
**“ESTIMATION OF CARDIOVASCULAR DISEASE
RISK PREDICTION BY ATHEROSCLEROTIC
CARDIOVASCULAR DISEASE (ASCVD) SCORE IN
HUMAN IMMUNODEFICIENCY VIRUS (HIV)
INFECTED PATIENTS WHICH IS CROSS
SECTIONAL STUDY”**

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ABBREVIATIONS

HIV	-	Human Immunodeficiency Viruses
CVD	-	Cardiovascular Disease
ASCVD	-	Atherosclerotic Cardiovascular disease
FRS	-	Framingham Risk Score
PLWH	-	People Living With HIv
ART	-	Antiretroviral Therapy
NCD	-	Non Communicable Diseases
IHD	-	Ischemic Heart Disease
TC	-	Total Cholesterol
HDL	-	High Density Lipoprotein Cholesterol
LDL	-	Low Density Lipoprotein Cholesterol
TG	-	Triglyceride
CIMT	-	Carotid Intima Media Thickness
FMD	-	Flow Mediated Dilation

ABSTRACT

AIM: To estimate risk of CVD in patients living with HIV by using ASCVD score and to compare ASCVD score with Framingham score in estimating the CVD risk.

BACKGROUND: Individuals with HIV are increasingly experiencing fewer infectious issues. However, over time, there's a noticeable rise in the incidence of chronic conditions such as cardiovascular disorders, cerebrovascular disease, diabetes, high blood pressure, and renal diseases as causes of mortality among this population. HIV associated immune dysfunction and chronic inflammation along with risk factors like high blood pressure, dyslipidemia, diabetes, alcohol, smoking, ART regimen are predisposing for CVD.

MATERIALS AND METHODS: The present cross sectional study was conducted among patient more than 18 years of age fulfilling inclusion criteria admitted in KLE DR.PRABHAKAR KORE HOSPITAL, BELAGAVI from 1st January 2023 Till 31st December 2023. Patients were assessed for physical biochemical examination after obtaining informed consent, the blood of the patient were analysed for dyslipidemia which include fasting lipid profile. All patients' data were collected. Patients redesigned performa and entered in excel sheet. The entire study was carried out using SPSS software, version 22 with a p-value threshold of 0.05 being considered statistically significant.

RESULT: In the present study total 106 patients fulfilled inclusion criteria out of which 70 were HIV positive and 36 were controls with mean age of 48 years in HIV and control population. 72 were total males and 34 were females. The ASCVD score was more in the HIV population than the control group with p-value 0.0006 (<0.05).

As compared to ASCVD score, Framingham score was more in HIV infected individuals as compared to controls with p-value 0.004 (<0.05)

CONCLUSION: Populations with HIV tend to have a higher cardiovascular disease (CVD) risk when assessed using the ASCVD score compared to those without HIV, with many falling into the low-risk category. The Framingham score is often more predictive of CVD risk as compared to ASCVD score.

KEYWORDS: Human Immunodeficiency Viruses, Cardiovascular Disease, Atherosclerotic Cardiovascular disease, Framingham Risk Score, Antiretroviral Therapy, Non Communicable Diseases, Total Cholesterol, High Density Lipoprotein Cholesterol, Low Density Lipoprotein Cholesterol, Triglyceride

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INTRODUCTION:

HIV-positive individuals are less likely to experience infectious complications initially, but eventually, these patients are developing non-communicable diseases such as diabetes, kidney related diseases, hypertension, cancer. HIV being low grade inflammatory disease, cardiovascular and cerebrovascular diseases are the main non-infectious causes of death for HIV-positive individuals.

HIV infection may contribute to the development of cardiovascular diseases due to its impact on immune system dysfunction and persistent inflammation. This potential influence exists in addition to traditional cardiovascular risk factors such as hypertension, tobacco use, and elevated cholesterol levels.

Certain studies suggest that dyslipidemia-related side effects of antiretroviral therapy (ART) are the reason for the elevated risk of cardiovascular disease. With the advancement of antiretroviral therapy (ART) improving the survival rates of those living with HIV, there's an increasing focus on the subsequent health challenges, notably the heightened risk of cardiovascular diseases (CVD). ART can lead to metabolic side effects, such as changes in lipid profiles and insulin sensitivity, which elevate the risk for CVD. As a result, there's a growing need to manage cardiovascular health alongside HIV treatment, particularly through lifestyle interventions and tailored ART selections that consider the cardiovascular risk of the individual.

Conversely though, when predicting an individual's cardiovascular risk, the atherosclerotic cardiovascular disease score (ASCVD) takes their race into account.

Need for The Study:

Individuals who are HIV positive are more likely to have dyslipidemia that increases the risk of Cardiovascular diseases. The ASCVD score was used to find out the risk in these individuals. HIV is a state of persistent low grade inflammation in and of itself. Assessment of cardiovascular risk is the greatest way to establish those who were at high risk and require intervention and risk modifications against those who were at low risk and do not require any kind of intervention or risk reduction.

We selected this subject because there is a dearth of research on it, and the studies that do exist are tiny sample sizes and poorly controlled.

An approximation of the Framingham risk score was made for the primarily white population. The risk score typically significantly underestimates the risk of CVD in non-white people. Race is taken into account by the ASCVD equation for predicting CVD risk.

OBJECTIVES

- 1) To estimate the risk of cardiovascular disease in patients living with HIV by using atherosclerotic cardiovascular disease (ASCVD) score.
- 2) To compare (ASCVD) atherosclerotic cardiovascular disease score with framingham score in estimating the cardiovascular disease risk.

REVIEW OF LITERATURE

AIDS ((Acquired Immunodeficiency Syndrome), also known as a disease of poverty, is a chronic,occasionally fatal condition which is due to HIV-1 and HIV-2 (Human Immunodeficiency Virus) . Currently,35 million individuals worldwide are infected with HIV , and there are 2 million new cases of HIV infection reported annually.This disease not only destroys the immune system but also it makes the body far more vulnerable to infections and other ailments⁽¹⁻²⁾

Structure and replication of HIV

Presently, the human immunodeficiency virus (HIV) is categorized into HIV-type 1 (HIV-1) and HIV-type 2 (HIV-2). HIV-1 is the predominant cause of AIDS and HIV-2 is confined only to specific regions in Central and Western Africa.

