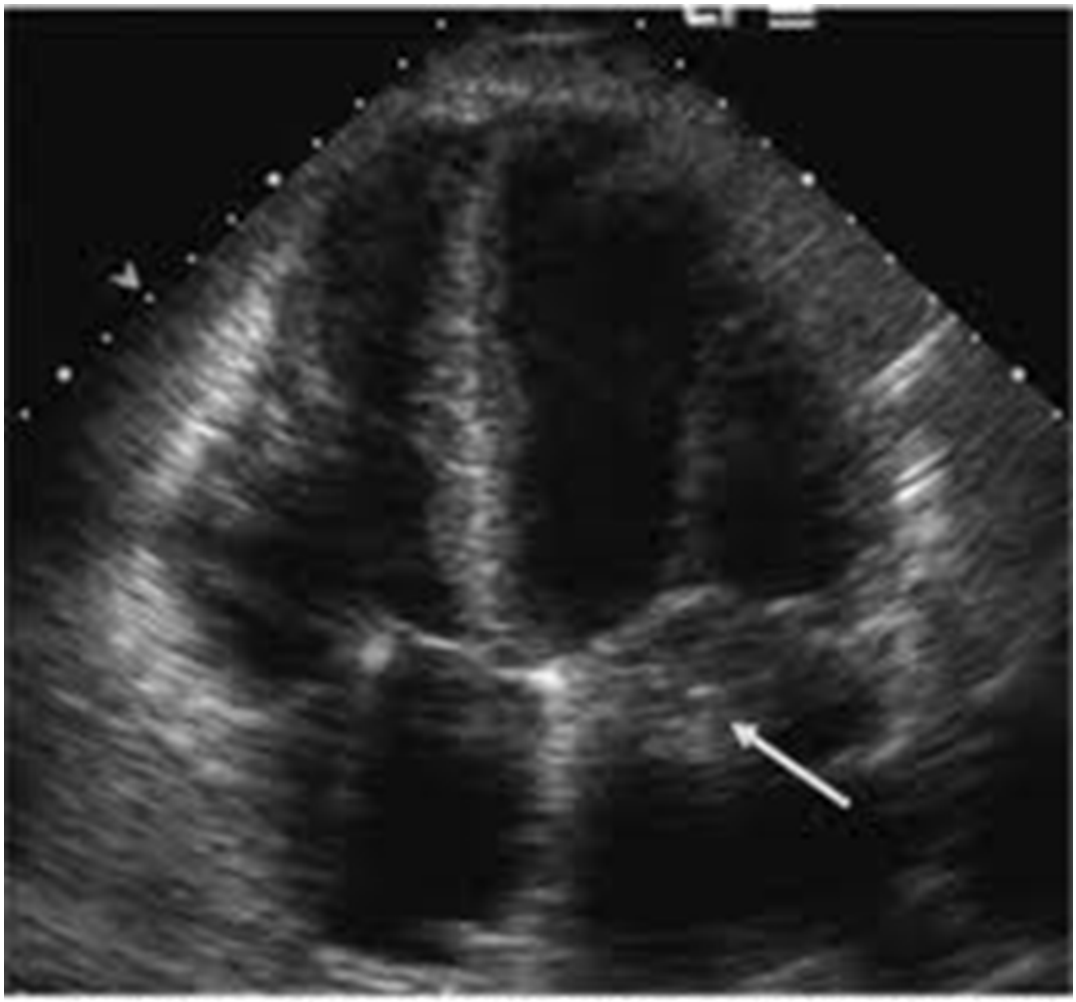
Transesophageal echocardiogram:

An echocardiogram use sound waves to produce image of heart at work. The echocardiogram allow doctor to get closer look at valves.

Computerized tomography (CT) scan and magnetic resonance imaging (MRI):

There may be a need for CT or MRI of brain, chest and other parts of your body if doctor thinks the infection has spread [26].

Vegetation on the atrial side of anterior leaflet of mitral valve due to candida in hiv positive patient



Vegetation on the atrial side of the anterior leaflet of the native mitral valve annulus (arrow).

TREATMENT

Apart from ART drugs antibiotics have to be added in the treatment of HIV associated endocarditis.

There is a need for high doses of intravenous antibiotics in hospital. Blood cultures help to identify type of microbes that is infecting the heart.

This information will help to choose the best antibiotic or combination to fight the infection.

Surgery

If infection damages heart valves, we may have symptoms and complications years after treatment.

Occasionally surgery is required to treat persistent infections or replace a damaged valve.

Surgery is sometimes needed to treat endocarditis that is caused by fungal infection.

MYOCARDIAL INVOLVEMENT

Myocarditis is defined as inflammation of myocardium having wide variety of presentations.HIV virus is been found out to be one of the prominent causes of dilated cardiomyopathy.

Pathogenesis of myocarditis in HIV shares multiple mechanics

Damage occurs through the following ways:

- Direct effects of causative agent
- Secondary immune response,which is triggered by causative agent
- Cytokine expression of myocardium (tumor necrosis factor [TNF]- alpha, nitric oxide synthase)
- Aberrant induction of apoptosis,

Myocardial damage consists of 2 main phases:

- Acute phase (first 2 wk)

Myocyte destruction as a direct consequence of offending agent.

- Chronic phase (>2 wk)

Continued myocyte destruction is of autoimmune nature, with associated different expression of human leukocyte antigen (HLA) in the myocytes.

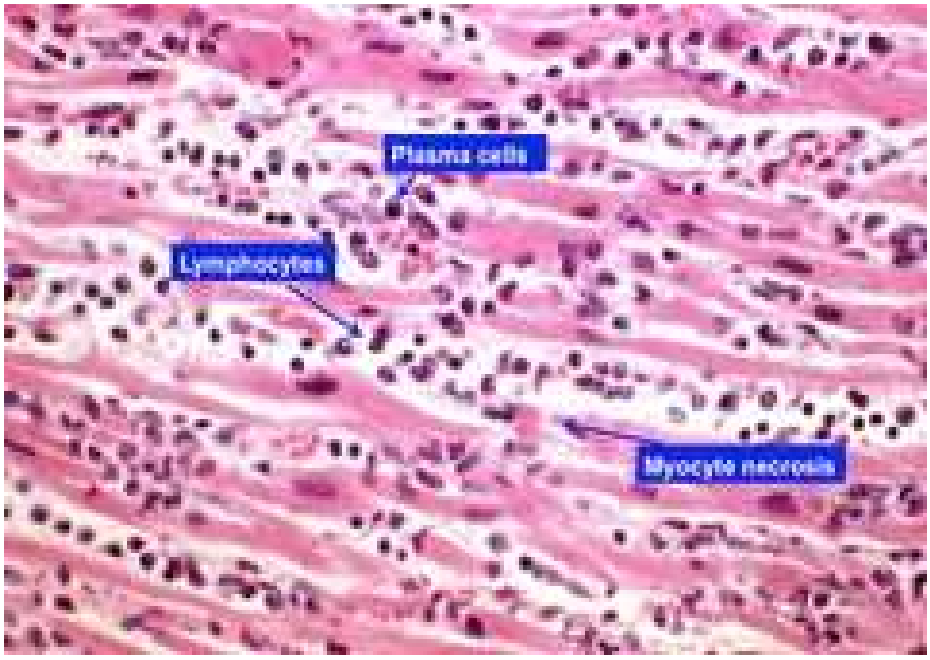
Histological section of HIV :

Consisted of multifocal or interstitial inclusions of small lymphocytes and isolated myocyte necrosis.

The lymphocytes that are seen includes T cells (CD2+, CD3+) and some cells that are not identified by the usual markers. Rarely B cells, monocytes, CD4+ cells, and natural killer (NK) cells were seen in the section.

The prognosis in patients who have dilated cardiomyopathy due to HIV is very poor[26-30].

HISTOLOGICAL APPEARANCE



Moreover the use of zidovudine has been found to be associated with mitochondrial myopathy.

Studies have shown that zidovudine causes destruction of ultrastructures of the mitochondria with inhibition of replication of mitochondrial DNA.

Patients with myocarditis can have a history of acute decompensation of heart failure, but may or may not have other cardiac dysfunction or have decreased cardiac risk.

The diagnosis is presumptive, relies on patient demographics and clinical course.

Patients present with mild symptoms of angina (concurrent pericarditis), fever, sweating, chills, and difficulty in breathing.

In viral myocarditis, patients present with history of recent (within 1-2 wk) flu like syndrome of fever, arthralgias , and malaise and pharyngitis, tonsillitis, or upper respiratory tract disease.

Patients with myocarditis present with, signs and symptoms of decompensation or heart failure (eg,tachycardia, gallop, mitral regurgitation, swelling of legs) and, in those with concomitant pericarditis, with pericardial friction rub.

When myocarditis is severe, it permanently damages heart muscle, this may result in

- **Heart failure:**

If left untreated, myocarditis damages heart's muscle to a point it can no longer pump blood effectively, causing heart failure.

- **Heart attack or stroke:**

If heart's muscle is injured and cannot pump blood, the blood that pools in heart forms clots. If a clot blocks one of the heart's arteries, it can cause a heart attack. If a blood clot in the heart travels to artery it leads to a stroke.

- **Arrhythmias:**

Damage to heart muscle causes disturbance in heartbeat regularity

- **Sudden death:**

If heart muscle is so damaged, it is possible the arrhythmia could cause the heart to suddenly stop beating (sudden cardiac arrest).

Diagnosis can be made out by using

- Electrocardiography,
- Echocardiography,
- Nuclear imaging,
- Nardiac magnetic resonance imaging and
- Endomyocardial biopsy.

ECG of acute myocarditis usually shows

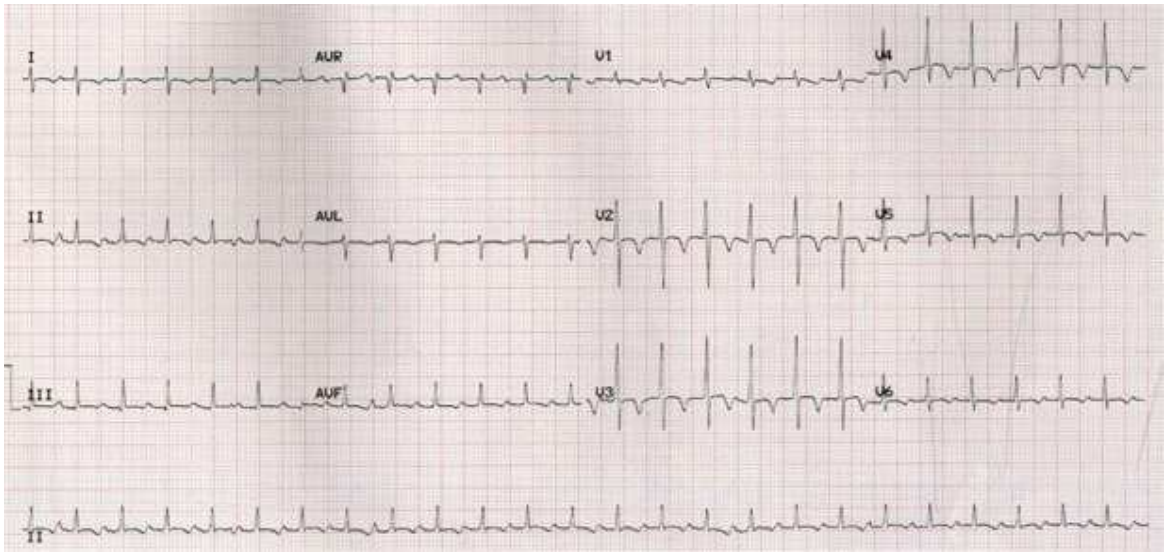
ST elevation without reciprocal ST depression was one of the characteristic finding during acute stage. Total QRS amplitudes in acute stage was low voltaged when compared to those QRS complexes before illness.

Above two finding helps us to differentiate from myocardial infarction.

Also diffuse T wave inversion can occur in these patients along with saddle shaped ST elevation.

Abnormal Q can occur if present, the leads showing Q waves are inversely with left ventricular (LV) ejection fraction[30,31].

ECG OF MYOCARDITIS showing diffuse T wave changes



TREATMENT

It is similar to routine management as managing a cardiac failure patient If myocarditis causes heart failure or rapid or irregular heartbeat, hospitalisation is required.Drugs for regulating heart beat may be required. If heart is weak, doctor may prescribe medications to reduce heart's workload or help eliminate excess fluid.

These medications include:

- **Angiotensin-converting enzyme (ACE) inhibitors:**

  That relax the blood vessels in heart and help blood flow more easily.

- **Angiotensin II receptor blockers (ARBs):**

  That relax the blood vessels in heart and help blood flow more easily

- **Beta blockers:**

  Work in multiple ways to treat heart failure and help control irregular heart rate

- **Diuretics:**

  Such as furosemide (Lasix), that relieve sodium and fluid retention

Treating severe cases:

In some severe cases of myocarditis, aggressive treatment is necessary, such as:

- **Intravenous (IV) medications:**

  IV delivery of medications improves the heart-pump function quickly.

- **A pump in the aorta (intra-aortic balloon pump):**

  In this a balloon is surgically inserted into aorta. As balloon inflates and deflates, it helps increase blood flow and decrease workload on heart.

- **Increasing the oxygen content of the blood (extracorporeal membrane oxygenation, or ECMO):**

  With severe heart failure, doctors sometimes recommend use of device to provide oxygen to the body. When blood is removed from body, it passes through ECMO machine which removes carbon dioxide and adds oxygen to blood. The newly oxygenated blood is returned to body.

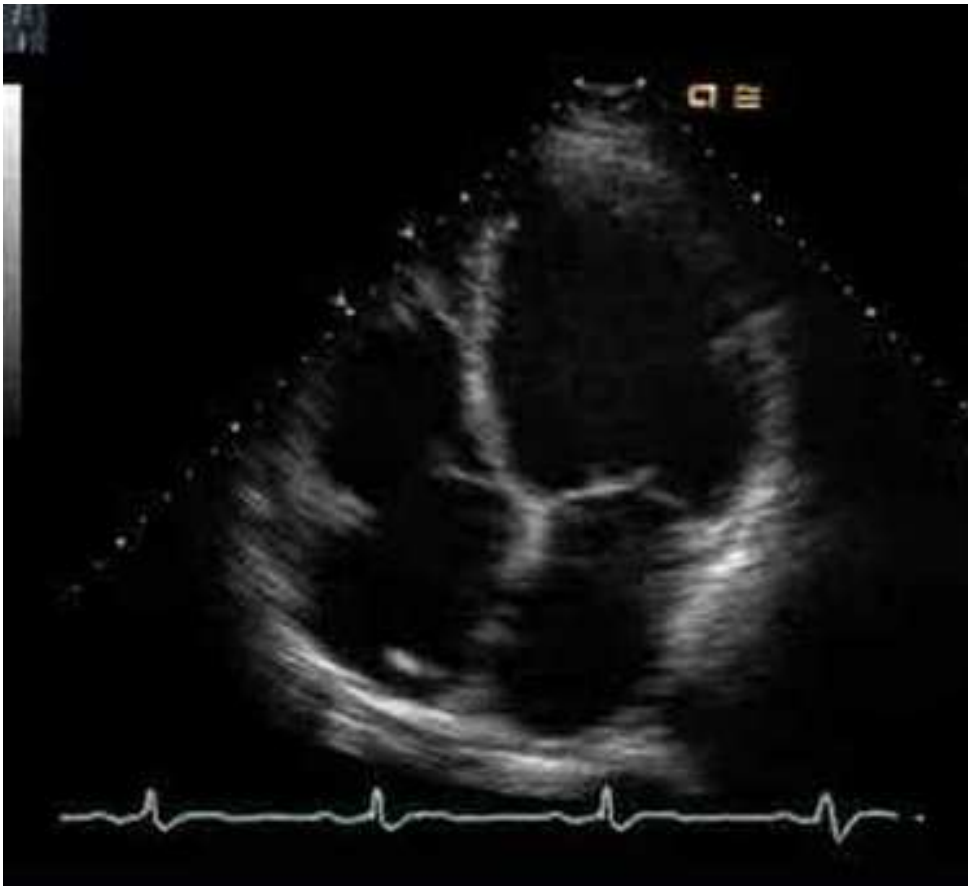
  The ECMO machine takes over the work of the heart.

  This treatment is used to allow the heart recover or to wait for other treatments..

Other management options include surgical techniques like

- ✓ Left ventricular reconstruction and
- ✓ Implantation of external restraint devices.
- ✓ Emerging specific techniques include agents to eradicate persistent viral infections and
- ✓ Immunomodulatory agents .
- ✓ Stem cells for cardiac regeneration and
- ✓ Gene therapy are in the pipeline[32-34].

Echo image of dilated cardiomyopathy due to myocarditis



AIDS RELATED MALIGNANCY

Malignancy is a known complication of immunodeficiency caused by HIV. Mechanism through which these malignancies occur varies among each of the tumours and types.

Tumours commonly seen with HIV include

- Hodgkins disease ,
- Non Hodgkins lymphoma,
- Kaposi sarcoma,
- squamous cell neoplasia,
- plasmacytoma[38].

Some secondary virus associated AIDS malignancies include

Ebstein-Barr virus (EBV) related Non-Hodgkin's lymphoma ,PCNS lymphoma, some systematic and oropharyngeal (T-cell) malignancies, Hodgkin's disease.

Human papillomavirus (HPV) associated Squamous cell neoplasia and other Non-AIDS defining cancers.

KS can occur in three different forms,

- Classic KS, described by Moritz Kaposi, is seen as plaques or nodules on the feet and hands, rarely enters the Viscera.It is seen in males age more than 70 years.

- Endemic KS, is similar to classic KS in its male pattern but differs in that it shows a bimodal age distribution, a larger group occurring at the age of 30 to 50 years, and a lesser group occurring at 5 to 10 years.

Clinically, these lesions are seen on the feet and in the hands, as plaques and enlarge to form nodules that can join to form florid lesions

This type of sarcoma occurs in the heart.