Belonging to the Lentivirus genus within the Retroviridae family, HIV shares genetic characteristics with this group. Infections by Lentiviruses are typically long-lasting, involve the central nervous system, exhibit an extended period of clinical latency, and demonstrate continuous viral replication. The retrovirus genome, which consists of two identical strands of single-stranded RNA, includes the structural genes gag, pol, and env.

In addition to these genes, the genomes of HIV-1 and HIV-2 also encompass a complex array of other regulatory and accessory genes. Both types are capable of leading to AIDS, with HIV-2 infections potentially linked to a greater incidence of central nervous system disorders. Moreover, HIV-2 is generally less virulent compared to HIV-1, resulting in a slower progression to AIDS. ⁽³⁻⁴⁾

STRUCTURAL PROTEIN

The **envelope proteins** gp120 and gp41 penetrate the surface of HIV-infected cells to attach to receptors before releasing the viral RNA. Because these proteins' spikes are heavily adorned with carbohydrates, antibodies have a hard time recognizing them.

The **matrix protein** forms a lining on the inner side of the viral membrane, playing an essential role when new virions emerge from the host cell's surface. This protein organizes into trimers that align adjacently upon the membrane

During infection, the **capsid protein** envelops the viral RNA in a cone-shaped coat and transports it within the cell. It produces stable hexamers that assemble into geodesic capsids in the manner of tiles.

The **nucleocapsid protein** binds to the viral RNA to form a stable complex that shields it. One nucleocapsid copy and a brief segment of RNA are linked together in this arrangement.⁽⁵⁾

VIRAL ENZYMES

Reverse transcriptase is pivotal in transcribing the viral RNA genome into DNA, which is then leveraged to synthesize additional viruses. This enzyme is captured mid-action, transforming viral RNA into a DNA helix, subsequently severing the RNA to synthesize a complementary DNA strand. Many current HIV treatments target and inhibit reverse transcriptase.

Integrase is responsible for incorporating the DNA version of the viral genome into the host cell's own genetic material. This allows HIV to lie latent within cells for

extended periods, posing significant challenges to eradication. Drugs that inhibit integrase offer a strategy against HIV persistence.

For HIV particles to reach full maturity, the action of HIV **protease** is indispensable. It cleaves the long polyprotein chains that constitute the virus's proteins into their functional units.

ACCESSORY PROTEINS ⁽⁶⁾

During the budding process, the **viral protein U** plays a role in facilitating the virus's release from the host cell by diminishing the binding affinity of new envelope proteins to cellular receptors.

The **viral infectivity factor** targets and neutralizes one of the host cell's defense proteins, compelling the cell to destroy this protective element.

The **viral protein R** is instrumental post-infection, directing the viral genome into the cell nucleus.

The **P6 protein** is involved in incorporating Vpr into nascent virions.

A **negative regulatory factor** inhibits the production of several key defensive proteins within the infected cell.

The **virion protein regulator** is responsible for managing the splicing and movement of viral RNA, achieved by binding to a specific hairpin structure within the RNA.

The **trans-activator of transcription protein** binds to a similar hairpin structure in the viral RNA, markedly amplifying protein synthesis.

Natural course and pathogenesis of HIV infection

HIV is unable to survive outside the lymphatic system or blood. Common household cleaning agents and disinfectants can rapidly inactivate the virus. Consequently, the virus requires direct exposure to tainted blood or bodily fluids, which can occur through injuries from needles or sharp implements, or through mucosal abrasions during sexual activity.⁽⁷⁾ Factors influencing HIV transmission include the host's vulnerability, the biological properties of the viral strain, and its density in the infected individual's body fluids.

The initial site of infection during heterosexual transmission, the most prevalent mode of infection worldwide, is the cervical mucosa.⁽⁸⁾

The virus infects dendritic cells and CD4+ lymphocytes via receptor-mediated mechanisms, facilitating its progression to local lymph nodes and into the bloodstream. Significant viral replication occurs in the lymphatic tissues of the infected mucosa and adjacent lymph nodes, even in the initial stages of infection.^(9,10)

Activated CD4+ T-cells, macrophages, and follicular dendritic cells are the principal targets for infection, all harboring viral particles. Infected cells may undergo lysis or become latently infected, particularly resting CD4+ T-cells and macrophages, which act as long-term reservoirs for the virus.⁽¹¹⁾ This presents a substantial obstacle to completely eliminating the infection, as it allows the virus to persist despite highly effective antiretroviral treatments.

RT-PCR amplification test can help in detecting Viral RNA in blood.⁽¹²⁾ The appearance of viremia in the plasma is a critical juncture in the natural progression of

HIV infection, signifying the individual's capacity to spread the virus ⁽¹³⁻¹⁴⁾ and offering the earliest opportunity to identify the infection via blood testing. ⁽¹⁵⁻¹⁸⁾

HIV AND NONCOMMUNICABLE DISEASES

HIV continues to be a major health issue due to its widespread occurrence.⁽¹⁹⁾ The advent and broad adoption of powerful antiretroviral therapy (ART) has significantly extended the life expectancy and decreased mortality rates linked to HIV. Presently, HIV is considered a long-term condition that necessitates continuous treatment and the allocation of adequate resources for patient care.⁽²⁰⁻²²⁾ Yet, beyond the initial year of antiretroviral therapy (ART), individuals are increasingly succumbing to non-infectious diseases related to HIV, rather than the virus itself.

RISK FACTOR FOR DEVELOPMENT OF NCD IN PLWH :-

- Chronic Inflammation due to HIV
- Aging
- Smoking
- Drugs
- Alcohol use
- Comorbidity with Hepatitis B OR C
- Lower economic status
- Anti-Retroviral drugs
- Obesity / Metabolic syndrome
- Unhealthy lifestyle and low physical activity

Approximately 15 million individuals on antiretroviral therapy are at an elevated risk for non-communicable diseases (NCDs) due to factors such as aging, the chronic impact of HIV, and long-term exposure to HIV treatments.⁽²³⁾ Globally, there has been a significant rise in premature death in 30-69 year old adults from non-communicable diseases with 15 million of these deaths occurring in middle and low income countries each year.⁽²⁴⁾