Histologically, all forms of KS appear as the same, seen as vascular clefts and spindle cells(these are the suspected tumor cells).

KS acts as an inflammatory lesion in the earlier stages, with more vascular reaction and less amount of spindle cells. But the volume of spindle cell increases as the lesions begins to mature.

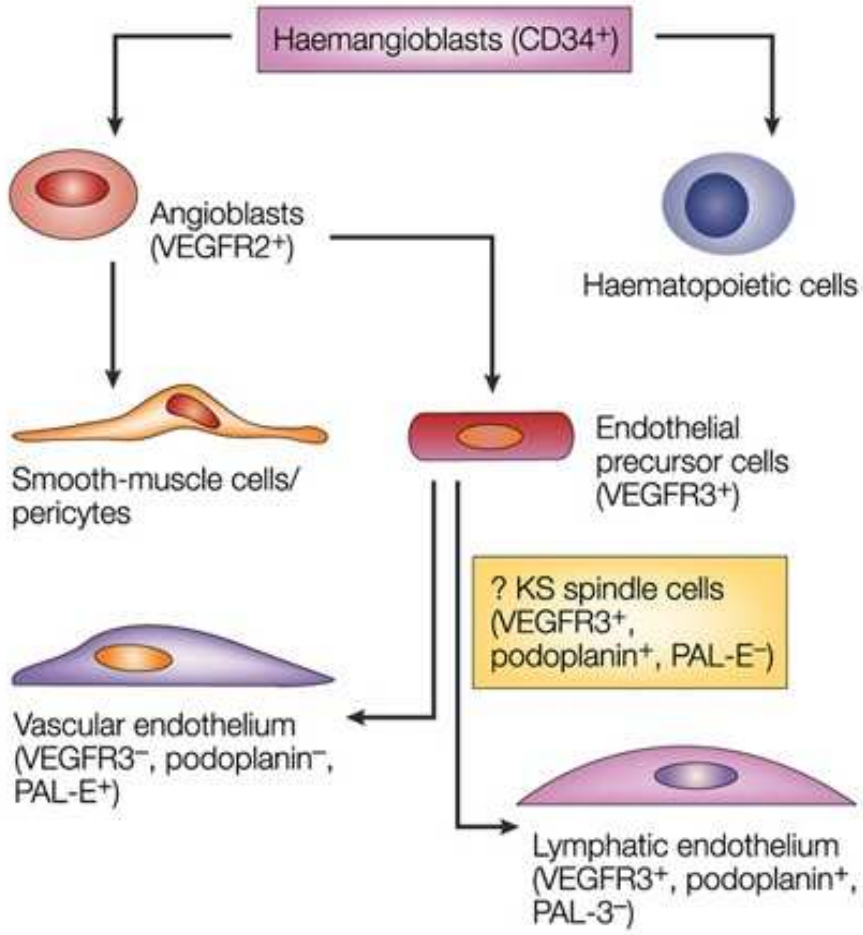
No typical or regular chromosomal or genetic pattern was in the KS spindle cells, and it is not found that KS is formed from a single clone or multiple clones.

Endemic Kaposi sarcoma of heart mostly involves the pericardium both visceral and parietal and in rare cases involves the myocardium , endocardium and coronary vessels.

Cardiac Kaposi sarcoma in HIV patients usually remains occult and often not diagnosed during the life time.

The involvement of Kaposi sarcoma in the pericardium usually does not affect the functioning of the heart but in rare cases it might lead to cardiac tamponade and constrictive pericarditis[39-42].

MECHANISM TO EXPLAIN PATHOGENESIS



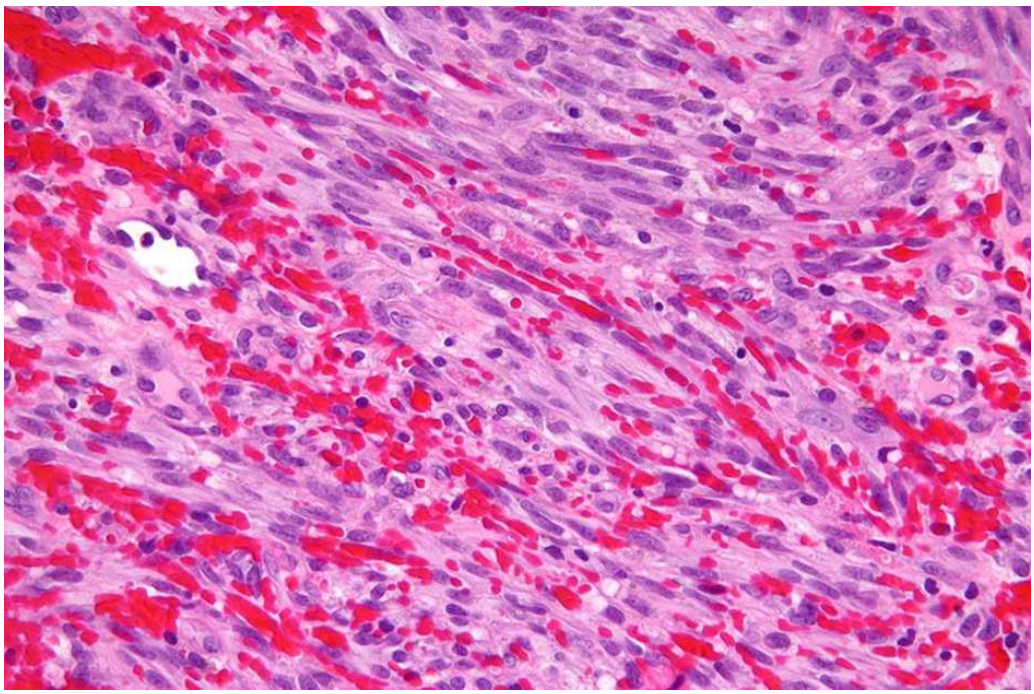
Even though direct lymphomas of the heart are rare, lymphomatous infiltration of the heart might occur due to metastasis from the extranodal sites eg. may be a result of contagious spread from a mediastianal mass. These are are derived from immunoblastic cells of B type or from burkitts.

The involvement can be a small isolated region or diffuse.

In recent times the occurrence of Kaposi sarcoma and non hodgkins lymphomas have dwindled down because of introduction of HAART.

The immunologic status of the patients is now better and hence the reason for lesser incidence of these complications[43-47].

Sectioning of Kaposi sarcoma showing increased vascularity, intracellular hyaline globs and many spindle cells



PULMONARY HYPERTENSION

Lung is one of the frequent target for HIV.The prevalence of HIV related pulmonary hypertension(HRPAH) is about 0.5%.

Association between viral load and CD4 count with HRPAH was not proved significantly in statistics,but pulmonary hypertension is severe in HIV patients.

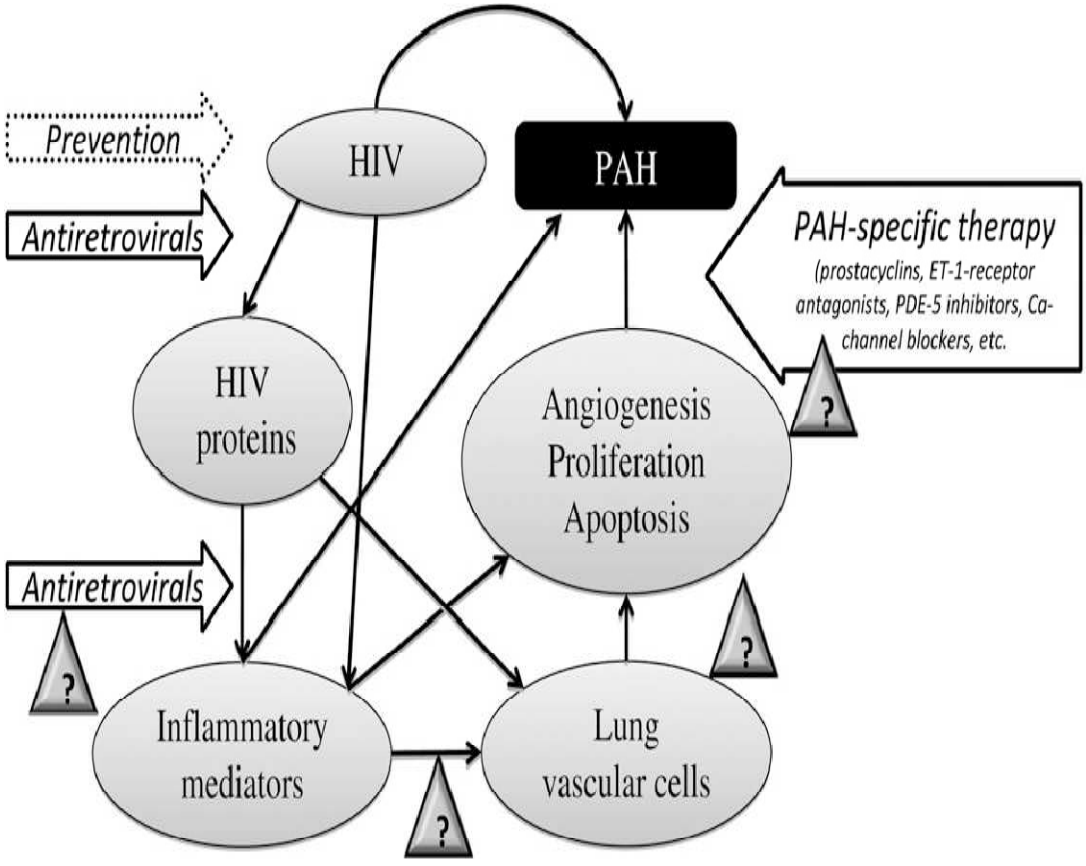
Histological examination of HRPAH and idiopathic PAH are not different.

Pulmonary vasculature is obliterated due to medial hypertrophy and increased proliferation of endothelial and smooth muscle cells.

Proposed mechanism is due chronic infection by hiv results in immune action and subsequent release of proinflammatory cytokines and release of growth factors during later stages. Also subcellular membrane disruption occurs in endothelial cells and smooth muscle cells.

Histology of frequently found plexiform lesions in vessels showed endothelial cells with enlarged endoplasmic reticulum,golgi stacks, vacuolation.

Viral proteins are implicated in all the above mentioned pulmonary vascular damage which includes envelope protein 120(env), hiv negative(nef) factor protein and transcriptor activation protein (tat).Envelope protein attaches to endothelium of vessels as they are vital for attachment of hiv to endothelium and apart from damaging endothelium they activate macrophage and monocyte to release proinflammatory cytokines. Tat and Nef are antigenic and they damage resting T cells[48-49].

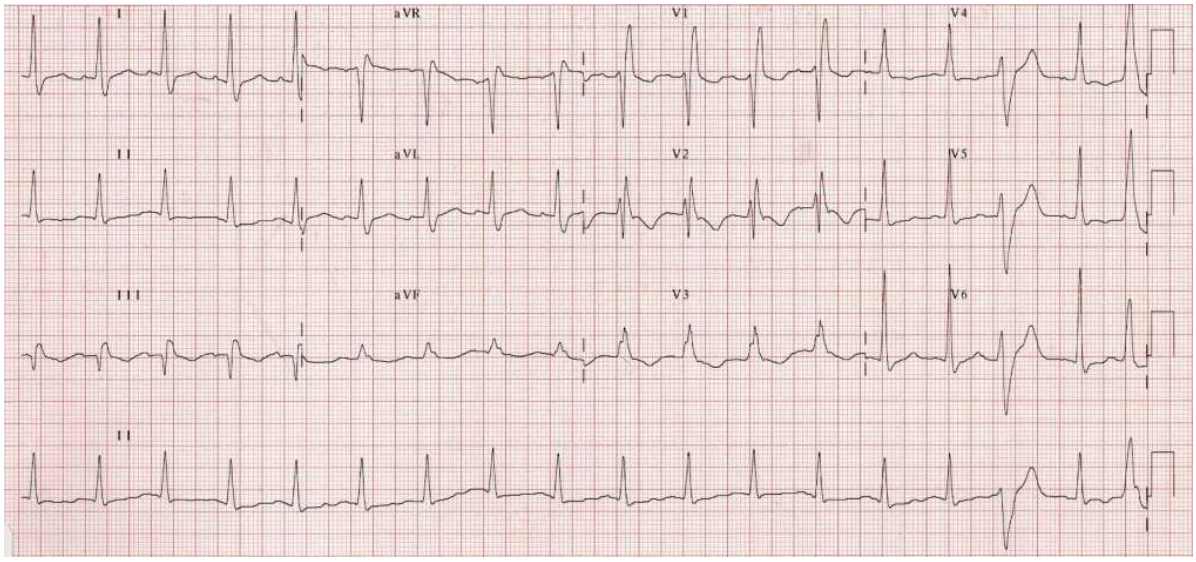


Above picture shows proposed mechanism of HRPAH.

Triangled area are places which need further research work.

ECG manifestation includes typically rightsided atrial enlargement, right ventricle enlargement, axis deviation to right and often an associated right bundle branch block. Echo reveals the presence of right atrial and right ventricular enlargement.

ECG of pulmonary hypertension showing Complete Right bundle branch block with secondary changes



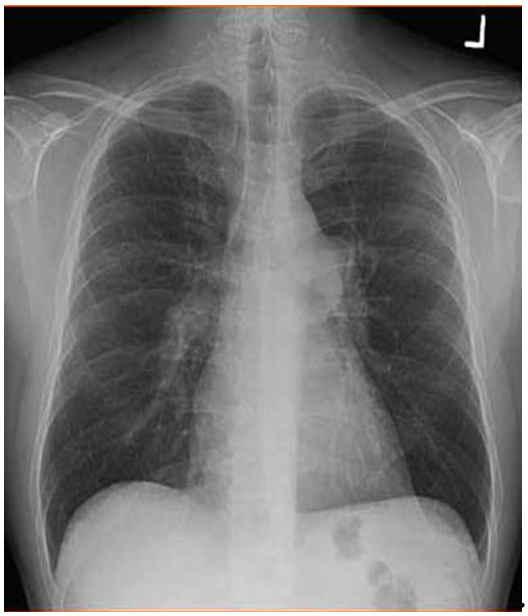
By using Doppler studies we can measure the regurgitant flow over the tricuspid valves with which we can estimate systolic right ventricular and systolic pulmonary artery pressure.

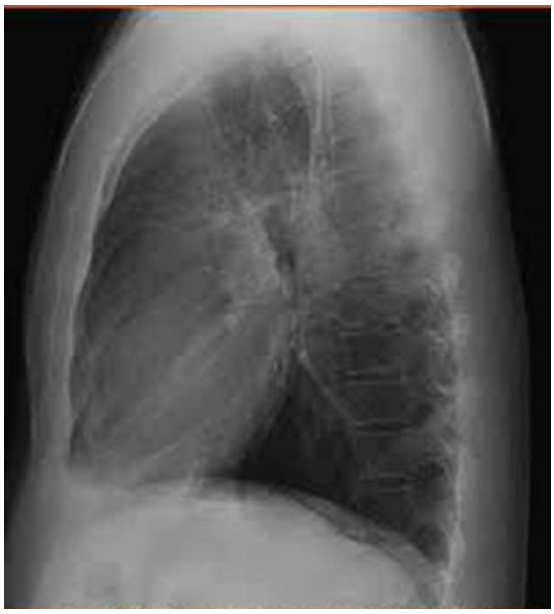
The most accurate measurements of pulmonary and intracardiac pressures can be made with catheterisation of the right sided heart.

Management includes vasodilators like calcium channel blockers, prostacyclin analogue like epoprostenol.

Use of betaprostane orally active prostacyclin is also being studied[50-52].