For those living with HIV, cardiovascular disease (CVD) stands as a leading cause of mortality, with the prevalence of ischemic heart disease (IHD) nearly doubling in association with the HIV infection.^(25,26) This may be related to increased frequency of CVD risk factors among people living with HIV (PLHIV), such as smoking, high blood pressure, and abnormal lipid levels.⁽²⁶⁾ Smoking, in particular, is linked to an increased likelihood of myocardial infarction (MI). Additionally, the commencement of antiretroviral therapy (ART) has been connected to a rise in MI occurrences,⁽²⁷⁾ especially in treatment plans that include abacavir, which have been linked to almost double the risk of MI.⁽²⁸⁾

HIV AND INFLAMMATION

Replication of HIV virus is being continuously suppressed by efficient ART regimen yet people living with HIV (PLWH) still experience persistent immune activation and inflammation due to various factors unrelated and related to HIV.⁽²⁹⁾

Non-AIDS related health issues such as cardiovascular diseases (CVD), physical decline, cancer, mental cognitive impairments, bone weakening, and liver and kidney dysfunctions are prevalent among PLWH.⁽²⁹⁾ Addressing these non-AIDS comorbidities is increasingly recognized as a critical component of comprehensive

healthcare for the aging PLWH demographic. In 2016, CVD was responsible for 56.9 million fatalities globally, making it the leading cause of death.⁽²⁹⁾

Chronic Inflammation & Immune Activation Mechanism In HIV

Continuous Viral Replication and Production

In the event of an HIV infection, rampant viral replication results in widespread inflammation, immune system activation, and a steady decline in CD4+ T-cells. The Strategies for Management of Antiretroviral Therapy (SMART) study found that consistent control of HIV duplication was linked with a reduced cardiovascular diseases (CVD) risk, as opposed to sporadic treatment. This outcome indicates a definite link between unregulated viral duplication and the risk of CVD.^(30,31) Additional studies have identified a connection between uncontrolled HIV duplication and the weakening of arterial endothelial cells, highlighting the importance of continuous antiretroviral therapy (cART) in reducing the cardiovascular risk for those with HIV/AIDS.

The measure of endothelial health, brachial artery flow-mediated dilation (FMD), showed marked improvement following HIV therapy that achieved viral suppression and immune restoration. Research conducted by Baker et al. indicated that the elasticity of arteries, both large and small, is adversely affected by untreated HIV, as assessed through pulse waveform analysis.

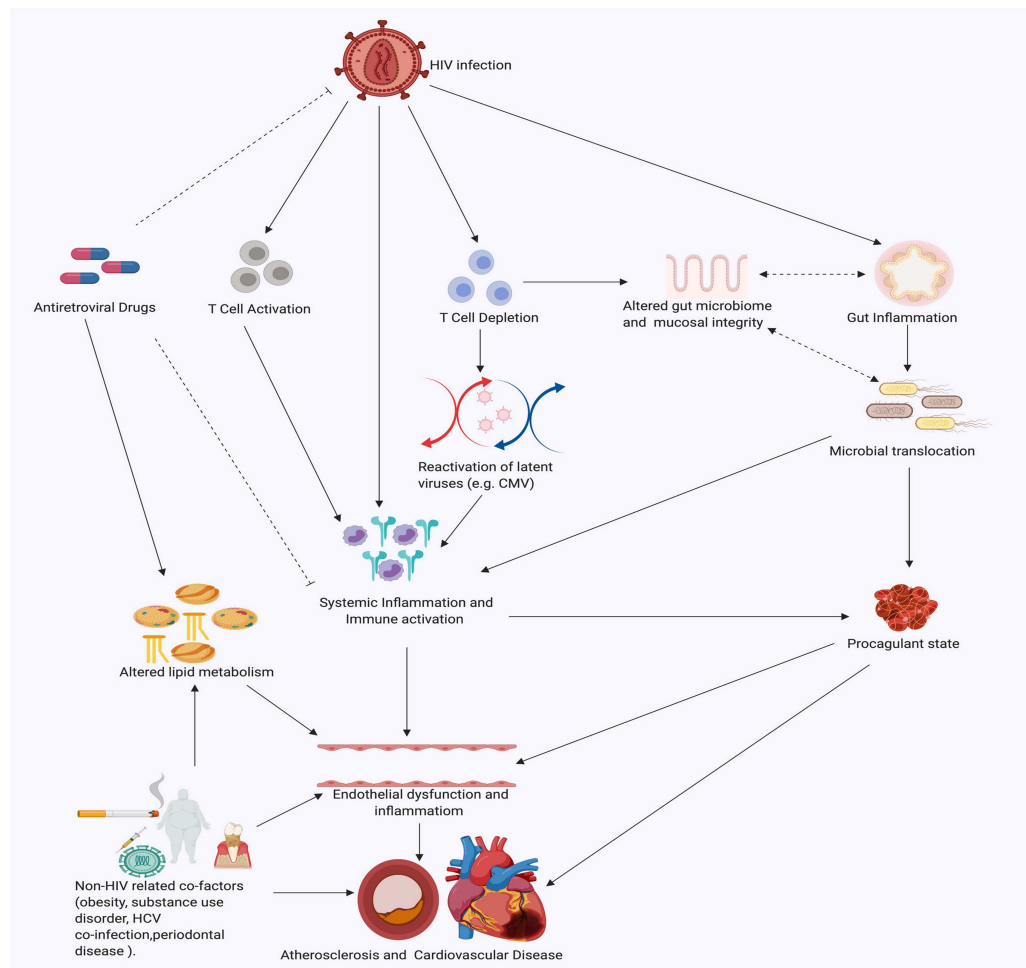


**Figure 1 - Pathophysiology of Chronic Inflammation & Immune Activation
Mechanism In HIV**

T-Cell Activation, Dysfunction and depletion

It is observed that Activation and depletion of CD4 T cells, along with activity of CD8 T cells, are the main reasons behind progression of HIV infection. With the initiation of early cART, there is a noted reduction in activated CD4 T cells (CD38+ HLA-DR+). Individuals with HIV exhibit substantially elevated levels of these cells compared to those without the virus.⁽³²⁾ Research linking the depletion of CD4 T cells to cardiovascular disease (CVD) has also identified a connection between heightened

CVD risk and low CD4 T Cell count.⁽³³⁻³⁵⁾ One particular study found that CD38+ HLA-DR+ CD4 T cells were high in HIV-positive women as compared to HIV-negative counterparts, with increased carotid artery stiffness associated with the higher number of these activated T cells.⁽³⁶⁾ CD4 T cells Activation are frequently found in atherosclerotic lesions in those without HIV, suggesting that during HIV infection, these cells may release cytokines that contribute to the development of atherosclerosis. Cytotoxic CD8 T cell production is a vital part of the body's adaptive immune response to HIV. In HIV-positive individuals, increase in CD8+ T cells (CD38+ HLA-DR+) activation is linked to damage to the carotid artery. An increase in the thickness of the carotid media intima (CIMT) has been associated with higher counts of CD8+ CD38+ HLA-DR+ T cells.

Monocyte/Macrophage Activation

Substantial evidence indicates that in the case of persistent HIV infection, the activation of monocytes and macrophages contributes to the worsening of atherosclerosis.⁽³⁷⁻³⁹⁾ In the state of inflammation, these activated cells secrete sCD163 (soluble CD163), which is a biomarker in serum linked with the extent of plaque and inflammation of the aorta in individuals living with HIV (PLWH). (37-40) sCD14 (Soluble CD14), is a marker of activation of macrophage in blood, is linked with subclinical atherosclerosis in those with HIV, thus establishing a link between the body's natural immune response and the cardiovascular disease (CVD) mechanisms in chronic HIV infection. Forty Once monocytes attach to the vascular endothelium and convert into tissue macrophages, the circulating monocytes engulf low-density lipoprotein (LDL) particles, leading to the creation of foam cells rich in lipid.⁽⁴¹⁾

This process leads to the release of pro-inflammatory cytokines and adhesion molecules that are crucial for the formation and growth of plaques.⁽⁴¹⁾

Chronic Inflammation and HIV-Mediated Immune Activation Effects on the Cardiovascular System

When viral replication is controlled by cART, immune activation for PLWH is notably reduced but not completely eradicated.. Comparing PLWH with HIV-negative controls before and during cART, there is a consistent increase in systemic inflammation. Higher levels of serum biomarkers like IL6, hs-CRP, D-dimer, sCD163, sCD14 was seen in HIV positive individuals on ART regimen as comparison to negative controls, which is indicative of the previously mentioned mechanisms.⁽³⁷⁻⁴¹⁾

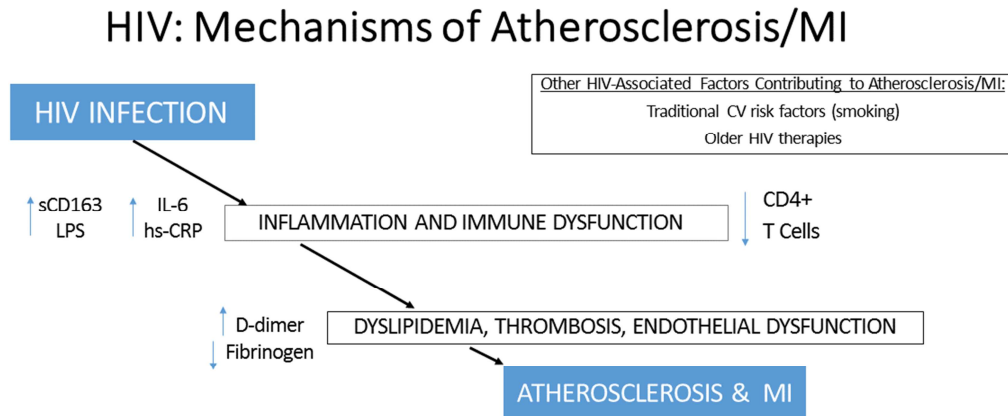


Figure 2 -Mechanisms of Atherosclerosis/MI In HIV

Lipid Metabolism and Atherosclerosis

In conditions that favor atherogenesis, such as the presence of oxidized low-density lipoprotein (LDL), nitric oxide is released from endothelium (vascular). Release of nitric oxide leads to altered vascular tone, increases the permeability of the endothelium, and stimulates the production of adhesion molecules and chemokines. Consequently, there is an accumulation of low-density lipids within the vascular walls. The consumption of oxidized LDL by macrophages leads to their transformation into foam cells, characterized by an increased output of growth factors, proinflammatory cytokines, and chemokines.⁽⁴¹⁾ This process contributes to plaque formation and the progressive thickening of the intima, indicative of atherosclerosis. In case the plaque ruptures or erodes, the lipid core and the thrombogenic collagen matrix become exposed to the bloodstream, potentially triggering fibrin deposition, platelet aggregation, and thrombus formation, which in severe cases, could result in vascular occlusion.⁽⁴¹⁾ People living with HIV (PLWH) undergoing combination antiretroviral therapy (cART) have shown a reduction in flow-mediated dilation (FMD). It has also been observed that the indicator of activation of vascular endothelial that is soluble vascular cell adhesion molecule-1 (VCAM-1), an indicator of vascular endothelial activation, is elevated in HIV-positive individuals, with cART reducing these levels. Clinical studies of untreated HIV-positive subjects have identified lipid abnormalities in those with advanced disease and significant immunosuppression, including high triglycerides, increased LDL levels, and reduced lipoprotein levels.⁽⁴²⁾ Dyslipidemia in untreated HIV may stem from immune activation and persistent inflammation. Elevated interferon-alpha (IFN-alpha) levels may impede triglyceride clearance in untreated PLWH, leading to increased blood levels.⁽⁴³⁻⁴⁵⁾

Conversely, low high-density lipoprotein levels in serum are linked to higher D-dimer and IL-6 levels.⁽⁴⁵⁾ Despite cART often improving low high-density lipoprotein cholesterol levels, the lipid profile in treated PLWH does not fully normalize.⁽⁴⁶⁻⁴⁷⁾

Hypercoagulability

In individuals living with HIV (PLWH), there is a correlation between increased risk of mortality from cardiovascular diseases (CVD) and elevated levels of hs-CRP and IL-6. Additionally, within this group, higher hs-CRP levels are linked with increased CIMT (carotid intima-media thickness). D-dimer levels, which are indicative of inflammation and a byproduct of the coagulation process, are also found to be higher in cases of HIV and exhibit a similar pattern in relation to CVD risk, akin to IL-6 and hs-CRP. Elevated D-dimer not only signifies a state prone to clotting but is also linked to inflammatory processes. Therefore, it is reasonable to infer that a state of heightened coagulability could escalate the likelihood of cardiovascular incidents, such as thrombosis, stroke, and myocardial infarction (MI).⁽⁴⁸⁻⁴⁹⁾

OTHER NON COMMUNICABLE DISEASES IN PLHIV

Research indicates that the prevalence of smoking among those with HIV is 2-3 fold higher compared to the general population, which could explain the higher overall occurrence of chronic obstructive pulmonary disease (COPD) within this group compared to those without HIV.⁽⁵⁰⁾ Even when smoking is taken into account, COPD appears more frequently in people living with HIV (PLHIV).⁽⁵⁰⁾ Definite mechanisms of lung disease in HIV positive people that are not related to smoking remain unclear, persistent HIV infection has been associated with changes in lung

microbiota and ongoing inflammation.⁽⁵⁰⁾ Nonetheless, smoking remains a key modifiable risk factor for COPD.