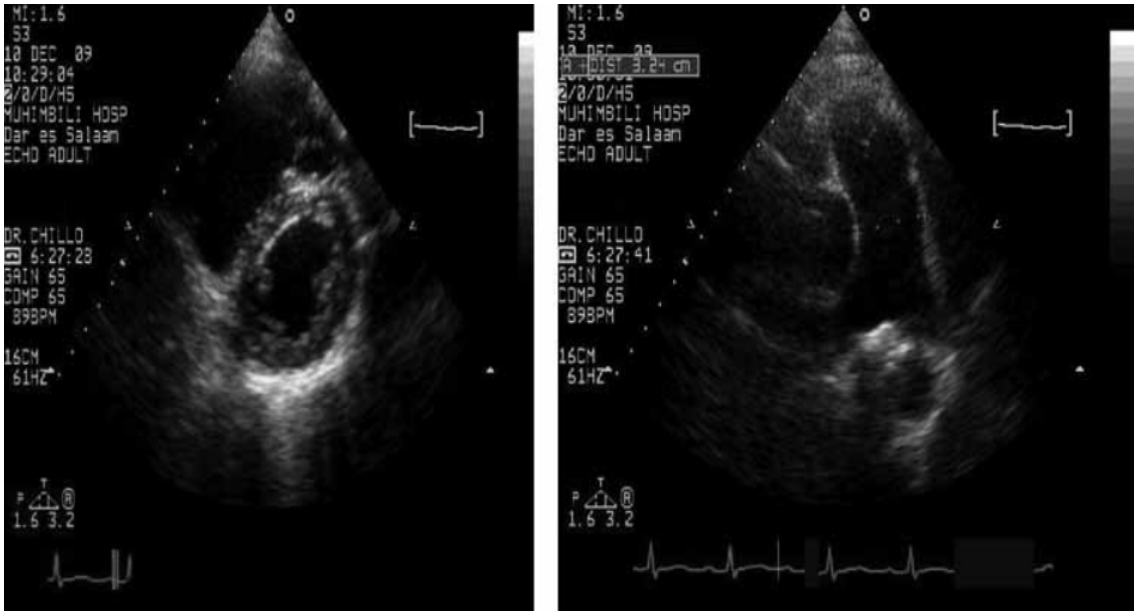
Chest posteroanterior and lateral film in a patient with a positive HIV test result demonstrate clear lungs with prominent hila and dilated pulmonary arteries. These findings are consistent with a diagnosis of pulmonary hypertension

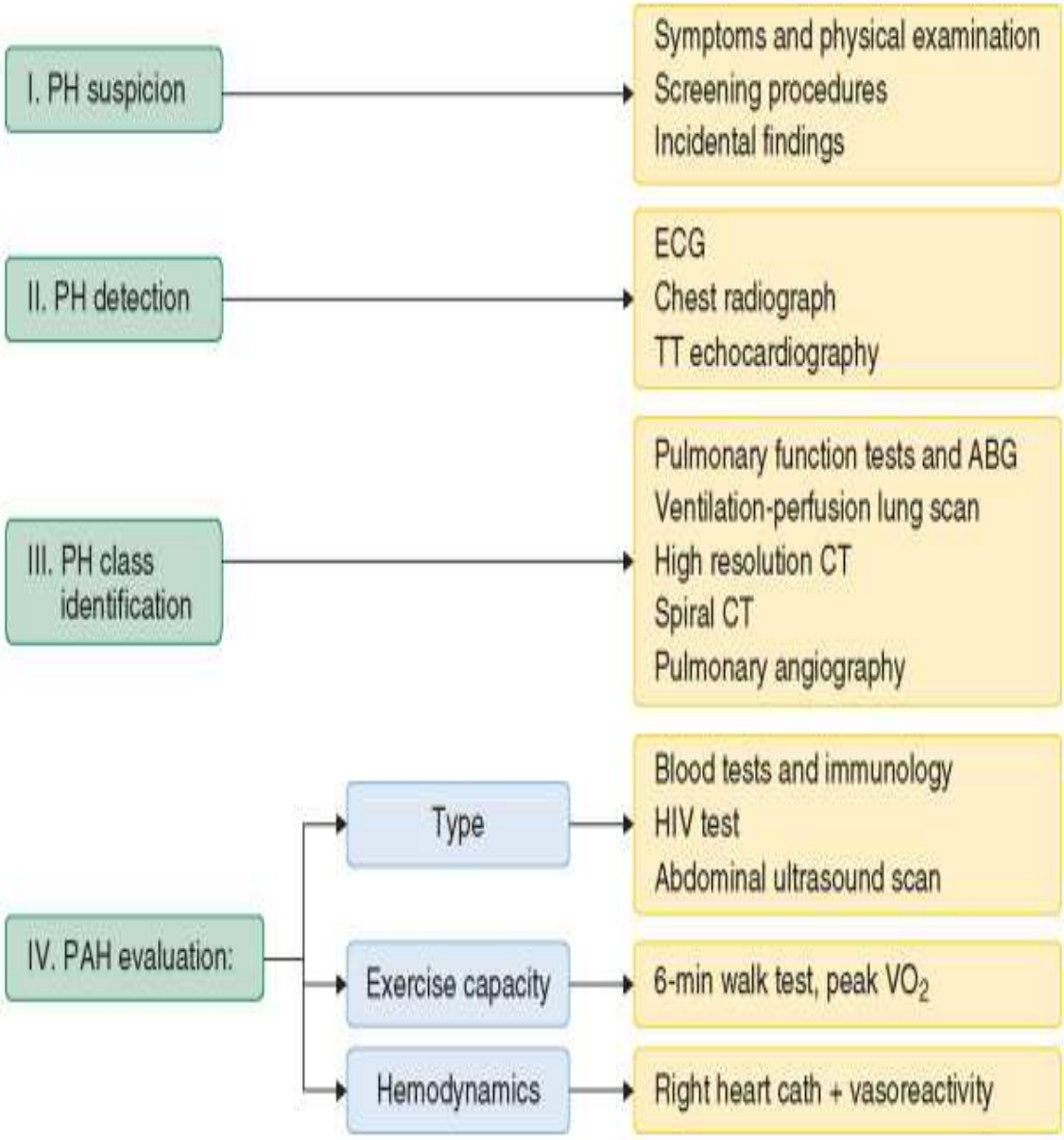




D-sign (left image) and dilated main pulmonary

artery (right image) in a patient with pulmonary hypertension





EVALUATION OF PAH

HIV WITH CORONARY ARTERY DISEASE AND ATHEROSCLEROSIS

With the advent of HAART the progression of HIV infection has slowed down and the patients are living for a longer period of time. As a result of increased life expectancy of the HIV patients now these patients are more prone to risk factors as a normal population for cardiovascular diseases.

At the same time HIV infection by itself is a separate independent risk factor for development of cardiovascular diseases. Moreover HAART especially protease inhibitors induced dyslipidemias and glucose intolerance which further increases the risk of atherosclerosis. Studies have found that HIV patients with coronary vascular disease develop myocardial infarction a decade earlier than the normal population and are more often smokers with deranged lipid profile. Thrombolysis in myocardial infarction score is lower in HIV patients .HIV infected patients have higher chance of restenosis than normal population.

HIV AND HYPERTENSION

Due to reduction in morbidity of HIV patients due to HAART long term adverse effects like dyslipidemia, altered glucose levels, lipodystrophy, and other metabolic complications are on the rise. There are important concerns about premature cardiovascular disease and the role of HAART in its development.

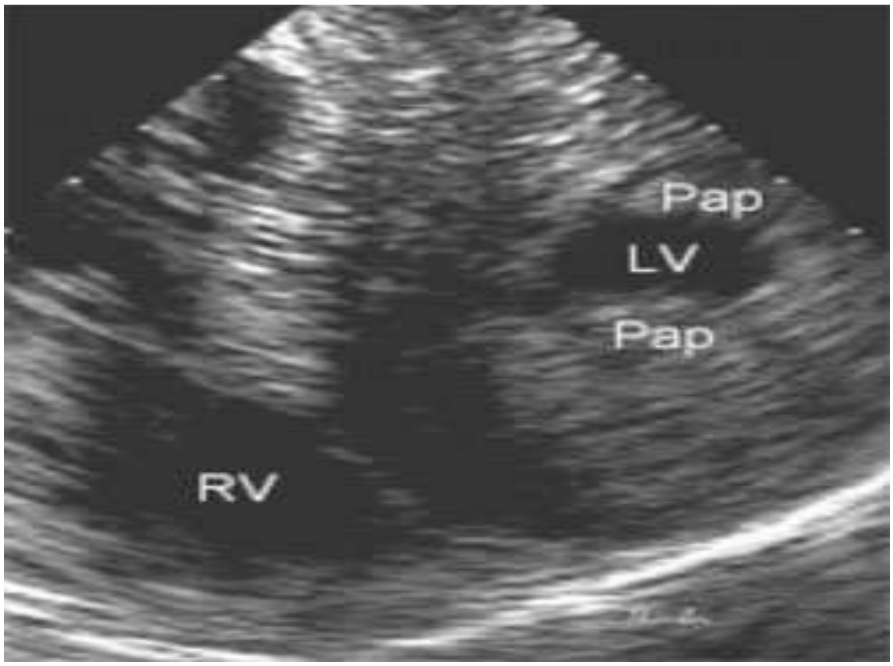
As hypertension is an important risk factor for cardiovascular disease, understanding the association between HAART and hypertension is vital.

Factors that are associated with hypertension include increasing age,long duration of the disease, diabetes, and elevated BMI and other traditional risk factors.

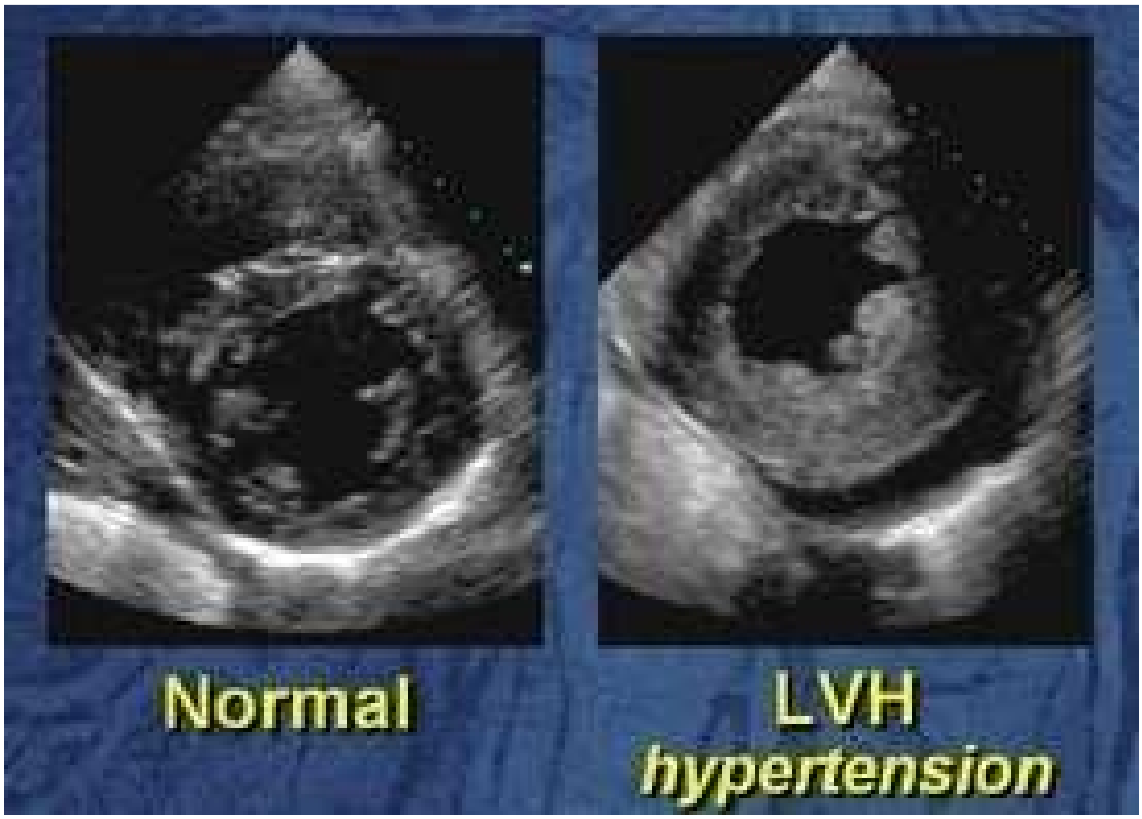
HIV infection was found to cause arterial stiffness and proinflammatory responses which leads to pre-mature vascular dysfunction causing elevated blood pressure. Besides endothelial dysfunction and accelerated vascular aging, subclinical HIV-associated kidney effects that occurs in HIV can also explain the high rates of hypertension among HIV patients

The occurrence of hypertension in HIV patients has been linked to metabolic syndrome and insulin resistance occurring in HIV patients. Some studies indicate that protease inhibitors or NNRTI in the occurrence of hypertension in HIV patients. Smoking is more associated with HIV patients which increases the occurrence of elevated blood pressure[53-57].

ECHO FEATURE OF SEVERE LVH



ECHO IMAGE OF NORMAL HEART AND LVH



HIV AND DIABETES

HIV patients are frequently seen with diabetes and metabolic complications.

Three groups of patients with diabetes and HIV are identified:

Those with preexisting diabetes who contract HIV, those who are found to have diabetes at onset of HIV infection, and others who develop hyperglycemia after start of therapy for HIV. Each subgroups has to be managed differently, because the mechanisms of metabolic dysregulation vary in each of the categories. Traditional risk factors play role also in the development of metabolic complications especially high body mass index, greater waist circumference or waist- to-hip ratio, lower socio economic status, and some ethnic backgrounds and culture added with high viral load, low CD4 count.

Altered glucose tolerance, and insulin resistance usually precede weight loss in HIVpatients.

Insulin resistance, rather than insulin deficiency, is usually the cause in the pathogenesis of diabetes in HIV-infected patients. Earlier reports ,of islet cell autoimmunity, or beta cell destruction has not been seen in HIV patients.

But autoimmune diabetes has been reported to develop in some HIV-infected patients after immune recovery during HAART therapy,

which is type one DM, and the antibody detected was glutamic acid decarboxylase. But majority of patients of were type2 DM.

Studied mechanism implicated in insulin resistance in HIV patients include

1) Endocrine abnormally especially growth hormone resistance,

2) Due to wasting of subcutaneous fat, there occurs visceral accumulation of fat in these patients, which creates increase in the levels of inflammatory cytokines such as TNFa.

3) HIV-infected patients with metabolic syndrome show alterations in inflammation and adipokines

4) Concurrent association of Hepatitis c virus which increases the risk due intrahepatic steatosis and intra heatic necrosis.patients above the age of 40 years are 3 times more at risk of getting diabetes

5) Drugs – PI drugs increase insulin resistance and reduce insulin secretion, by altering GLUT-4 mediated transport of molecules of glucose. PIs interfere with the cellular retinoic acid-binding protein type 1 (CRABP 1) that combines with the peroxisomal proliferator-activated receptor (PPAR). Inhibition of this PPAR causes adipocyte inflammation, and release free fatty acids and hence insulin resistance. This altered glycemic profile resolves in most patients when PI drugs are discontinued. Metabolic effects vary among each PI.

Indinavir causes insulin resistance with no effect on lipid metabolism, but lopinavir and ritonavir cause increase in the fasting triglycerides and free fatty acids levels, but do not alter the insulin sensitivity.

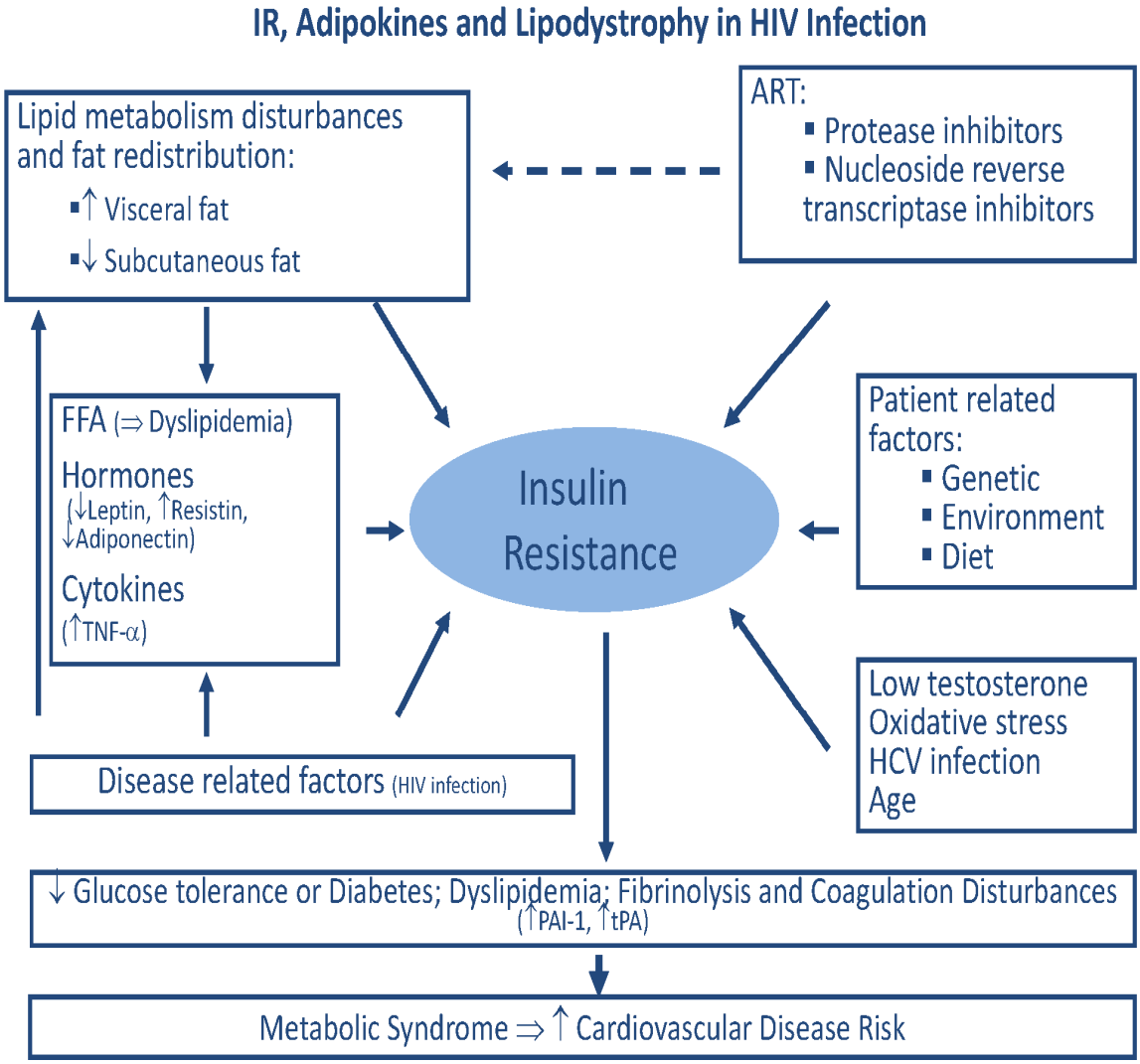
Indinavir and ritonavir inhibits GLUT -4 receptors, HIV patients treated for 3 months with nelfinavir, indinavir, liponavir or saquinavir, show alterations in first phase insulin release with a one fourth reduction in beta cell function.

This shows that there is no group effect of PIs on diabetic and its profile and that each of the PIs must be studied individually, When their metabolic effects are considered.

The treatment implication is that treating doctor must know predominant mechanism of diabetes due to each of the drugs given, only then proper antidiabetic treatment can be given.

NRTI's especially zidovudine,stavudine,didanosine are associated with insulin resistance by causing mitochondrial dysfunction, lipodystrophy.

Drugs like pentamidine, a drug used for treatment of pneumocystis carinii infection in HIV is associated with rise glucose status. so it is important to know about other drugs used in HI[58-60].



Treatment of Coronary Risk Factors in HIV Patients

The primary treated strategy involves tackling the metabolic syndrome associated with HIV infection. Weight loss , exercise, smoking cessation and correcting dyslipidemias are the cornerstone in treatment of risk factors of coronary heart disease in HIV patients

WEIGHT LOSS

If the body weight of the patients is reduced moderately it will lead to drastic improvement in lipid profile, hypertension, and diabetes, inflammatory and thrombotic markers.