Chronic kidney disease (CKD) stands as a prevalent negative outcome of HIV. Currently, 2 to 10% of individuals with HIV are affected by CKD, though these figures have varied since the onset of the epidemic. Before antiretroviral therapy (ART) became available, HIV-associated nephropathy was frequently observed. The primary contributors to CKD include ART and risk factors like aging, dyslipidemia, smoking, diabetes & high blood pressure.⁽⁵¹⁻⁵²⁾ Those with HIV are at a two to twentyfold higher risk of developing ESRD (end-stage renal disease) as compared to the general population.⁽⁵³⁾ During the later stages of ART, the incidence of CKD was higher, suggesting that the extensive use of potentially kidney-damaging antiretrovirals might be a leading cause of CKD. Antiretrovirals such as indinavir, tenofovir disoproxil fumarate and atazanavir have been associated with impaired kidney function.^(51,52,55)

In the period following the introduction of (HAART) highly active antiretroviral therapy, disease related to the liver has become a significant cause of illness and mortality in individuals with HIV. It has been noted that between 20% and 93% of those on antiretroviral therapy (ART) exhibit increased levels of liver enzymes.⁽⁵⁴⁾ The heightened risk of liver damage in this population is attributed to several factors, including alcohol abuse, concurrent infections with hepatitis B and C viruses due to overlapping risk behaviors, the toxic effects of ART on the liver, the virus itself, and metabolic issues leading to NAFLD (non-alcoholic fatty liver disease).⁽⁵⁶⁻⁵⁸⁾

Hepatotoxicity, particularly associated with some older antiretroviral drugs, is a common side effect of ART and can significantly contribute to liver pathology.⁽⁵⁶⁻⁵⁷⁾ Nonetheless, NAFLD has emerged as the predominant liver condition among aging individuals with HIV, with metabolic liver diseases becoming increasingly common in this demographic.⁽⁵⁶⁾

Individuals with HIV are observed to have a higher rate of osteoporosis and bone fractures compared to those without HIV of similar age and gender.^(59,60) This increased risk may be due to factors unique to HIV or common risk elements. The commencement of antiretroviral therapy (ART), especially treatments that include TDF (tenofovir disoproxil fumarate) , lead to bone density reduction.⁽⁵⁹⁾ In comparison to the general population, people living with HIV (PLHIV) aged 41–50 and over 51 have shown a higher prevalence of osteoporosis. Moreover, a study within the Multicenter AIDS Cohort found that HIV-positive men aged 50 to 59 experienced a higher rate of fractures.⁽⁶¹⁾ The occurrence of osteoporosis was notably higher in the late-HAART period compared to the pre-HAART period, with an increase in cases over time and with advancing age.

The mental health burden among the PLHIV community is approximately double that of the non-HIV population. The incidence of depression among PLHIV has increased to 2-7 fold as compared to HIV negative individuals.⁽⁶²⁾ Both HIV & mental health disorders can increase susceptibility to the virus. Addressing mental health is crucial in HIV treatment, as it is linked to adherence to ART.

PLHIV have a higher propensity for alcohol misuse and are more prone to develop COPD (chronic obstructive pulmonary disease), IHD (ischemic heart disease) , disease related to liver , mental health challenges, and kidney diseases. Osteoporosis

rates were notably higher in PLHIV within the 41–50 and over 51 age brackets. Consequently, the incidence of non-communicable diseases (NCDs) understandably escalates with age.

SPECTRUM OF CVD IN PLWH:-

- Coronary Artery Disease
- Heart Failure / Cardiomyopathy
- Cerebrovascular accidents { hemorrhagic and ischemic stroke, TIA }
- Atrial fibrillation
- Peripheral artery disease
- Vascular Dysfunction
- Pericarditis
- Myocarditis

Cardiovascular disease (CVD) represents a significant health challenge for individuals with HIV.⁽⁶³⁾ While any heart condition can affect HIV-positive people, coronary artery disease (CHD) is the most frequently encountered form of CVD, often presenting as acute coronary syndrome (ACS).^(64,65)

A greater occurrence of unstable, high-risk atherosclerotic plaques has been observed in this population, suggesting that multiple factors may converge to elevate the risk of CVD among those with HIV.⁽⁶⁶⁾ Individuals with CVD generally experience a reduction in life expectancy by 6% to 15%, face a higher rate of CHD compared to the non-HIV population, and tend to be affected at an earlier age.

Like risk factors in HIV negative people, the following can contribute to CVD in those living with HIV:

Nonmodifiable risk factors:-

- Old age
- Significant Family history of CVD

Modifiable risk factors:-

- Dyslipidemia
- Hypertension
- Diabetes
- Smoking
- Diet
- Compromised immunity
- Activation of immune system
- Inflammation associated with HIV
- ART regimen

RISK FACTORS OF CARDIOVASCULAR DISEASE

Lipid Profiles

Abnormal Blood lipid profiles are typically considered atypical when they exhibit elevated (TG) Triglycerides (≥ 150 mg/dL); (TC) Total cholesterol (≥ 200 mg/dL); (LDL) Low-density lipoprotein cholesterol (≥ 130 mg/dL), or diminished (HDL) high-density lipoprotein cholesterol (< 40 mg/dL). The condition characterized by such irregularities in these lipid levels is known as dyslipidemia.