SMOKING CESSATION

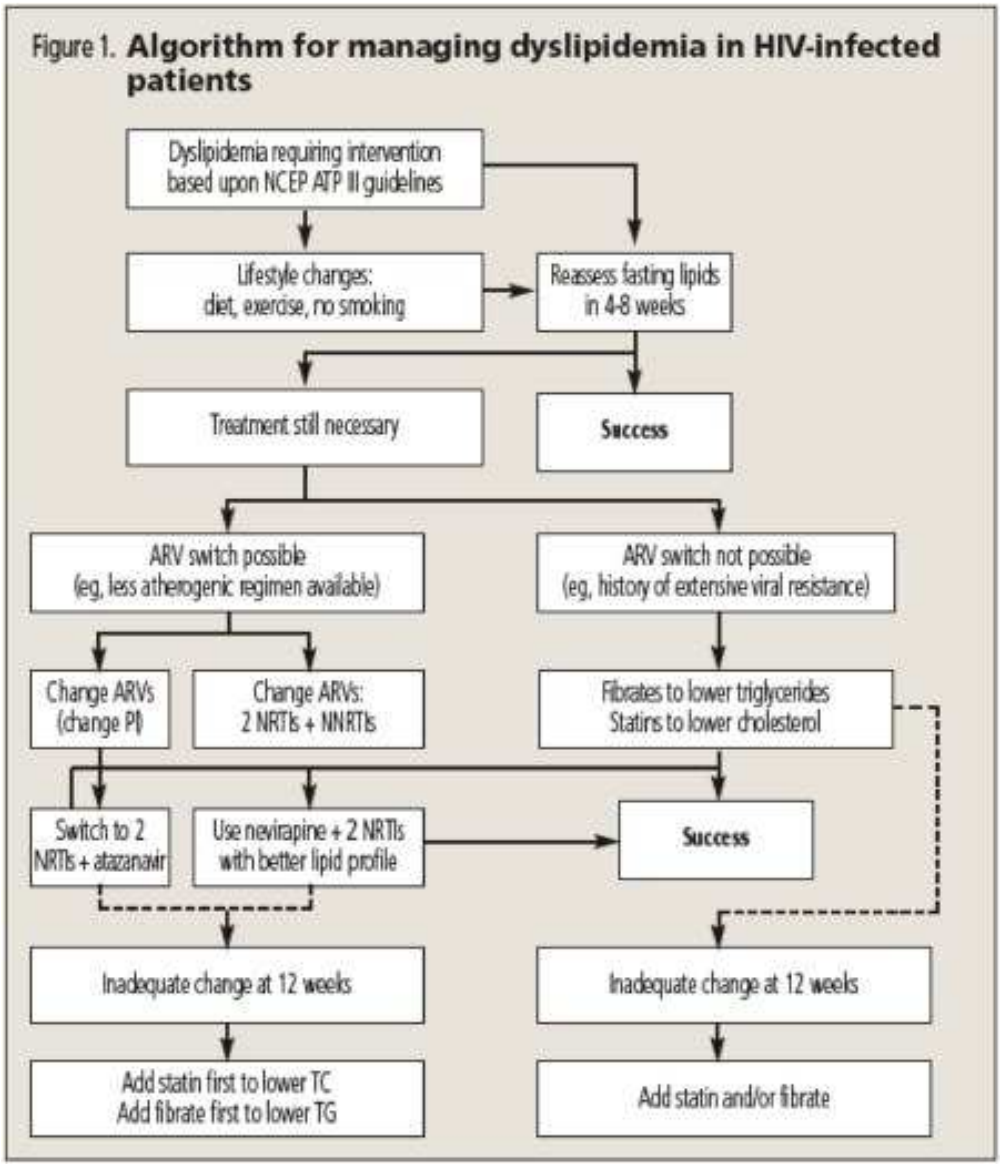
Studies reveal that about two third of the HIV patients are smokers. Smoking cessation must be a prime focus in the care of HIV patients who are smokers.

HIV AND HYPERLIPIDEMIA

Hyperlipidemia in HIV patients is due to increase in very low density lipoprotein and intermediate density lipoproteins. This altered lipid profile is common patients on antiretroviral therapy, particularly with protease inhibitors and stavudine,it can also occur during long duration the disease per se. Guidelines given by National Cholesterol Education Program (NCEP) cannot be implemented completely to HIV- patients. Mechanism of hyperlipidemia due to these drugs was thought to be due to reduction in the clearance of VLDL and triglyceride remnants,or an insulin resistance which causes sequestration of free fatty acids adipose tissue due resistance occurring there, with resulting increased VLDL – TGL production.Hence these patients are at increased risk of adverse cardiovascular events.

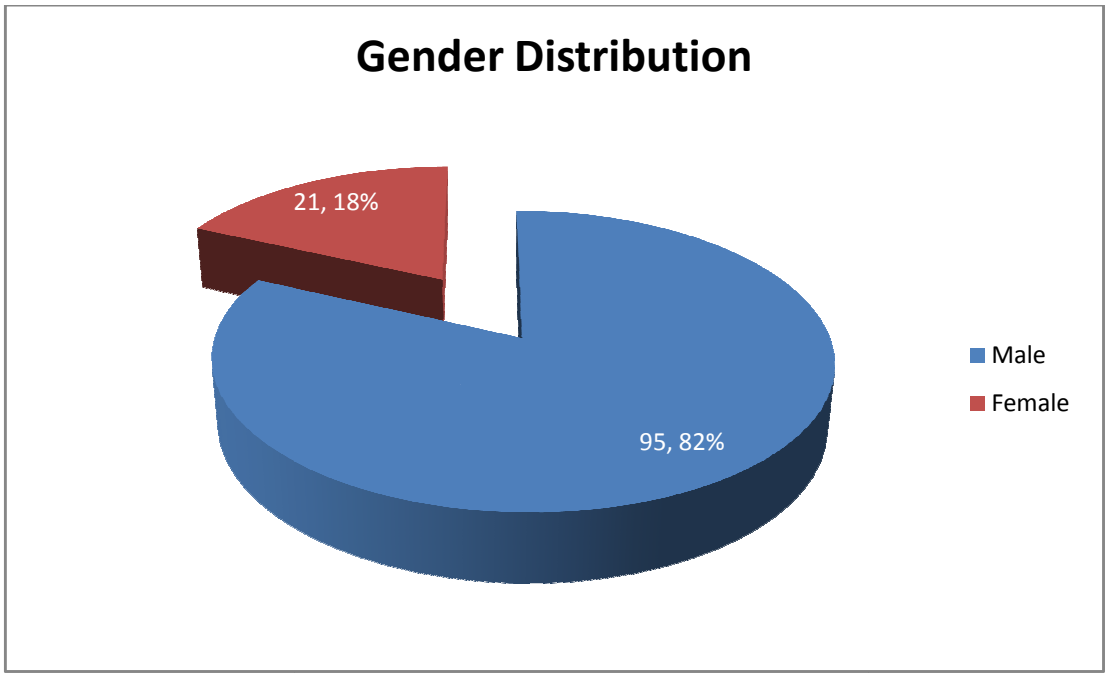
These patients are refractory to routine pharmacotherapy due to above mechanism and NCEP's recommendation has to be modified. A patient's cardiac risk should first be assessed. Measures like low-fat diet, weight reduction, and exercise, must be instituted early. Drug treatment is recommended for patients with familial combined hyperlipidemia, for patients with triglyceride values more than 1000 mg/dL. Drugs used are niacin and statins, fibric acid derivatives and probucol. Any drug change

among antiretroviral drugs, that has suspected to be causing hyperlidemia should be carefully done as it can result in dangerous virus rebound and disease progression. NCEP guidelines advise monitoring low-density-lipoprotein cholesterol levels four to six weeks after starting lipid lowering therapy and then at three months in HIV negative patients; more frequent assay has to be done in HIV-infected patients[62-64].



RESULTS

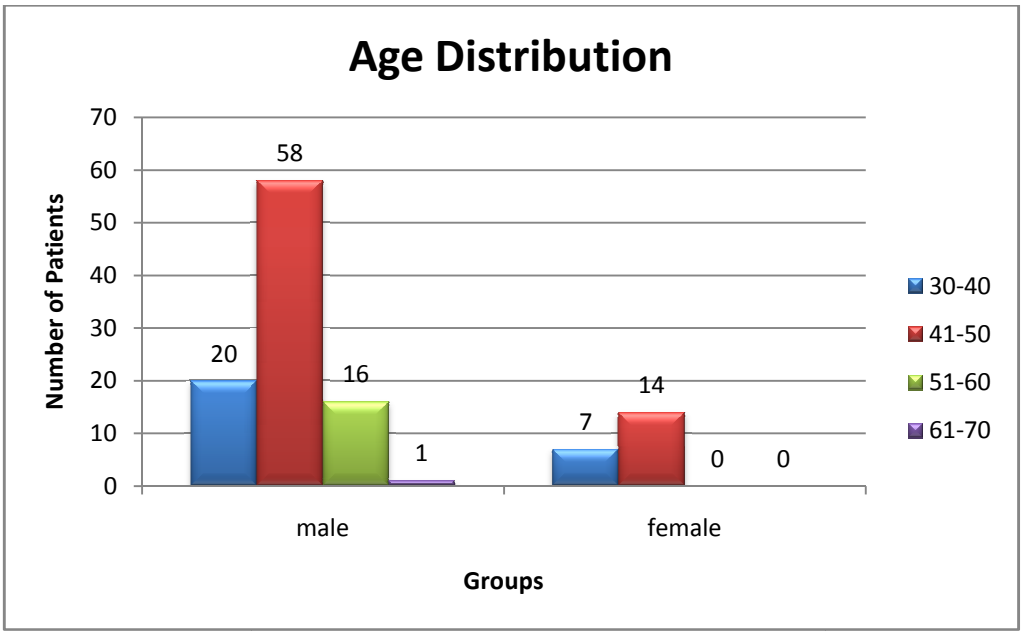
Gender



Gender	Male	Female	Total
Number	95	21	116
Percentage	82	18	100

82% of the patents were males and 18% were females.

AGE

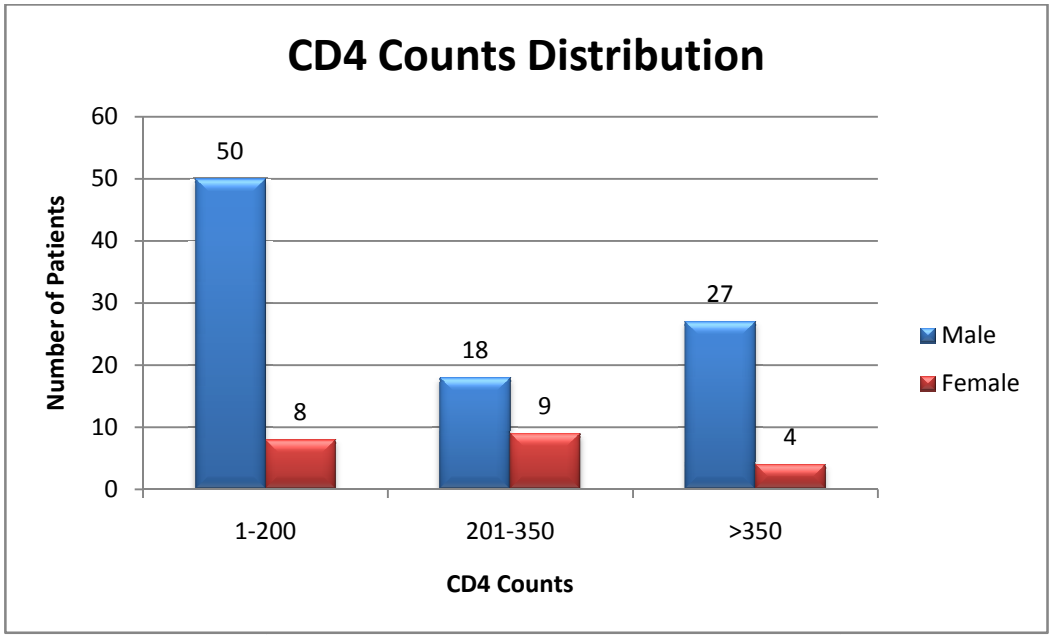


Age Group	male	%	female	%
30-40	20	21.05	7	33.33
41-50	58	61.05	14	66.67
51-60	16	16.84	0	0.00
61-70	1	1.05	0	0.00
Total	95	100	21	100

Age	male	female
N	95	21
Mean	45.20	41.52
SD	5.84	6.09
P value Unpaired t test	0.107	

In our study majority of the patients are in the age group 41-50 years and only 17% of the males and no females above 50 years of age.By conventional criteria age difference between males and females is considered to be not statistically significant since $p > 0.05$.this establishes the fact that in spite of difference in the number of observations in each gender group, we are dealing with statistically similar groups.

CD4 COUNT



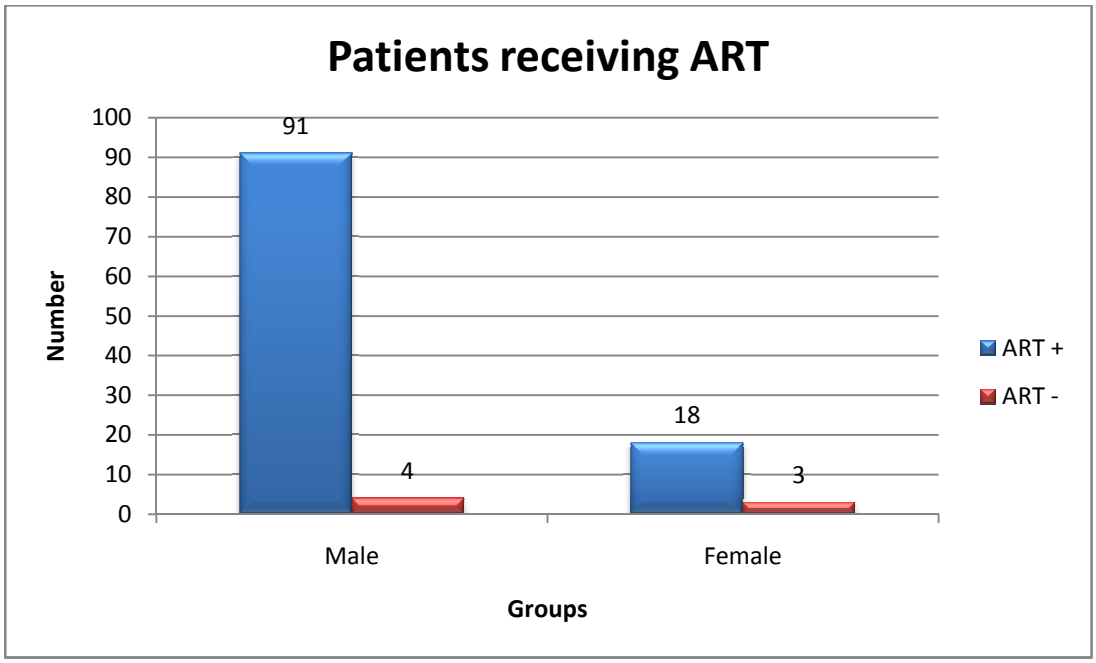
CD4 Count	Male	Percentage	Female	Percentage
1-200	50	53	8	38
201-350	18	19	9	43
>350	27	28	4	19
Total	95	100	21	100
Proportion with CD4 < 200 (%)	P value Fisher's exact test	0.335		

CD4 Count	Male	Female
Number of Observations	95	21
Mean Age	271.49	271.48
Standard Deviation	208.53	153.06
P value Unpaired t test	0.9998	

58(45.5%) of the patients had CD4 count less than 200, 31% had CD4 count between 201-350 and 23.5% had CD4 count more than 350.

By conventional criteria the association between the gender groups and CD4 count is considered to be not statistically significant since $p > 0.05$. Even the Proportion with CD4 < 200 is not considered to be statistically significant since $p > 0.05$.

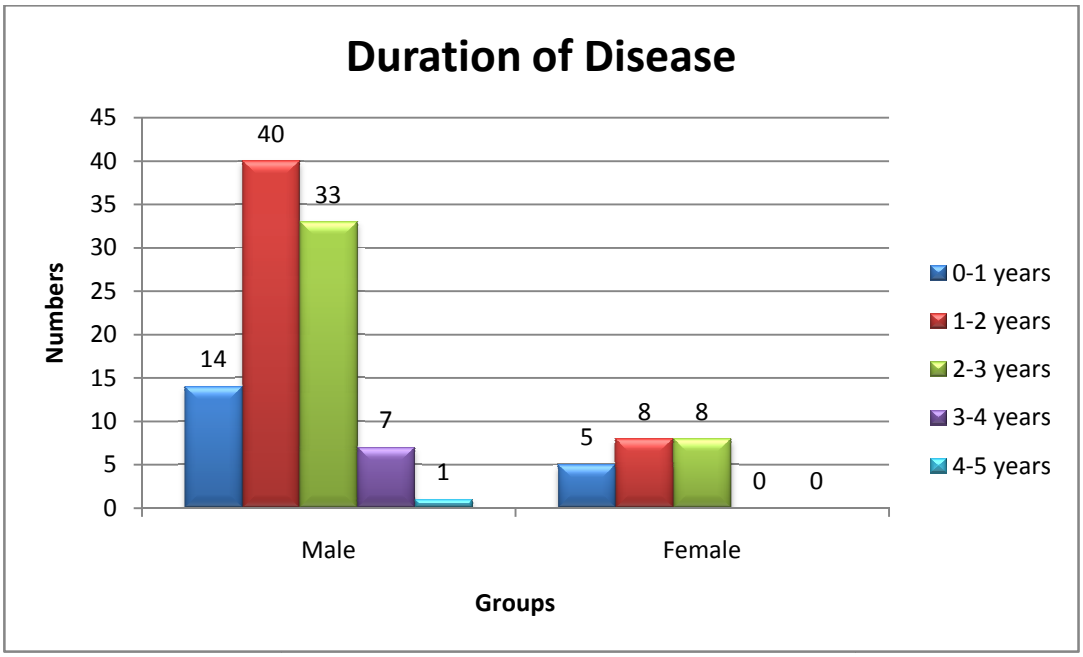
PATIENTS RECEIVING ART



Patients receiving ART	Male	Percentage	Female	Percentage
ART +	91	96	18	86
ART -	4	4	3	14
Total	95	100	21	100
P value Fisher's exact test	0.1106			

109 patients (91%) were on Anti Retroviral Therapy and 9% are not on ART. By conventional criteria the association between the gender groups and patients on ART is considered to be not statistically significant since $p > 0.05$.