Antiretroviral therapy (ART) has been observed to adversely affect lipid levels in the blood. Reports from Malaysia indicate that dyslipidemia was present in 82% of

individuals undergoing ART.⁽⁷¹⁾ In Asia, data indicates that individuals commencing antiretroviral therapy (ART) exhibit varying degrees of lipid abnormalities. Specifically, elevated triglycerides were observed in 7-39% of patients total cholesterol (TC) in 3-8%, low-density lipoprotein (LDL) in 2-17%, and high-density lipoprotein (HDL) in 19-86%. These figures highlight the prevalence of dyslipidemia among patients on ART, which is a concern due to its association with cardiovascular risks.^(67-70,72-74) Conversely, for those with previous ART exposure, the prevalence of irregular lipid markers was 39–54%, 26–54%, 22-48%, and 14-68%, respectively.^(67,68,71-75) Additionally, a study in Thailand showed a higher incidence of abnormal lipid levels among individuals on ART regimens that included protease inhibitors (PI).⁽⁷⁶⁾ Research across various income levels has demonstrated a significant link between the use of ART and the presence of detrimental lipid profiles.^(67,73,74,77,78)

Metabolic disturbances, including dyslipidemia, hyperinsulinemia, and changes in body fat distribution, affect approximately half of the individuals undergoing HAART treatment.^(79,80) The typical features of dyslipidemia associated with HAART include high levels of triglycerides, reduced HDL cholesterol, and increased total cholesterol, sometimes accompanied by raised LDL cholesterol levels.⁽⁸¹⁾

This lipid profile is also associated at a cellular level with insulin resistance and disorders involving the redistribution of body fat, which can affect the effectiveness of HAART. However, dyslipidemia may manifest without clear signs of lipoatrophy or insulin resistance. This suggests that dyslipidemia could either be an early indicator of disease that prompts earlier detection and more comprehensive clinical assessment of HIV patients, or it may not be directly linked to these

conditions. With increased relative risk of myocardial infarction of 1.2/year was associated with the use of protease inhibitors, individuals with HIV and persistent dyslipidemia are considered to have a heightened risk of cardiovascular incidents.^(82,83)

Pathogenesis of HIV-associated dyslipidemia

Dyslipidemia associated with HIV was acknowledged well before the widespread adoption of HAART regimens that include protease inhibitors.^(84,85) The anti-inflammatory and antioxidant capabilities of HDL cholesterol have linked its deficiency to early immune activation in HIV infection, with implications that may extend beyond the development of atherosclerosis.⁽⁸⁶⁻⁹⁰⁾

HDL cholesterol and triglyceride-rich lipoproteins are produced and released into the bloodstream by the liver and intestines. HIV-1 disrupts the normal process of cholesterol transport out of macrophages, mediated by the ATP-binding cassette transporter A1, leading to reduced HDL levels in the plasma.⁽⁹¹⁾ Inflammation typically activates enzymes which further decrease HDL level in plasma like phospholipase A2 and endothelial lipase.⁽⁹⁰⁾

In cases of hypertriglyceridemia, HDL becomes enriched with triglycerides, making it more susceptible to removal by hepatic lipase.⁽⁹⁰⁾ The dyslipidemia observed in HIV closely mirrors that in other chronic infections, with a noted association between elevated interferon α levels and higher plasma triglyceride levels in advanced stages of HIV.⁽⁹²⁻⁹⁴⁾

Drug-naïve individuals with HIV exhibit elevated levels of tumor necrosis factor (TNF) α , which can increase if patients develop opportunistic infections. TNF α interferes with insulin's ability to inhibit lipolysis and disrupts the metabolism of FFA (free fatty acids) and their oxidation.⁽⁹⁵⁾

Finally, reductions in total plasma cholesterol, HDL, and LDL may be indicative of the nutritional status of HIV patients, often characterized by weight loss and protein insufficiency.⁽⁹⁶⁻⁹⁷⁾

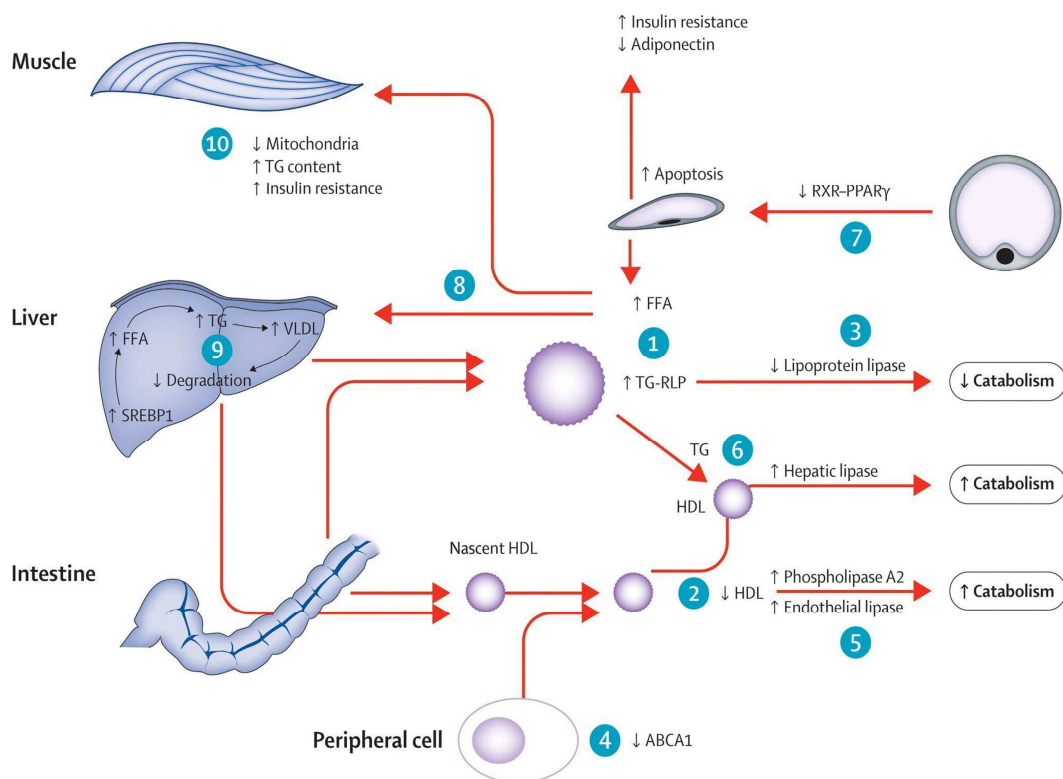


Figure 3 - Mechanism of HIV Associated Dyslipidemia

Figure: Schematic representation of selected mechanisms underlying HIV-associated dyslipidaemia