DURATION OF DISEASE

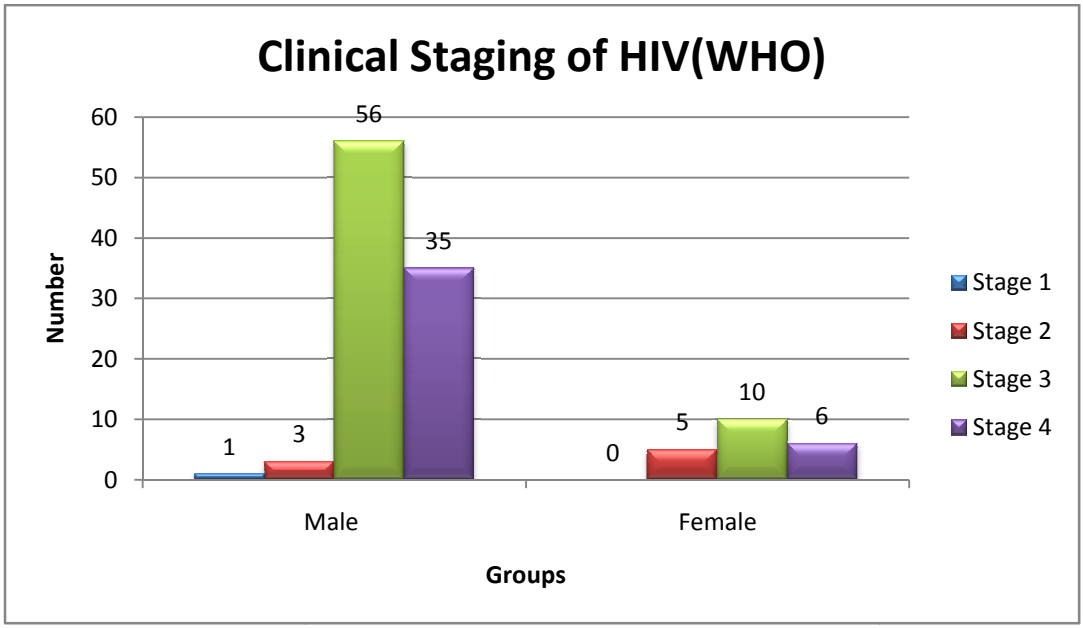


Duration of disease in years	Male	Percentage	Female	Percentage
0-1 years	14	15	5	24
1-2 years	40	42	8	38
2-3 years	33	35	8	38
3-4 years	7	7	0	0
4-5 years	1	1	0	0
Total	95	100	21	100

Duration of Disease in years	Male	Female
Number of Observations	95	21
Mean Age	2.37	2.10
Standard Deviation	0.86	0.82
P value Unpaired t test	0.17128	

Majority of the patients (40%) had the disease between 1-2 years. 8% of the males and none of the females had the disease beyond 3 years. By conventional criteria the association between the gender groups and duration of the disease is considered to be not statistically significant since $p > 0.05$

CLINICAL STAGING OF HIV (WHO)

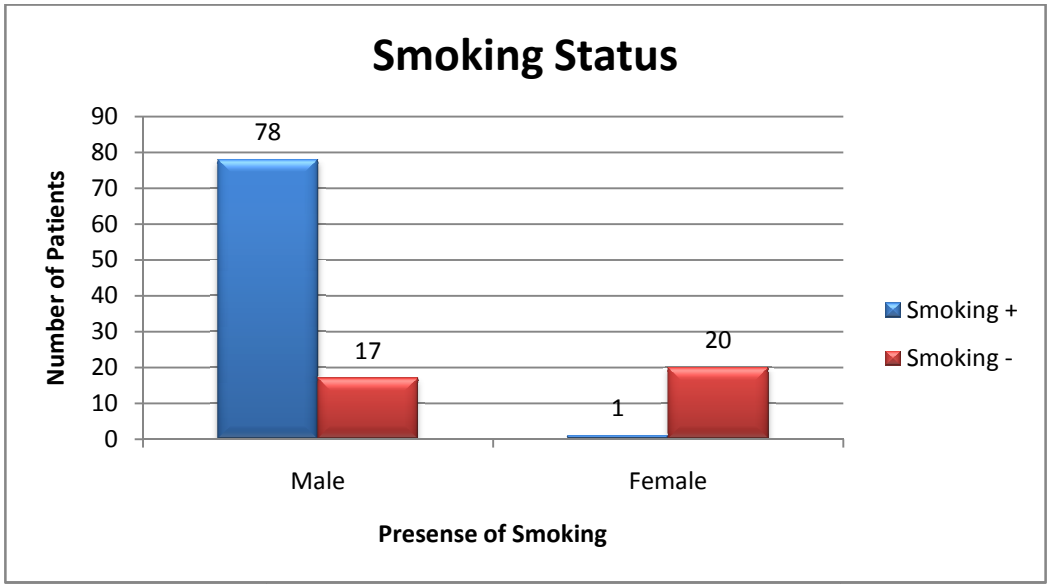


Clinical Staging of HIV(WHO)	Male	Percentage	Female	Percentage	P value Fisher's exact test
Stage 1	1	1	0	0	0.009
Stage 2	3	3	5	24	
Stage 3	56	59	10	48	
Stage 4	35	37	6	29	
Total	95	100	21	100	

At presentation most patients were in the WHO HIV clinical stage 3 (53.5%) followed by stage 4 (33%).By conventional criteria the association between the gender groups and clinical staging of HIV is considered to be statistically significant since $p < 0.05$.

This indicates that there is a true difference among groups and the difference is significant. In simple terms male patients in our study tended to be in stage 3 and 4 of the clinical staging when compared to female patients. In respect of WHO clinical staging, 96% of HIV positive male subjects were in WHO Stages 3 and 4 compared to 77% of female subjects at the same stages. This difference is true and significant and has not occurred by chance.

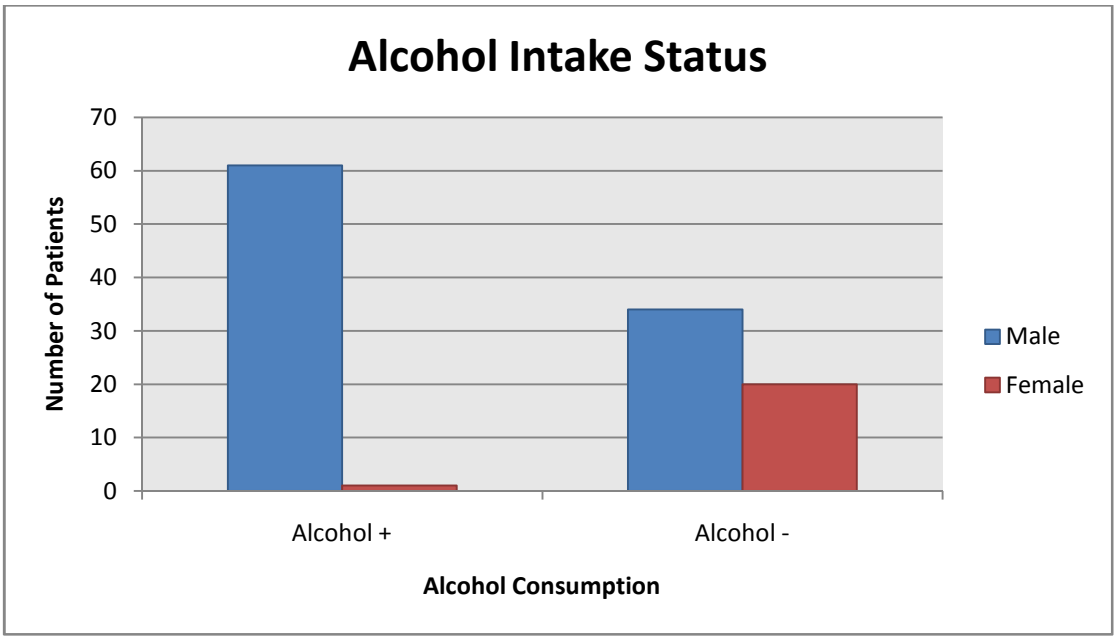
SMOKING



Smoking Status	Male	Percentage	Female	Percentage
Smoking +	78	82	1	5
Smoking -	17	18	20	95
Total	95	100	21	100
P value Fisher's exact test	0.0001			

In our study 82% (n=78) of male subjects and 5% (n=1) of the female subjects had the habit of smoking. By conventional criteria the association between the gender groups and smoking is considered to be statistically significant since $p < 0.05$.This indicates that there is a true difference among groups in reference to smoking habit and the difference is significant. It indicates significantly more men are smokers. This difference is true and has not occurred by chance.

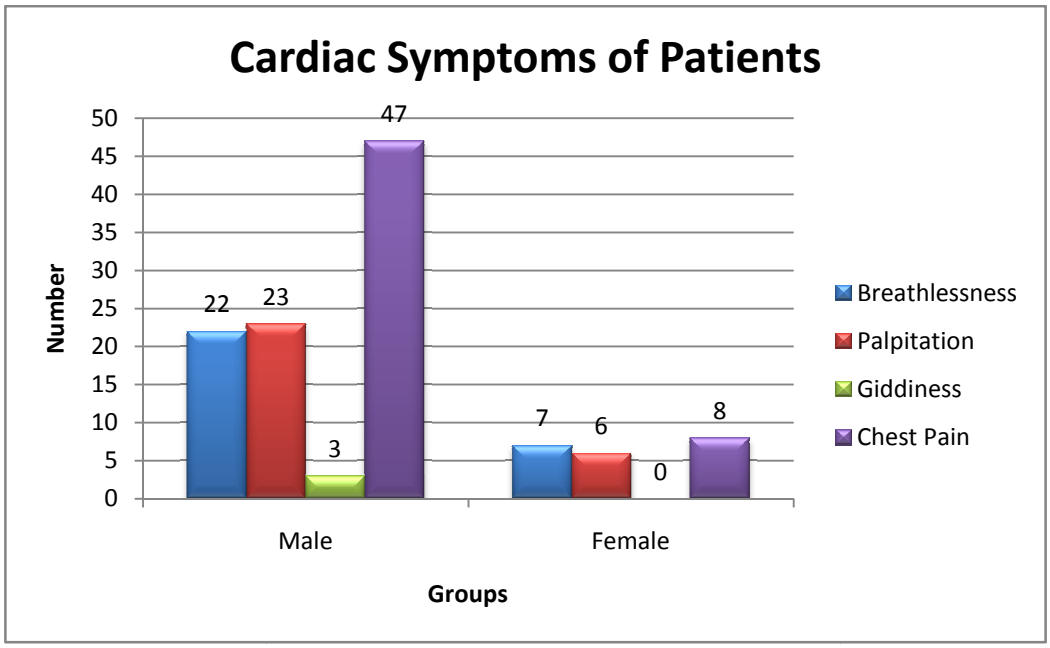
ALCOHOL



Alcohol Intake Status	Male	Percentage	Female	Percentage
Alcohol +	61	64	1	5
Alcohol -	34	36	20	95
Total	95	100	21	100
P value Fisher's exact test	0.0001			

In our study 64% (n=61) of male subjects and 5% (n=1) of the female subjects had the habit of consuming alcohol. By conventional criteria the association between the gender groups and alcohol consumption is considered to be statistically significant since $p < 0.05$.This indicates that there is a true difference among groups in reference to consuming alcohol and the difference is significant. It indicates significantly more men are alcohol consumers among HIV+ patients in our study. This difference is true and has not occurred by chance.

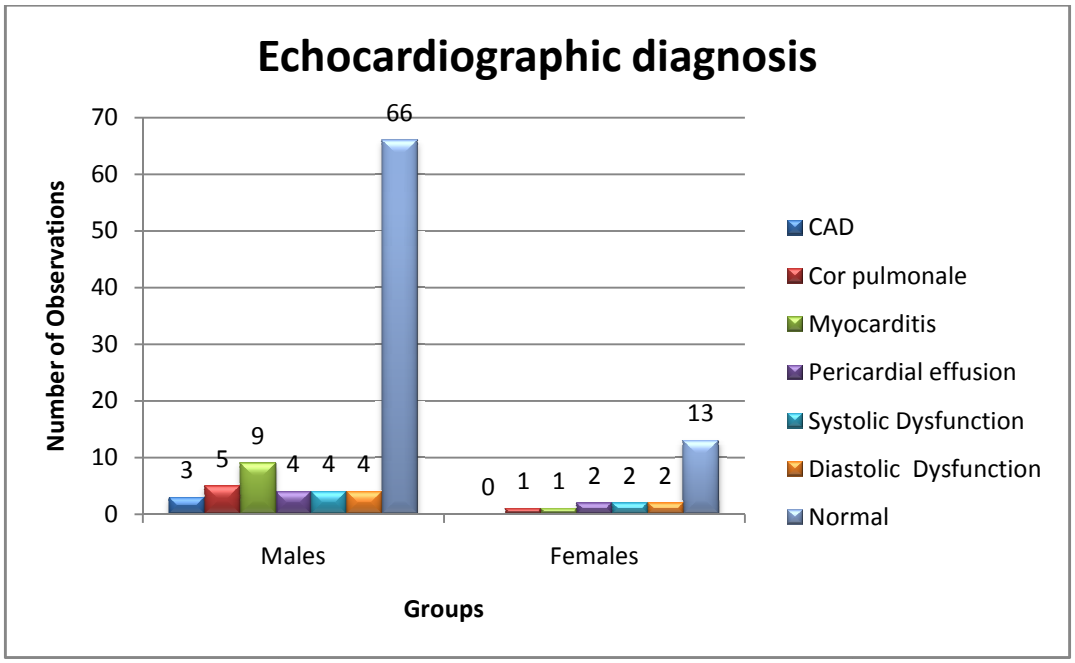
Cardiac Symptoms



Cardiac Symptoms ofPatients	Male	Percentage	Female	Percentage
Breathlessness	22	23	7	33
Palpitation	23	24	6	29
Giddiness	3	3	0	0
Chest Pain	47	49	8	38
Total	95	100	21	100

The most common presenting cardiac symptom was chest pain (43.5%), while the least common was giddiness (1.5%). Breathlessness and palpitation were present in 28% and 26.5% of the patients, respectively. Multiple complaints were common and 38% of the patients had more than one symptom.

ECHOCARDIOGRAPHIC DIAGNOSIS



ECHO Diagnosis	Males	Percentage	Females	Percentage
CAD	3	3	0	0
Corpulmonale	5	5	1	5
Myocarditis	9	9	1	5
Pericardial effusion	4	4	2	10
Systolic Dysfunction	4	4	2	10
Diastolic Dysfunction	4	4	2	10
Normal	66	69	13	62
Total	95	100	21	100

The most common echocardiographic diagnosis was myocarditis, pericardialeffusion, systolic dysfunction and diastolic dysfunction, each at 7% respectively. 65.55 of the study subjects were significantly normal.

DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF ECHOCARDIOGRAPHIC DIAGNOSES

	CAD	Corpulmonale	Myocarditis	Pericardial effusion	Systolic Dysfunction	Diastolic Dysfunction	Normal
N	3	6	10	6	6	6	79
Mean (SD) age (years)	46.7(4.2)	45.8(5.2)	40.8(4.3)*	39.3(6.9)*	41.3(8.9)	43.8(3.8)	45.5(5.90
Males (%)	100	83	90	67	67	67	84
Mean (SD) SBP (mmHg)	110.7(19.7)	115(5.2)*	109.4(5.7)*	104.7(8.2)	114(8.4)	115(3.7)	120.1(9.7)*
Mean (SD) DBP (mmHg)	79.3(16.8)	77(5.3)	71.2(4.8)*	73.3(7.2)	69.7(5.1)*	71.3(5.0)	75.6(7.7)*
Mean (SD) pulse rate (b/min)	84.7(10.5)	115.7(4.6)*	116.3(5.80*	100(1.8)	88(9.2)	81.8(7.0)	81.5(7.4)*
Mean (SD) Hb (g/dl)	8.7(0.5)	8.4(1.4)	8.5(1.0)	9.7(0.8)	9.2(0.6)*	8.8(1.0)*	8.0(1.1)
Mean (SD) WBC count in cells ×	5.7(0.03)*	11.8(2.7)*	12.1(0.09)*	11.1(0.6)*	5.7(0.8)*	5.2(0.8)	4.6(1.5)

109/l							
Mean (SD) serum creatinine (µmol/l)	83(15)	93(16)	93(19)	82(18)	72(17)*	100(9)	102(23)
Mean (SD) serum cholesterol (mmol/l)	1.74(0.09)*	1.91(0.07)	1.87(0.05)	1.86(0.04)	2.10(0.45)	1.78(0.17)	1.90(0.16)
Mean (SD) CD4 cell count (cells/µl)	503(18)*	300(66)*	88(23)*	352(136)*	510(105)*	562(78)*	238(192)*
% on ART	100	100	100	100	83	100	92
Mean (SD) duration on ART (Years)	2.3(0.06)	2.3(1.2)	2(0.08)	2.6(0.5)	2.3(1.2)	2.5(0.5)	2.3(0.9)
Presenting cardiac symptoms (n)**							
Palpitations	0*	2*	6	2*	2*	1*	30
SOB	3*	6*	6*	5*	3*	3*	35
Chest pain	3*	2*	7*	3*	5*	2*	34
Giddiness	0	0	0	0	0	0	3

- P<0.05, ** Multiple diagnosis present

As shown in Table 2, the echocardiographic diagnoses did not differ significantly between men and women, or between patients on ART and those not on ART.