Mechanisms are caused by effects of the virus itself and effects of highly active antiretroviral treatment (HAART). The two fundamental biochemical disturbances are: (1) increased triglyceride (TG)-rich lipoproteins (RLPs), particularly very-low-density lipoprotein (VLDL); and (2) decreased high-density lipoprotein (HDL). The inflammatory cytokine response to HIV infection: (3) decreases lipoprotein lipase activity, which results in accumulation of TG-RLP; (4) decreases cholesterol efflux from peripheral cells via the ATP-binding cassette protein A1 (ABCA1), which results in decreased formation of HDL; and (5) increases activity of phospholipase A2 and endothelial lipase, which results in increased catabolism of HDL. Increased plasma TG results in (6) abnormal TG-enrichment of HDL, which increases catabolism via hepatic lipase. HAART causes redistribution of adipose tissue as a result of (7) decreased retinoid X receptor-peroxisome proliferator-activated receptor γ (RXR-PPAR γ) activity. (8) Free fatty acid (FFA) spillovers from apoptotic peripheral adipocytes increase FFA flux to the liver and skeletal muscle. In the liver (9), increased FFA supply and upregulation of the TG synthetic pathway, through the sterol regulatory element binding protein-1 (SREBP1) and downstream targets, increase hepatic TGs and ultimately secretion of TG-rich VLDL, while protease inhibitors interfere with intracellular degradation of VLDL and related particles. In the muscle (10), HAART is associated with mitochondrial depletion, which in turn compromises FFA oxidation; as a result, intramyocellular and intermyocellular TG content increases. Insulin resistance in liver and skeletal muscle compounds the metabolic disturbances, including dyslipidaemia.

Antiretroviral-induced dyslipidemia

The complex mechanisms behind dyslipidemia in those undergoing HAART are influenced by a mix of factors related to medication, hormonal and immune responses, and genetic tendencies.^(98,99) Prior to HAART initiation, individuals with HIV displayed abnormal lipoprotein levels when compared to non-HIV individuals, and these discrepancies intensified upon starting treatment. The lipid profile associated with HAART typically includes increase in total cholesterol, LDL cholesterol, triglycerides and decrease in HDL cholesterol.⁽⁹⁶⁻⁹⁸⁾ This profile may also present elevated levels of lipoprotein (a), various apolipoproteins, and small dense LDL particles, which are indicative of underlying proatherogenic changes.

Studies into the dynamics of lipoprotein metabolism have shown that the accumulation of triglyceride-rich very low-density lipoprotein (VLDL) in the plasma can arise from several sources: reduced breakdown due to HIV itself, heightened production due to HAART, or diminished catabolism caused by protease inhibitors. The interaction of FFA (free fatty acids) with the key regulatory protein SREBP1 may also contribute to the reduced breakdown of triglyceride-rich lipoproteins by tissue and plasma lipase⁽⁹⁹⁻¹⁰¹⁾ disrupt the normal metabolism of lipids and free fatty acids after meals,⁽¹⁰¹⁻¹⁰²⁾ and affect the capture of fatty acids in the periphery due to HAART.⁽¹⁰³⁾ Additionally, PI (protease inhibitor) therapy has been associated with unusual accumulation of fat within muscle cells, leading to increase in insulin resistance and increased levels of triglyceride-rich lipoproteins and apolipoprotein B in the plasma.⁽¹⁰⁴⁾

HAART-induced mitochondrial alterations, particularly when protease inhibitors are used alongside nucleoside reverse transcriptase inhibitors (NRTIs), lead

to the inhibition of mitochondrial DNA polymerase γ . This suppression hampers energy production, disrupts the respiratory chain, and leads to a reduction in mitochondrial DNA content. Such impairment of the mitochondrial respiratory chain can trigger irregularities in adipocytes and other cells, potentially causing lipoatrophy. Additionally, development of insulin resistance and dyslipidemia occur because of mitochondrial dysfunction within skeletal muscle.⁽¹⁰⁵⁻¹⁰⁶⁾

Diagnosis of HIV-associated dyslipidemia

The diagnosis of dyslipidemia in HIV patients involves measuring serum or plasma levels of triglycerides, as well as total cholesterol, LDL cholesterol, and HDL cholesterol after a fasting period of 12 hours.

Diabetes and Abnormal Blood Glucose Levels

Research indicates that between 3% and 15% of individuals living with HIV also suffer from diabetes mellitus (DM).⁽¹⁰⁷⁻¹¹⁰⁾ The disparity in these findings could stem from variations in the demographics of the study participants, diagnostic methods for DM, and other interfering factors. While certain studies suggest a heightened susceptibility to type 2 diabetes among those with HIV,^(107,111-112) others have not found a significant link or have observed a contrary effect.⁽¹⁰⁹⁾

The direct impact of HIV on the development of type 2 diabetes remains a topic of ongoing discussion.⁽¹¹⁰⁾ Known risk enhancers for diabetes include infection with the hepatitis C virus (HCV),⁽¹⁰⁹⁾ specific substance use such as corticosteroids and opiates, and reduced testosterone levels.⁽¹¹⁰⁾ Additionally, fat loss associated with antiretroviral therapy (ART),⁽¹⁰⁹⁾ increased visceral adiposity,^(108,111) and inflammation

related to HIV.⁽¹¹²⁾ are also considered potential risk factors for diabetes in those affected by HIV.

A variety of elements are speculated to play a role in the pathophysiology, such as the use of antiretroviral medications, concurrent HCV infection, lipodystrophy, mitochondrial dysfunction, an increase in circulating free fatty acids, fat deposition in muscles and internal organs, elevated proinflammatory markers, and genetic factors. The exact contribution of each of these factors is yet to be fully understood. Certain protease inhibitors are known to disrupt insulin signaling through their impact on Glut-4, while some nucleoside reverse transcriptase inhibitors are associated with mitochondrial dysfunction, contributing to insulin resistance.

Clinical trials have demonstrated that metformin and thiazolidinediones (glitazones) are effective in reducing insulin resistance in individuals with diabetes and HIV.