A univariate analysis was performed to compare having the different echocardiographically determined diagnoses in the HIV-positive patients and those without it. The table below gives the results following analysis.

Echocardiographically determined diagnoses	**Demographic and clinical characteristics**
CAD	• Lowercholesterol levels (1.74 vs 1.90mmol/l, p = 0.033) • Higher white blood cell (WBC) count (5.7 vs 4.6× 109 cells/l, p = 0.001). • Higher CD4cell counts (503 vs 238 cells/µl, p <0.000). • More likely to present with palpitations (p = 0.001), breathlessness (p = 0.000), and chest pain (p = 0.000),
Corpulmonale	• Lower systolic BP (115 vs 120.1 mmHg, p <0.032) • Higher mean resting pulse rate (115 vs 81.5 beats/min, p < 0.000).

	• Higher white blood cell (WBC) count (11.8 vs 4.6× 109 cells/l, p = 0.001). • Higher CD4 cell counts (300 vs 238 cells/µl, p <0.000). • More likely to present with palpitations (p = 0.001), breathlessness (p = 0.003), and chest pain (p = 0.000),
Myocarditis	• Younger (40.8 vs 45.5 years, p <0.004) • Lower systolic BP (109.4 vs 120.1 mmHg, p <0.000) • Lower diastolic BP (71.2 vs 75.6 mmHg, p <0.012) • Higher mean resting pulse rate (116.3 vs 81.5 beats/min, p < 0.000). • Higher white blood cell (WBC)count (12.1 vs 4.6× 109 cells/l, p = 0.000). • Lower CD4cell counts (88 vs 238 cells/µl, p <0.000). • More likely to present with breathlessness (p = 0.003), and chest pain (p = 0.021),

Pericardial effusion	• Younger (39.3 vs 45.5 years, p <0.040) • Higher white blood cell (WBC) count (11.1 vs 4.6× 109 cells/l, p = 0.000). • Higher CD4 cell counts (352 vs 238 cells/μl, p <0.049). • More likely to present with palpitations (p = 0.001), breathlessness (p = 0.001), and chest pain (p = 0.000),
Systolic Dysfunction	• Lower diastolic BP (69.7 vs 75.6 mmHg, p <0.02) • Higher haemoglobin level (9.2 vs 8.0 g/dl, p = 0.002). • Higher white blood cell (WBC) count (5.7 vs 4.6× 109 cells/l, p = 0.006). • Lower serum creatinine (72 vs 102μmol/l, p = 0.003) • Higher CD4 cell counts (510 vs 238 cells/μl, p <0.000). • More likely to present with palpitations (p = 0.001), breathlessness (p = 0.000), and chest pain (p = 0.002),

Diastolic Dysfunction	• Higher CD4 cell counts (562 vs 238 cells/μl, p <0.000). • More likely to present with palpitations (p = 0.000), breathlessness (p = 0.000), and chest pain (p = 0.000),

DISCUSSION

HIV infection is a pandemic now. Its incidence and prevalence is increasing rapidly. The disease affects all the systems in the body.

But cardiac involvement in HIV infection is common but not clinically appreciated.

Cardiac involvement in HIV infection is common and occurs quite early in disease process.

Our study showed that majority of patients with HIV infection had echocardiographic findings which were clinically significant.

This shows that echocardiography w as an essential tool for diagnosis of subclinical cardiac abnormalities and it will help in instituting management at an earlier stage.

The subjects in our study resembled a typical HIV cohort.

In our study 82% of the patents were males and 18% were females.

Majority of the patients are in the age group 41-50 years and only 17% of the males and no females above 50 years of age.

The male female ratio is 4.5:1. All these findings were consistent with the Key findings of HIV Estimations 2012 report by NACO.

All the patients presented with symptoms of cardiac disease. 32% of the patients were confirmed with cardiac disease based on invasive cardiac investigations. 58(45.5%) of the patients had CD4 count less than 200, 31% had CD4 count between 201-350 and 23.5% had CD4 count more than 350.109 patients (91%) were on Anti Retroviral Therapy and 9% are not on ART.

Majority of the patients (40%) had the disease between 1-2 years. 8% of the males and none of the females had the disease beyond 3 years. At presentation most patients were in the WHO HIV clinical stage 3 (53.5%) followed by stage 4 (33%).

In our study 82% of male subjects and 5% of the female subjects had the habit of smoking. **In our study** 64% of male subjects and 5% of the female subjects had the habit of consuming alcohol.

The most common echocardiographic diagnosis was

1) Myocarditis,

2) Pericardial effusion,

3) Systolic dysfunction and

4) Diastolic dysfunction

each at 7% respectively.

65.55% of the study subjects were significantly normal.

Hiv And Coronary Artery Disease

In our study HIV patients with CAD presented with Lower cholesterol levels, higher white blood cell (WBC) count, higher CD4 cell count and symptoms of palpitations, breathlessness and chest pain.

Traditional risk factors lead to increased coronary artery disease in the population.

HIV infection is independently associated with increased cardiovascular risk.

ART also contributes to risk of coronary artery disease especially in patients on protease inhibitors.

Usually increased cholesterol levels, smoking and decreased CD4 levels are recognized risk factors for CAD.

But in our study apart from smoking status other variables are not in tune with previous studies which have found increased incidence of heart attacks and other cardiovascular events among HIV infected induviduals.

Hiv And Corpulmonale

Incidence of pulmonary hypertension was significant in our study.

In our study HIV patients with corpulmonale presented with lower systolic BP, higher mean resting pulse rate, higher white blood cell (WBC)

count, higher CD4 cell counts and symptoms of palpitations, breathlessness and chest pain. The association between HIV, corpulmonale and pulmonary hypertension is well established.

Common presentation of our patients with pulmonary hypertension was shortness of breath, and in all cases it was attributed to lung disease.

These patients did not had any advanced immunosuppression, in other words there was no relation to the disease stage as reflected by CD4 count in these patients.

The majority of our patients presentation was shortness of breath, which was actually out of proportion to the physical findings, and the CD4 count was more than 250cells. we found that there was no single predictor of pulmonary hypertension.

The echocardiographic picture was dilated right ventricle and dilated right atrium with prominent D-sign on the parasternal short-axis view. Main pulmonary artery was also dilated in most cases.

The prognosis of these patients isusually poor All the findings in our study in relation to corpulmonale correlate well with previous research studies in this area.

HIV and Myocarditis

DCM is a documented cardiac abnormality in HIV patients. Patients with DCM had more advanced immunosuppression. In other words dilated cardiomyopathy in HIV was associated with advanced immunosuppression and lower CD4 cells.

Myocarditis and direct HIV invasion of myocardial tissue are the most common causes of dilated cardiomyopathy in HIV patients.

In our study

HIV patients with myocarditis presented with younger age group, lower systolic BP, lower diastolic BP, higher mean resting pulse rate, higher white blood cell, lower CD4 cell counts and symptoms of breathlessness and chest pain.

All the findings in our study in relation to myocarditis

Correlate well with previous research studies in this area, especially the low CD4 count, which is an excellent predictor of the presence of LV dysfunction. ART and proper nutrition has reduced the incidence of myocarditis. They improve survival and enhance quality of life

HIV and Pericardial effusion

In our study

It was found that pericardial effusion was commonly seen in our HIV patients, with a pattern ranging from asymptomatic effusions that are mild to severe pericardialeffusion.

Pericardial disease is the most common cardiovascular Presentation of HIV infection and it was found to be independent of CD4 count and serum albumin levels.

As the incidence of HIV infection is on the rising trend, pericardial effusion and its sequential complications may become a major cardiac problem in future in HIV people.

Pericardiocentesis is not possible in most of these patients because most of the detected effusions are small and even when indicated for the relief of tamponade, its diagnostic accuracy was found to be low.

In our study

HIV patients with pericardial effusion presented with younger age group, higher white blood cell, higher CD4 cell counts and symptoms of palpitations, breathlessness and chest pain.

All the findings in our study in relation to pericardial effusion correlate well with previous research studies in this area. HIV patients have an increased risk for pericardial effusions.

The development of pericardial effusion predicts a poor prognosis. This is because end stage HIV infection usually affects the pericardium through malignancies and infections.

HIV and Systolic Dysfunction

In HIV patients, studies show that systolic dysfunction is present in about one third of the cases in echocardiography studies . There has been a significant reduction in myocardial contractility .

As the disease become more and more severe the myocardial dysfunction was found to be more .

The decrease in systolic function has been found to be the cause of increased mortality and morbidity in hiv patients and cardiac failure has been found to occur in about 5 percent of cases.

Hence it is very important to screen for early diagnosis of systolic dysfunction to increase the life span of the patients.

In our study

HIV patients with systolic dysfunction presented with lower systolic BP, higher haemoglobin level, higher white blood cell, lower serum creatinine, higher CD4 cell counts and symptoms of palpitations, breathlessness and chest pain. Usually systolic dysfunction is associated with low CD4 count.

But in our study it does not tally with previous research studies. All the findings in our study in relation to systolic dysfunction correlate well with previous research studies in this area

HIV and Diastolic Dysfunction

The prevalence of diastolic dysfunction is found to be 30 percent in studies.

In HIV patients the ventricles became more and more non compliant and ventricular filling defects were noted in echocardiography signalling diastolic dysfunction. The dysfunction was found to be more severe in patients with advanced disease.

In our study

HIV patients with higher CD4 cell counts and symptoms of palpitations, breathlessness and chest pain. Diastolic dysfunction is alsoassociated with low CD4 count. Again here it does not tally.

Diastolic dysfunction may be the first indication of underlying cardiac disease in HIV. All the findings in our study in relation to diastolic dysfunction correlate well with previous research studies in this area.

Traditional risk factors also contribute to development of Diastolic dysfunction. Asymptomatic diastolic dysfunction is common among HIV infected individuals. HIV infection and Zidovudine increase the risk of cardiac diastolic dysfunction.

The symptoms of cardiac disease like of palpitations, breathlessness and chest pain were considerable in our study subjects.

They could be attributable to pulmonary disorders.

Patients with HIV infection are known to develop multiple pulmonary opportunistic infections.

All the patients with cardiac disease had anaemia. This may be due to advanced stage in HIV disease. Cardiac function will also be worsened by anaemia.

CONCLUSIONS

- 82% of the patents were males and 18% were females
- Majority of the patients are in the age group 41-50 years
- The male female ratio is 4.5:1
- 32% of the patients were confirmed with cardiac disease
- 45.5% of the patients had CD4 count less than 200
- 91% were on Anti Retroviral Therapy
- 8% of the males had the disease beyond 3 years
- 53.5% of the patients were in the WHO HIV clinical stage 3
- Most common echocardiographic diagnosis was myocarditis, pericardial

  effusion, systolic dysfunction and diastolic dysfunction, each at 7% respectively
- Smoking status correlated with HIV and coronary artery disease
- Low CD4 count correlated with HIV and myocarditis

LIMITATIONS

- Small sample size
- Confounding factors like traditional risk factors of cardiac disease
- Causal relationship cannot be established
- Other predictors like viral load not studied

SUMMARY

- Cardiac disorders in HIV infected individuals are common
- Non-invasive investigations like echocardiography helps in early diagnosis of asymptomatic cardiac disease in HIV infected individuals
- ART improves clinical outcomes in HIV infected individuals with cardiac disease

RECOMMENDATIONS

- Large prospective studies are needed in order to confirm the observation in this study

- Clinicians should be vigilant in addressing known risk factors for coronary disease in their patients with HIV infection and also should be aware of the various cardiovascular manifestations of HIV infection

- CVD risk assessment and reduction measures should be implemented effectively especially in HIV infected individuals

- Lifestyle modification, including smoking cessation, increased physical activity, weight reduction for those who are overweight orobese, and education on healthy dietary practices should form part of activities of support groups tending to HIV infected individuals

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PROFORMA

S.No./ART No.

Name :

Age/Sex :

Occupation :

Complaints : Present /Absent Duration Grade

Chest Pain :

Breathlessness :

Palpitation :

Pedal edema /

Abdominal distention :

Past History :

DM : Yes / No

SHT : Yes / No

Personal History :

Smoker : Yes/ No

Alcoholic : Yes / No

Clinical Findings :

Stage of Disease :

Duration since diagnosis :Whether on ART : Yes / No

INV:

CBC :Hb	TC	ESR	Plt count	
Blood sugar	RBS	FBS	PPBS	
Urea	**Creatinine**			
LFT	T.B.	T.Prot		
	SGOT	SGPT	SAP	
Lipid profile	TC VLDL	TGL	LDL	HDL
Urine Alb	Sugar	deposits		
CD_4 count				
ECG				
CXR – PA				
ECHO	N/Dilated			
RA				
RV				
LA		EF :		
LV				
Pulm. Artery				
RV Dys.function	Present / Absent			
LV Dys.Function	Present / Absent			
RWMA	Present / Absent		Site	
LVH	LVDD		I/II/II/IV	
Effusion	Mild/Mod./severe			
Valves	AV	PV	TRPG	
	MV	TV	PHT	Mild/Mod/Sever

REPORT:

ABBREVIATION

AIDS	-	Acquired immunodeficiency syndrome
HIV	-	Human Immunodeficiency Virus
ART	-	Anti Retroviral Therapy
PI	-	Protease Inhibitors
SHT	-	Systemic Hypertension
DM	-	Diabetes mellitus
ECG	-	Electrocardiogram
DCM	-	Dilated cardiomyopathy
EF	-	Ejection Fraction
PHT	-	Pulmonary Hypertension
PE	-	Pericardial Effusion
RA	-	Right atrium
RV	-	Right ventricle
LA	-	Left atrium
LV	-	Left ventricle
IVS	-	Interventricular septum
DD	-	Diastolic Dysfunction
NCEP	-	National Cholesterol Eradication Program
CBC	-	Complete blood count

EBV	-	Ebstein barr virus
FBS	-	Fasting Blood Sugar
RFT	-	Renal function test
LFT	-	Liver function test
ECHO	-	Echocardiogram
CXR	-	Chest x ray
LVH	-	Left ventricular hypertrophy
RWMA	-	Regional wall motion abnormality
TRPG	-	Tricuspid regurgitation peak gradient

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	: Echocardiographic findings in HIV infected patients at Govt.Stanley Hospital, Chennai
Principal Investigator	: Dr.S.Dhanraj
Designation	: PG in MD (Gen.Med)
Department	: Department of General Medicine Government Stanley Medical College, Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.04.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

25/11/13

CONSENT FORM

1) I AGREE TO PARTICIPATE IN STUDY TITLED"ECHOCARDIOGRAPHIC DIAGNOSIS IN HIV POSITIVE PATIENTS"

2) I CONFIRM THAT I HAVE BEEN TOLD ABOUT THIS STUDY IN MY MOTHER TONGUE & HEVE HAD THE OPPURTUNITY TO ASK QUESTIONS

3) I UNDERSTAND THAT MY PARTICIPATION IS VOLUNTARY & I MAY REFUSE TO PARTICIPATE AT ANY TIME WITHOUT GIVING ANY REASON AND WITHOUT AFFECTING MY BENEFITS

4) I AGREE NOT TO RESTRICT THE USE OF ANY DATD OR RESULTS THAT ARISE FROM THE STUDY

5) I AGREE TO DO ECHOCARDIOGRAM

NAME OF PARTICIPANT: SIGN/THUMB PRINT

INVESTIGATOR:

S.No	ART.No	Age(yrs)	SEX	Symptom	BP mmHg	P.Rate	DM	HT	Smoking	Alcohol	Durati	Stage	On ART	Hb	TC	ESR	Pl.count	FBS	PPBS
1	987	52	m	SOB	108/72	120	n	n	y	n	4	4	y	9.8	13000	41730	1.6	98	122
2	788	38	F	SOB	110/76	116	N	N	N	N	3	3	Y	8	12880	5/18	1.8	76	110
3	664	50	M	SOB	110/80	112	N	N	Y	N	3	3	Y	9	11890	3/16	1.3	86	118
4	880	45	M	SOB,CHES	114/76	122	N	N	Y	Y	1	3	Y	9.9	13520	10/18	1.65	86	135
5	1022	42	M	CHEST	120/86	114	N	N	Y	N	1	3	Y	7	6540	2/14	1.9	94	138
6	998	48	M	SOB,PALP	118/72	110	N	N	Y	Y	2	4	Y	6.8	13220	5/15	1.99	90	132
7	1210	35	M	CHEST	112/68	120	N	N	Y	N	3	3	Y	7.5	12220	3/12	2.03	94	138
8	1140	38	M	CHEST	110/70	122	N	N	Y	Y	1	3	Y	8.2	11205	6/15	1.86	90	125
9	1056	44	M	PALITATI	108/68	114	N	N	Y	N	2	4	Y	7.8	13542	5/12	1.78	88	128
10	1145	40	M	SOB,	110/72	124	N	N	Y	Y	1	3	Y	8.5	13587	14/19	1.85	94	134
11	1123	40	M	SOB,	108/68	120	N	N	N	N	2	4	Y	9	11203	8/19	1.68	85	124
12	665	38	F	PALPITATI	100/68	112	N	N	N	N	1	3	Y	7.5	11100	10/19	1.72	94	125
13	884	40	M	CHEST	102/70	110	N	N	Y	N	2	4	Y	7.2	11420	11/22	1.85	90	130
14	990	42	M	CHEST	110/68	115	N	N	Y	Y	3	3	Y	9.5	12405	6/16	1.9	88	124
15	1150	40	M	SOB,CHES	114/80	120	N	N	Y	N	2	4	Y	11	12500	4/12	1.99	99	139
16	716	51	M	SOB,PALP	120/80	106	N	N	Y	Y	3	2	Y	8.8	11800	6/16	1.4	82	112
17	845	48	F	Sob	90/70	100	N	N	N	N	2.5	3	Y	9	11980	12/20	1.65	78	124
18	644	34	M	Chest	110/70	98	N	N	N	Y	2	2	Y	11	11200	5/15	2	90	118
19	978	30	F	Sob	100/62	100	N	N	N	N	2	2	Y	10	10400	4/12	1.8	80	126
20	888	46	M	Sob,palpit	110/80	102	N	N	Y	Y	3	3	y	9.2	11600	8/20	2	69	114
21	1990	40	M	Sob,chest	108/78	98	N	N	Y	N	3	1	Y	10	11000	3/12	1.6	80	120
22	1880	38	m	PALPITATI	110/80	102	n	n	y	y	3	3	y	9	10500	8/20	1.8	80	120
23	676	53	m	chest pain	120/70	100	n	n	y	y	4	4	y	9.9	6700	11/20	2	86	99
24	41266	32	f	sob	110/70	98	n	n	n	n	6 mon	3	n	9.2	5800	10/22	2.5	74	122
25	1668	30	f	SOB,CHES	100/62	88	n	n	n	n	2	3	y	10	4400	9/22	2.2	60	122
26	1224	43	m	PALPITATI	122/78	78	n	n	y	n	2	3	y	9	5800	11/20	2	86	98
27	1540	48	m	PALPITATI	120/70	80	n	n	y	y	3	4	y	8.5	6040	9/15	2.05	90	134
28	1684	42	m	sob chest	112/68	84	n	n	y	n	2	3	y	8.6	5700	8/15	2.12	84	125
29	1558	45	m	sob	114/72	90	n	n	y	n	3	4	y	9.3	6700	7/16	1.95	75	125
30	1654	48	m	chest pain	118/76	74	n	n	y	y	2	4	y	9.2	5014	6/12	1.82	68	124
31	994	39	f	chest pain	112/70	82	n	n	n	n	2	3	y	9	4700	5/14	1.75	70	120
32	887	42	m	sob	110/66	80	n	n	y	n	3	4	y	9.9	5200	6/16	1.98	75	125
33	942	43	m	chest pain	112/68	74	n	n	y	n	2	3	y	9.4	6000	5/12	2.09	74	128
34	1240	41	m	PALPITATI	110/68	70	n	n	y	n	2	3	y	9.1	5120	4/12	1.98	71	124
35	1354	43	m	sob	n	88	n	n	y	y	2	4	y	8.4	4215	5/16	1.84	68	114
36	1264	48	m	sob	116/78	90	n	n	y	n	2	3	y	8.2	4521	10/30	1.82	70	112
37	1250	45	m	chest pain	114/68	84	n	n	y	n	3	3	y	8.1	4687	12/28	1.7	82	124
38	1397	41	f	palpitatio	120/68	75	n	n	y	y	3	4	y	7	5023	14/48	1.64	88	130
39	1640	42	m	sob	124/74	65	n	n	y	n	2	3	y	9.2	4021	13/39	1.98	90	126
40	1452	48	m	chest pain	112/68	80	n	n	y	n	3	3	y	8.4	4210	10/42	1.84	160	230
41	1356	50	m	palpitatio	120/72	84	n	n	y	n	2	4	y	9	4702	12/45	1.72	175	255

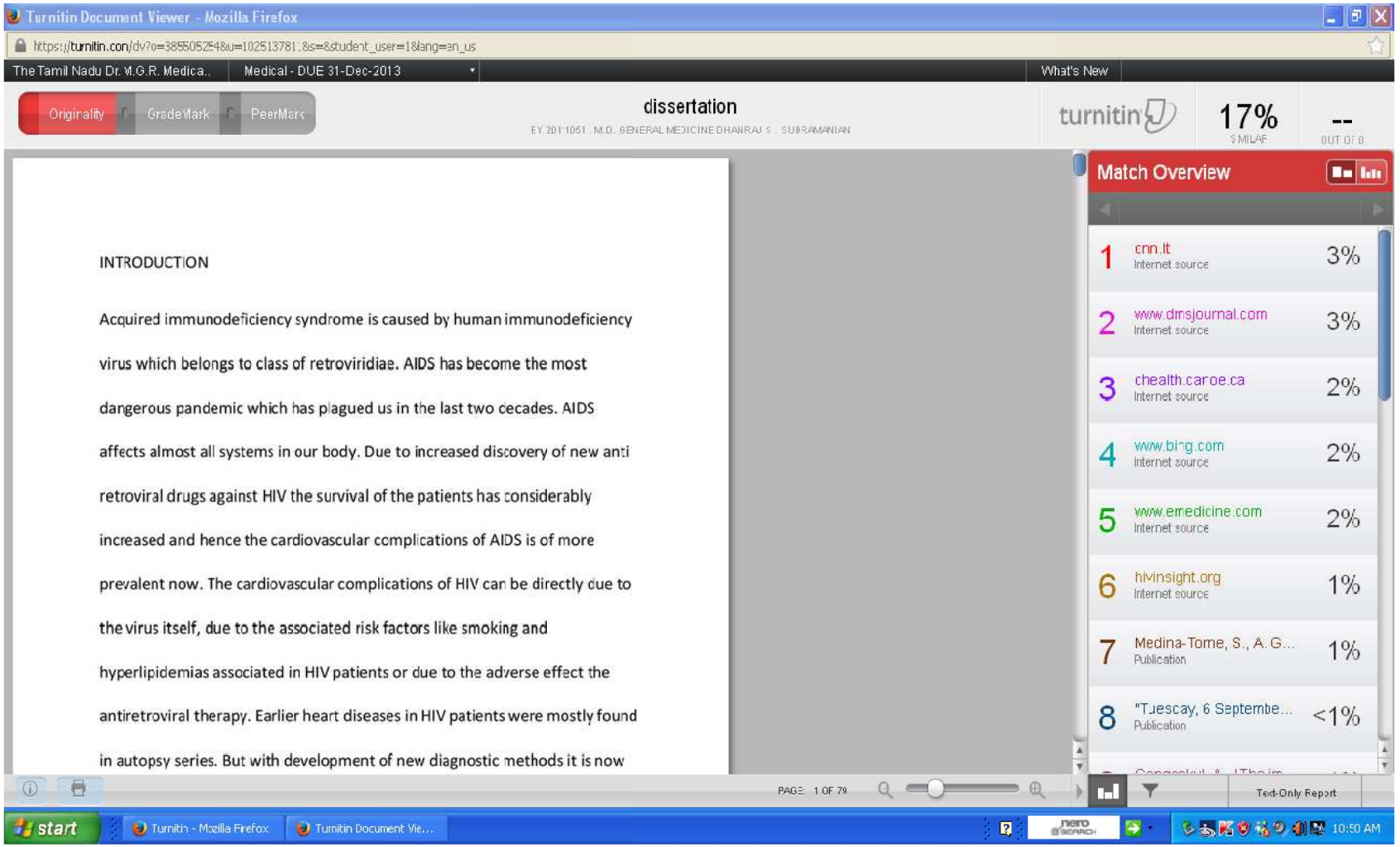
u-aib	U- sug	u-dep	TotBil	t.prot	SGOT(I	SGPT(I/	SAP(I/U	Urea(m	Creat(	T.cl	TGL	LDL	HDL	VLDL	CD4	CXR	ECG	USG	ECHO
nl	nl	3-4 PUS	1	6	43	40	65	36	1.1	190	140	112	40	38	252	MULTILO	SINUS	N study	SEVERE
Nl	Nl	1-2 pus	0.8	5.6	29	48	76	40	1	180	138	118	38	30	275	MULTILO	SINUS	N	MODERA
Nl	Nl	3 -5 pus	0.7	5.9	42	56	80	28	0.8	186	138	110	36	28	290	RIGHT	SINUS	N	SEVERE
Nl	Nl	2-3 pus	0.9	6.5	30	40	86	25	0.7	195	150	120	40	35	220	MULTILO	SINUS	N	SEVERE
Nl	Nl	3-5 pus	1.1	6.9	58	44	90	30	0.9	200	156	116	38	45	400	MULTILO	SINUS	N	SEVERE
Nl	Nl	1-2 pus	1.2	5.5	30	24	88	26	1.1	194	144	124	42	54	354	MULTILO	SINUS	N	MODERA
NL	NL	3-5 PUS	1	5.8	26	38	74	24	1.2	186	160	120	35	52	115	CARDIO	SINUS	N	GLOBAL
NL	NL	2-4 PUS	0.8	6	35	40	72	28	1	190	154	118	38	45	100	CARDIO	SINUS	N	GLOBAL
NL	NL	3-4 PUS	0.9	5.2	38	28	98	24	0.8	195	152	110	32	40	90	NORMAL	SINUS	N	GOLBAL
NL	NL	3-5 PUS	0.7	5.5	30	25	70	21	0.6	184	148	105	30	35	95	CARDIO	SINUS	N	GLOBAL
NL	NL	2-4 PUS	0.8	5.3	42	36	68	20	0.9	178	135	102	28	30	74	NORMAL	SINUS	N	GLOBAL
NL	NL	2-4 PUS S	0.9	5	30	28	86	29	0.7	185	128	100	35	35	65	CARDIO	SINUS	N	GLOBAL
NL	NL	3-5 PUS	0.8	5.2	45	45	90	21	0.9	190	110	108	40	32	104	CARDIO	SINUS	n	GLOBAL
NL	NL	2-5 PUS	0.7	5.6	42	34	86	25	1.1	188	114	100	35	38	96	CARDIO	SINUS	N	GLOBAL
NL	NL	3-5 PUS	1	6	48	29	78	21	1	180	124	108	48	40	100	CARDIO	SINUS	N	GLOBAL
Nl	Nl	2 -5 pus	1.1	4.5	44	48	92	29	1.1	190	120	115	50	38	38	CARDIO	SINUS	N	GLOBAL
Nl	Nl	2-3 pus	1	5.9	29	46	85	33	1	186	112	108	46	40	340	N	T	N	Mild
Nl	Nl	Occ.pus	0.9	6	43	45	72	42	0.6	180	108	102	45	38	180	N	LOW	N	Mod
Nl	Nl	pus&	1	5.5	30	56	78	40	0.9	190	112	106	38	44	200	MILD	LOW	N	Mod
Nl	Nl	Few pus	0.8	5.8	18	24	88	38	1	188	112	100	36	40	400	N	N	N	Mild
Nl	Nl	Occ.ep	0.8	5.5	26	38	96	43	0.6	184	106	102	47	42	435	N	T	N	Mild
nl	nl	1-2 pus	1	5.9	20	25	80	40	0.8	190	140	90	39	36	525	N	N	N	Mild
nl	nl	occ. Pus	0.9	6	34	46	122	28	0.9	208	150	122	43	50	440	N	T	n	mild
nl	nl	3-4 PUS	1	5.8	44	23	66	34	0.6	180	142	110	49	44	584	N	N	n	mild
nl	nl	2-3 pus	0.9	6.2	38	24	86	26	0.5	198	148	114	52	40	422	N	T	n	mild
nl	nl	Occ.pus	0.8	5.8	40	45	90	30	0.6	194	134	98	32	46	400	N	n	n	mild
nl	nl	3-5 pus	1.1	6.2	44	56	84	25	0.9	300	147	112	45	41	554	N	T	n	mild
nl	nl	2-4 PUS	1	5.9	38	27	75	28	0.8	180	130	105	32	49	660	N	n	n	mild
nl	nl	3-5 pus	0.9	6.4	34	24	68	22	1	195	145	110	40	42	604	N	n	n	grade 1
nl	nl	2-4 PUS	1.1	5.2	21	29	92	30	1.1	198	138	114	42	45	590	N	n	n	grade 2
nl	nl	3-5 pus	0.8	5	34	42	78	23	0.9	175	132	102	34	44	640	N	n	n	grade 2
nl	nl	2-5 PUS	1	5.5	15	24	82	21	1.1	165	135	106	42	46	606	N	n	n	grade 2
nl	nl	3-5 pus	1.1	5.9	38	27	72	24	0.8	260	145	120	41	43	550	N	n	n	n
nl	nl	2-4 PUS	0.9	5.2	43	36	68	22	0.9	240	142	115	35	48	664	N	n	n	n
nl	nl	3-5 pus	1.1	6	34	28	78	25	1.1	258	134	118	24	42	540	N	n	n	n
nl	nl	2-4 PUS	0.9	5.1	20	28	54	22	1	180	210	102	32	41	490	N	n	n	grade 2
nl	nl	3-5 pus	0.7	4.5	22	24	62	23	0.8	170	198	124	35	45	402	N	n	n	n
nl	nl	2-4 PUS	0.8	5.4	28	26	80	27	0.9	154	221	114	28	48	442	N	n	n	grade 2
nl	nl	2-5 PUS	1	5.2	14	30	72	29	0.7	168	181	108	45	50	542	N	n	n	n
nl	nl	2-5 PUS	0.8	5.1	20	24	64	24	0.8	210	135	112	35	42	435	N	n	n	n
nl	nl	3-5 pus	0.9	4.9	18	28	78	26	0.9	225	125	120	38	48	443	N	n	n	n

42	1520	45	m	palpitatio	160/100	90	n	n	n	n	3	4	y	9.2	5210	10/40	1.9	90	130
43	1245	44	m	giddiness	170/112	78	n	n	n	n	2	4	y	8.4	5640	14/42	1.72	88	124
44	1365	50	m	chest	120/90	74	n	n	y	y	2	3	y	8.6	5710	12/36	1.8	90	125
45	1345	48		chest	88/60	85	n	n	y	y	3	4	y	8.2	5420	14/42	2.05	82	135
46	1268	42		chest	124/88	95	n	n	y	y	2	3	y	9.2	6021	13/36	1.92	84	134
47	776	44	M	CHEST	110/70	100	N	N	Y	Y	2	3	Y	6	5400	22/30	1	89	122
48	791	40	M	SOB	120/78	78	N	N	Y	Y	1	3	Y	8.8	4800	14/28	1.6	95	118
49	565	49	M	PALPITATI	122/78	88	N	N	Y	Y	2	3	Y	9	6420	18/28	1.5	73	132
50	899	42	F	SOB,CHES	120/80	92	N	N	N	N	3	3	Y	8	5300	20/36	2	85	124
51	5781	44	M	PALPITATI	130/68	82	N	N	Y	Y	2	3	Y	9	6000	20/30	2.4	86	112
52	1003	58	M	SOB,PALPI	108/78	78	N	N	Y	Y	3	3	Y	9	7800	22/26	0.6	90	108
53	1333	59	M	CHEST	114/82	76	N	N	Y	Y	3	4	Y	8.6	6800	22/30	1.2	100	126
54	989	46	M	PALPITATI	120/80	86	N	N	Y	Y	2	4	Y	9	4000	8/20	1.4	86	132
55	1445	44	M	CHEST	110/70	86	N	N	Y	Y	2	3	Y	8	3200	11/22	2	88	140
56	1345	46	M	SOB,	120/70	90	N	N	Y	Y	1	3	Y	7	4600	16/22	1.3	78	128
57	1277	40	M	CHEST	130/80	82	N	N	Y	Y	2	4	Y	9	300	10/18	1.7	104	118
58	789	34	M	SOB	110/70	86	N	N	Y	Y	1	3	N	8	5500	14/32	1.8	90	120
59	795	46	M	SOB	120/60	86	N	N	Y	Y	2	3	Y	9	7000	14/28	1.1	86	138
60	963	41	M	PALPITATI	130/70	78	N	N	Y	Y	3	3	Y	7	4300	16/32	1.5	76	136
61	1046	42	M	PALPITATI	122/78	78	N	Y	N	N	4	4	N	8	4750	14/26	1	86	134
62	712	43	M	SOB,PALPI	120/70	76	N	N	Y	Y	3	3	Y	7.5	4300	14/42	1	60	118
63	863	45	M	SOB	114/80	84	N	N	N	N	2	3	Y	8	2500	20/40	1.8	66	140
64	1645	34	F	CHEST	118/70	78	N	N	N	N	1	2	N	10	3100	22/44	2	80	136
65	964	46	M	SOB,PALPI	120/68	90	N	N	N	Y	2	3	Y	8	4900	14/34	1	72	120
66	667	40	M	Sob,palpit	132/74	90	N	N	Y	Y	3	4	Y	6	4600	10/22	0.6	88	128
67	1994	50	M	Sob,chest	120/80	86	N	N	Y	Y	1	3	Y	8	4400	8/18	1.8	92	132
68	1763	48	M	PALPITATI	110/80	78	N	N	N	N	2	3	Y	9	2800	4/16	2	100	120
69	456	44	F	SOB	130/70	76	N	N	N	N	1	2	Y	8	3600	2/20	2	88	118
70	34122	52	M	CHEST	120/66	76	N	N	Y	Y	2	3	Y	7.8	4000	2/12	1	90	122
71	2443	46	M	SOB,CHES	118/76	84	N	N	N	N	3	4	Y	9	2900	32/48	1.2	86	124
72	1337	50	M	CHEST	120/80	88	N	N	Y	Y	1	3	Y	9	6800	14/36	0.9	92	232
73	1435	44	M	SOB	114/78	84	N	N	Y	Y	1	3	Y	7	4500	6/24	1.3	98	126
74	1686	51	M	sob chest	120/78	88	N	N	N	N	2	3	Y	8	2400	10/20	1	96	124
75	1598	45	M	sob	120/88	92	N	N	Y	Y	3	4	Y	8	4550	11/20	1.6	94	138
76	1788	48	F	PALPITATI	120/86	90	N	N	N	N	2	3	Y	7	4900	10/19	1.75	78	116
77	897	36	M	SOB	120/80	92	N	N	Y	Y	2	3	Y	9	2300	16/32	1.6	110	122
78	899	52	M	CHEST	110/66	90	N	N	N	N	2	3	Y	9	3700	14/34	2	84	110
79	967	44	M	chest pain	110/70	88	N	N	Y	Y	3	3	Y	8	4100	16/38	1.1	92	122
80	1117	47	F	PALPITATI	124/74	74	N	N	N	N	2	3	Y	9	5200	11/32	1.9	84	130
81	1269	43	F	sob	122/76	78	N	N	N	N	3	2	Y	6	5000	24/38	1.8	102	124
82	1467	42	F	CHEST	128/74	74	N	N	N	N	2	4	Y	7	4400	20/30	1	78	132
83	1279	36	M	chest pain	110/80	68	N	N	Y	Y	2	3	Y	8.8	3000	15/32	1	96	122

nl	**nl**	**2-4 PUS**	**1.1**	**5.4**	**17**	**25**	**82**	**22**	**1.1**	**190**	**122**	**112**	**35**	**40**	**542**	**N**	**n**	**n**	**n**
nl	**nl**	**3-5 pus**	**0.9**	**5.6**	**16**	**28**	**54**	**28**	**0.9**	**184**	**130**	**102**	**40**	**50**	**461**	**n**	**n**	**n**	**n**
nl	**nl**	**2-4 pus**	**0.8**	**6.1**	**14**	**28**	**60**	**26**	**1**	**182**	**142**	**114**	**34**	**42**	**488**	**n**	**n**	**n**	**REGIONA**
nl	**nl**	**3-5 pus**	**0.7**	**5.9**	**16**	**25**	**64**	**24**	**0.8**	**174**	**138**	**124**	**36**	**46**	**523**	**n**	**n**	**n**	**REGIONA**
nl	**nl**	**2-5 PUS**	**1.1**	**5.4**	**19**	**29**	**72**	**25**	**0.7**	**165**	**136**	**106**	**42**	**45**	**497**	**n**	**n**	**n**	**REGIONA**
nl	nl	1-4 PUS	**1.2**	**6.4**	**32**	**38**	**86**	**23**	**1**	200	144	114	32	40	**180**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	1-2 pus	**0.8**	**5.9**	**34**	**46**	**74**	**34**	**1**	194	142	116	40	42	**1466**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	2-5 pus	**0.6**	**5.5**	**34**	**42**	**84**	**22**	**1**	188	138	108	42	48	**156**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	2-3 pus	**1**	**6**	**36**	**50**	**82**	**28**	**0.6**	184	136	118	36	44	**156**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	2-5 pus	**1**	**6.2**	**42**	**46**	**88**	**22**	**0.8**	190	140	120	40	48	**232**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	1-2 pus	**0.9**	**5.8**	**38**	**44**	**86**	**24**	**1**	170	132	104	38	52	**122**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	2-54 PUS	**1**	**5**	**40**	**48**	**98**	**30**	**1**	186	112	108	46	40	**86**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	2-4 PUS	**1.2**	**5.6**	**36**	**46**	**82**	**24**	**1**	180	108	102	45	44	**192**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	1-4PUS	**0.8**	**6.6**	**40**	**44**	**88**	**20**	**1**	192	112	106	28	44	**232**	**RBBB**	**N**	**N**	**N STUDY**
NL	NL	3-5 PUS	**1**	**6.2**	**42**	**58**	**98**	**32**	**1**	188	102	100	36	40	**198**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	2-5 PUS	**1.2**	**6.2**	**42**	**46**	**88**	**46**	**1**	184	106	112	46	42	**67**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	2-4 PUS S	**1**	**6**	**36**	**44**	**82**	**24**	**1**	190	142	90	43	36	**232**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	3-5 PUS	**1.2**	**5**	**30**	**42**	**76**	**34**	**1.2**	168	130	122	42	50	**188**	**NQRS**	**N**	**N**	**N STUDY**
NL	NL	OCC. PUS	**0.8**	**6**	**43**	**48**	**102**	**22**	**1**	184	142	110	48	44	**178**	**QRS**	**N**	**N**	**N STUDY**
NL	NL	3-5 PUS	**1**	**6.2**	**42**	**54**	**86**	**32**	**1.1**	198	148	116	51	40	**343**	**QRS**	**N**	**N**	**N STUDY**
Nl	Nl	1 -5 pus	**0.6**	**5.8**	**38**	**44**	**82**	**18**	**1.1**	194	134	98	32	46	**188**	**NQRS**	**N**	**N**	**N STUDY**
Nl	Nl	2-3 pus	**1**	**5.8**	**44**	**48**	**98**	**20**	**1.2**	174	144	112	44	41	**177**	**NQRS**	**N**	**N**	**N STUDY**
Nl	Nl	Occ.pus	**1.2**	**5.5**	**48**	**48**	**118**	**32**	**0.8**	188	140	108	46	38	**330**	**NQRS**	**N**	**N**	**N STUDY**
Nl	Nl	pus&	**1.1**	**5.2**	**46**	**42**	**88**	**18**	**0.8**	193	138	110	48	40	**134**	**NQRS**	**N**	**N**	**N STUDY**
Nl	Nl	Few pus	**0.8**	**6**	**44**	**58**	**98**	**25**	**1.2**	200	150	122	50	48	**166**	**NQRS**	**N**	**N**	**N STUDY**
Nl	Nl	Occ.ep	**0.8**	**6.2**	**50**	**48**	**96**	**42**	**1**	196	146	118	34	38	**222**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	few pus	**1.1**	**5.8**	**46**	**48**	**82**	**44**	**0.8**	178	128	98	34	40	**178**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	occ. Pus	**1.2**	**6.2**	**58**	**58**	**88**	**48**	**1.2**	186	148	116	38	44	**198**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-4 PUS	**0.8**	**6**	**48**	**62**	**94**	**32**	**1**	198	110	100	36	40	**244**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-3 pus	**1.2**	**5.8**	**52**	**44**	**88**	**18**	**1.2**	182	144	8	43	44	**196**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	Occ.pus	**1**	**5.8**	**46**	**64**	**107**	**32**	**0.8**	194	140	118	38	42	**298**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-5 pus	**0.8**	**5.5**	**48**	**60**	**88**	**50**	**0.8**	176	128	108	32	36	**136**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-4 PUS	**1**	**5.8**	**48**	**50**	**82**	**20**	**1**	186	138	114	36	38	**138**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	3-4 pus	**1.2**	**6**	**44**	**62**	**86**	**20**	**0.8**	190	130	110	44	48	**311**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-4 PUS	**0.8**	**6**	**44**	**62**	**98**	**46**	**0.8**	192	134	114	46	50	**122**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	3-4 pus	**1**	**5.4**	**56**	**54**	**104**	**16**	**1.1**	196	132	108	48	52	**128**	**QRS**	**N**	**N**	**N STUDY**
nl	nl	2-5 PUS	**0.8**	**6.2**	**48**	**42**	**86**	**42**	**1**	188	138	98	28	34	**342**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-5 pus	**1.2**	**6.2**	**48**	**66**	**107**	**34**	**1.2**	186	122	92	32	33	**108**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-4 PUS	**0.8**	**6.6**	**48**	**66**	**107**	**43**	**2**	180	134	88	34	38	**222**	**RBBB**	**N**	**N**	**N STUDY**
nl	nl	1-2 pus	**1.2**	**6**	**44**	**68**	**118**	**18**	**1.3**	190	144	102	42	42	**190**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	2-4 PUS	**0.8**	**5.8**	**36**	**64**	**82**	**54**	**0.6**	168	126	80	46	36	**50**	**NQRS**	**N**	**N**	**N STUDY**
nl	nl	3-5 pus	**1.2**	**6.4**	**32**	**48**	**107**	**26**	**0.8**	178	146	86	30	42	**146**	**QRS**	**N**	**N**	**N STUDY**

84	1222	48	F	palpitatio	110/70	66	N	N	N	N	3	4	Y	9	5110	12/32	2.1	106	108
85	1871	54	M	sob	120/78	76	N	N	Y	Y	3	4	Y	8	3600	10/36	2.2	84	114
86	1434	34	M	chest pain	112/76	74	N	N	Y	Y	3	4	Y	8	6200	8/30	1	98	134
87	1663	45	M	CHEST	110/68	76	N	N	Y	Y	3	4	Y	8.5	5500	14/26	1.3	86	122
88	669	46	M	palpitatio	120/78	78	N	N	Y	Y	2	3	Y	8	6600	16/34	1.5	96	128
89	1358	44	M	giddiness	130/78	82	N	N	Y	Y	2	4	Y	8.4	2600	10/32	1.4	84	121
90	1398	54	M	SOB,	130/72	80	N	N	Y	Y	2	4	Y	10	3100	4/12	2	96	115
91	1321	56	M	chest	120/80	80	N	N	Y	Y	3	4	Y	5	4200	14/22	1	84	119
92	1765	45	F	CHEST	110/70	82	N	N	N	N	1	3	Y	7	4400	18/34	1	82	125
93	344	58	M	SOB	120/80	86	N	N	Y	N	2.5	4	Y	9	2600	16/36	1.4	110	131
94	568	44	F	PALPITATIO	110/70	96	N	N	N	N	3	4	Y	8	6550	14/28	1.6	86	133
95	703	39	M	CHEST PAI	120/80	88	N	N	Y	Y	2	3	Y	6.5	5800	16/32	0.7	96	129
96	808	41	M	SOB,PALPI	122/66	80	N	N	Y	Y	1	3	Y	8	7100	14/34	1.4	86	115
97	673	42	M	PALPITATIO	118/80	70	N	N	N	N	1	3	N	9	7500	10/24	2	104	123
98	913	43	M	SOB	122/78	72	N	N	Y	Y	2	3	Y	7	6700	14/20	2	96	115
99	919	39	M	CHEST PAI	120/70	74	N	N	Y	Y	3	4	Y	7.5	5800	8/28	1	88	139
100	932	49	M	CHEST PAI	120/70	74	N	N	N	Y	1	3	Y	8.2	4800	20/30	1.5	96	118
101	835	41	F	SOB	110/80	78	N	N	N	N	2	4	Y	8	4900	10/34	1	107	118
102	934	48	F	PALPITATIO	120/60	72	N	N	N	N	2	2	N	9	3400	14/28	1.8	86	121
103	469	52	M	PALPITATIO	124/78	94	Y	N	N	N	2	3	Y	7.6	5600	14/30	1.2	84	125
104	474	50	F	CHEST PAI	130/82	84	N	N	N	N	3	4	Y	6.5	6000	18/38	1.9	80	129
105	1593	51	M	CHEST PAI	128/80	80	N	N	N	N	3	3	Y	8	4200	12/26	2.5	96	135
106	1853	44	M	SOB	120/80	86	N	Y	Y	Y	3	2	N	8	8200	2/10	1.5	86	118
107	1661	42	M	PALPITATIO	110/70	80	N	N	Y	Y	4	4	Y	7	6200	20/44	1.2	78	140
108	775	43	M	SOB	120/66	82	N	N	Y	Y	3	3	Y	8.8	3400	4/12	1	82	121
109	894	54	M	CHEST PAI	120/84	84	N	N	Y	Y	4	3	Y	7.8	4300	16/28	1.6	88	114
110	566	36	M	PALPITATIO	118/80	78	N	N	Y	Y	4	4	Y	7.2	3100	20/34	2	110	130
111	779	56	M	CHEST PAI	120/68	76	N	N	N	N	4	3	Y	8	2200	16/32	1.1	90	126
112	580	45	M	SOB,PALPI	128/76	78	N	N	Y	Y	3	4	Y	9	4200	15/30	1.5	96	116
113	660	39	M	SOB	120/80	66	N	N	Y	Y	5	4	Y	10	2000	12/26	2	110	134
114	2221	45	M	PALPITATIO	130/80	84	N	N	Y	99	3	3	y	6	2300	10/28	2.3	86	128
115	3234	63	M	PALPITATIO	120/74	98	N	N	N	Y	2	3	Y	5.3	5000	14/28	1.2	88	106
116	642	44	M	CHEST PAI	110/82	84	N	N	Y	Y	1	3	Y	5.4	5000	12/34	0.9	78	124
117	560	44	M	SOB,PALPI	124/76	78	N	N	Y	Y	3	4	Y	9	4500	15/30	1.5	96	112

nl	nl	1-4 PUS	0.8	6	44	44	82	18	1.3	186	132	94	32	48	270	NQRS	N	N	N STUDY
nl	nl	2-5 PUS	1	5.8	46	44	86	20	1.1	192	118	80	50	36	200	NQRS	N	N	N STUDY
nl	nl	2-4 PUS	0.8	6.2	48	60	88	25	1	194	128	88	32	38	102	NQRS	N	N	N STUDY
nl	nl	3-5 pus	1.4	5.8	44	44	86	40	1.4	184	114	96	34	46	98	NQRS	N	N	N STUDY
nl	nl	2-5 PUS	0.8	5.2	52	52	88	48	1	202	136	104	38	32	229	NQRS	N	N	N STUDY
nl	nl	3-5 pus	0.8	5.8	44	46	94	46	1.2	188	122	88	36	48	86	NQRS	N	N	N STUDY
nl	nl	2-4 pus	0.8	5.2	54	42	82	38	0.6	194	128	84	40	52	254	NQRS	N	N	N STUDY
nl	nl	3-6 pus	1.2	6	54	48	118	44	0.8	176	134	92	41	50	246	NQRS	N	N	N STUDY
nl	nl	2-5 PUS	0.8	6	44	48	102	42	1.2	190	142	90	51	48	239	NQRS	N	N	N STUDY
NL	NL	Few pus ce	0.8	6.2	48	48	112	22	1.2	174	122	92	34	38	110	NQRS	N	N	N STUDY
NL	NL	Occ.pus ce	1.4	6.2	42	54	112	34	0.8	186	128	102	32	42	128	NQRS	N	N	N STUDY
NL	NL	1-2 pus cel	1	6	44	66	102	42	1.3	196	144	106	40	44	108	NQRS	N	N	N STUDY
NL	NL	2-3 pus cel	0.8	6	56	48	86	32	1.2	198	132	108	43	46	112	NQRS	N	N	N STUDY
NL	NL	2-4 PUS CE	1.2	5.8	48	48	112	40	0.8	184	136	104	46	48	110	NQRS	N	N	N STUDY
NL	NL	1-3 pus cel	1.2	6	58	47	102	18	1.4	172	122	96	39	46	178	NQRS	N	N	N STUDY
NL	NL	2-5 pus cel	1.2	6	48	54	102	26	0.8	184	128	98	38	48	248	QRS	N	N	N STUDY
NL	NL	3-4 pus cel	1.4	5.8	44	64	86	25	1	190	120	92	36	43	126	NQRS	N	N	N STUDY
NL	nl	1-3 pus cel	1	5.8	46	48	88	22	0.8	184	138	88	44	40	210	NQRS	N	N	N STUDY
NL	NL	2-5 pus cel	0.8	6	44	44	88	20	1.2	192	142	86	45	42	306	NQRS	N	N	N STUDY
NL	NL	2-4 PUS CE	1.7	5.5	44	64	107	32	1.2	196	144	94	48	48	158	NQRS	N	N	N STUDY
NL	NL	1-2 pus cel	1	5.8	44	42	88	23	1	185	138	90	32	52	312	QRS	N	N	N STUDY
NL	NL	Occ.pus ce	1	5.5	44	44	82	25	1.4	189	140	92	33	50	118	NQRS	N	N	N STUDY
NL	NL	OCC. PUS C	1	6	48	64	94	62	1.2	191	138	86	29	44	542	NQRS	N	N	N STUDY
NL	NL	2-3 pus cel	1.2	6.4	44	62	88	32	0.8	188	134	88	30	46	142	QRS	N	N	N STUDY
NL	NL	2-3 pus cel	0.7	6.2	44	44	88	25	1.2	200	144	98	31	54	88	NQRS	N	N	N STUDY
NL	NL	1-2 pus cel	0.8	6.6	40	47	107	32	1.2	188	146	94	43	38	142	NQRS	N	N	N STUDY
NL	NL	2-4 PUS CE	1.2	5.8	28	64	107	34	0.8	210	148	110	42	36	154	NQRS	N	N	N STUDY
NL	NL	1-4 PUS CE	1.2	6	48	64	102	32	0.8	184	134	104	36	40	234	NQRS	N	N	N STUDY
NL	NL	2-5 pus cel	0.8	6.2	44	62	88	43	1.4	194	130	102	31	42	76	QRS	N	N	N STUDY
NL	nl	3-6 pus cel	2	5.5	42	44	98	34	1.4	186	132	98	36	38	114	NQRS	N	N	N STUDY
NL	NL	1-4 PUS CE	1	5.2	44	42	98	34	1	164	120	90	35	44	194	QRS	N	N	N STUDY
NL	NL	2-3 pus cel	1.2	5.5	30	68	88	44	0.8	170	118	82	36	46	224	NQRS	N	N	N STUDY
NL	NL	OCC. PUS C	0.8	6.2	32	47	102	32	0.8	172	124	78	40	48	116	QRS	N	N	N STUDY
NL	NL	2-5 pus cel	0.8	6.2	44	62	88	43	1.4	194	130	102	31	42	76	QRS	N	N	N STUDY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INTRODUCTION
Acquired immunodeficiency syndrome is caused by human immunodeficiency virus which belongs to class of retroviridiae. AIDS has become the most dangerous pandemic which has plagued us in the last two cecades. AIDS affects almost all systems in our body. Due to increased discovery of new anti retroviral drugs against HIV the survival of the patients has considerably increased and hence the cardiovascular complications of AIDS is of more prevalent now. The cardiovascular complications of HIV can be directly due to the virus itself, due to the associated risk factors like smoking and hyperlipidemias associated in HIV patients or due to the adverse effect the antiretroviral therapy. Earlier heart diseases in HIV patients were mostly found in autopsy series. But with development of new diagnostic methods it is now
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INTRODUCTION Acquired immunodeficiency syndrome is caused by human immunodeficiency virus which belongs to class of retroviridiae. AIDS has become the most dangerous pandemic which has plagued us in the last two decades. AIDS affects almost all systems in our body. Due to increased discovery of new anti retroviral drugs against HIV the survival of the patients has considerably increased and hence the cardiovascular complications of AIDS is of more prevalent now. The cardiovascular complications of HIV can be directly due to the virus itself, due to the associated risk factors like smoking and hyperlipidemias associated in HIV patients or due to the adverse effect the antiretroviral therapy....