Metformin, when administered as a monotherapy, can reduce levels of HbA1c (glycated hemoglobin) by approximately 1%. Its benefits include a low risk of hypoglycemia when not combined with other drugs, negligible impact on weight, and affordability. Moreover, metformin is associated with a decreased risk of cardiovascular disease (CVD) by reducing ongoing inflammation in HIV.⁽¹¹³⁾ However, gastrointestinal reactions like nausea and diarrhea are common side effects due to metformin's gastrointestinal sensitivity. A notable drug-drug interaction occurs between metformin and dolutegravir, which can lead to increased levels of metformin and the rare but serious side effect of lactic acidosis. Hence, careful monitoring is advised when these medications are used together.⁽¹¹⁴⁾

Hypertension

Over a six-month period of antiretroviral therapy (ART), there was a rise in the mean SBP and DBP, with hypertension developing in 10% HIV positive patients. A study conducted in Thailand observed that the occurrence of hypertension was comparably prevalent among patients who were new to ART and those who had been on the treatment for some time (22% vs. 28%).⁽¹¹⁷⁾ Additionally, it was noted that approximately half of the HIV patients receiving ART in Malaysia were also affected by hypertension.

The pathophysiological factors contributing to hypertension in adults with HIV encompass a range of mechanisms such as the movement of microbes across the gut barrier, persistent inflammatory responses, fluctuations in immune function and recovery, the affinity of the virus for certain cells, alterations in fat distribution, the role of fat-derived hormones, and kidney conditions associated with HIV.

Microbial Translocation

The connection between microbial translocation from the gut and the development of hypertension in individuals with HIV has been established.^(116,120) The HIV virus targets mainly CD4 T cells which are present in gut-associated lymphoid tissue, potentially compromising the mucosal barrier and permitting microbes to infiltrate the bloodstream.^(116, 120-122)

In HIV infections, markers such as sCD14 (soluble CD14) and lipopolysaccharide are associated with increased blood pressure.⁽¹¹⁶⁾ A case-control study within a cohort of HIV-positive patients identified a link between the onset of hypertension and elevated initial levels of lipopolysaccharide. There was a significant

correlation between the plasma levels of sCD14 and lipopolysaccharide (Spearman $\rho=0.62$; $P=0.01$) in patients with hypertension, but not in those without hypertension or in uninfected individuals, indicating a unique connection to microbial translocation in hypertensive patients with HIV.

Furthermore, lipopolysaccharide is implicated in arterial rigidity and the loss of endothelial cells.⁽¹²³⁾ There is also a noted interaction between the RAAS (renin-angiotensin-aldosterone system) and lipopolysaccharide. Some Studies have shown lipopolysaccharide can enhance the activity of angiotensin II by interacting with leukocytes that adhere to the endothelium. Additionally, lipopolysaccharide appears to upregulate angiotensin receptors on endothelial cells, thereby activating the RAAS. These receptors are part of a pathway regulated by NADPH (nicotinamide adenine dinucleotide phosphate), which leads to formation of ROS (reactive oxygen species), endothelial impairment, and ultimately, hypertension.⁽¹²³⁾

ART-Mediated Inflammation

It has been illustrated that protease inhibitors (PIs) initiate inflammatory pathways. PI-mediated production of ROS via macrophage accumulation and mitochondrial effects, which changes the cytokines that are naturally present in adipocytes.⁽¹²⁴⁾ Additionally It has been shown that vascular collagen deposition, arterial stiffness, and kidney damage are related to ROS overproduction.

Immune Reconstitution

In adults with HIV, there is a notable decline in CD4 T-cell numbers, which tend to recover rapidly with the initiation of antiretroviral therapy (ART). Studies have shown that a greater frequency of hypertension is associated with lower initial

CD4 T-cell counts after beginning ART. It has been noted that individuals with baseline CD4 counts above 500 cells per millimeter did not experience changes in arterial stiffness or blood pressure due to ART.⁽¹²⁵⁾

HIV-positive young adults display immune system characteristics similar to those found in older adults without HIV. Even after starting ART in HIV positive people and after achieving viral suppression, still aged immune profiles persist. Decrease in telomerase length is the mechanism of aging in non-HIV populations.⁽¹²⁶⁾ Premature shortening of telomeres by affecting mitochondrial function occurs by HIV virus.⁽¹¹⁷⁾ It is theorized that this immune aging, as evidenced by shortened telomeres, contributes to the increased incidence of cardiovascular diseases in HIV-positive individuals.

Lipodystrophy

Both HIV and the treatment with antiretroviral therapy (ART) have been identified as potential causes of lipodystrophy, which includes both lipoatrophy (loss of fat) and lipohypertrophy (gain of fat). This condition, characterized by the simultaneous increase of central body fat and decrease of peripheral body fat, may contribute to the development of hypertension.⁽¹¹⁸⁾

In studies involving HIV-positive adults with varying exposure to ART, both lipoatrophy and lipohypertrophy emerged as independent risk factors for hypertension. Notably, individuals with even a slight degree of lipoatrophy were found to have a fivefold higher risk of hypertension, even after adjusting for BMI (body mass index), compared to those without any lipodystrophy. Furthermore, the dysregulation of the RAAS (renin-angiotensin-aldosterone system), often seen in

conjunction with HIV & ART-induced lipodystrophy and lipohypertrophy, has been linked to hypertension.

Specifically, Protease inhibitors (PIs) are linked with disturbances in lipid metabolism and lipodystrophy, which in turn may be related to an increased risk of hypertension and cardiovascular diseases.^(119,127,128)

Role of Leptin

Leptin, an adipokine that is part of the body's inflammatory response—which can also be stimulated by HIV—primarily affects the aorta and hypothalamus.^(129,130) Studies have shown that individuals with HIV who are on antiretroviral therapy (ART) exhibit increased levels of leptin, independent of their body mass index.⁽¹³¹⁾ The presence of leptin receptors in the hypothalamus and aorta is known to trigger the activation of both the RAAS (renin-angiotensin-aldosterone system) and sympathetic nervous system.^(129,130) Elevated levels of leptin are responsible for stimulating the sympathetic nervous system.⁽¹³⁰⁾ Furthermore, leptin has a direct impact on the medulla oblongata and prompts the liver to produce acute-phase reactants, thereby activating both RAAS and central nervous system.⁽¹³⁰⁾

HIV-Related Renal Disease Microalbuminuria

In those with HIV, there is an independent association between hypertension and microalbuminuria, an indicator of renal impairment. Studies indicate that the frequency of albuminuria is higher in HIV individuals compared to the Non-HIV population, with estimates suggesting up to four times the prevalence in the former group. Among adults living with HIV, there is a heightened occurrence of albuminuria, which may be linked to ongoing inflammatory processes as well as the

direct impact of the HIV virus itself. Microalbuminuria has been identified as a standalone predictive factor for the development of hypertension.^(115,132)

HIV-Related RAAS activation

Aberrant activation of the renin-angiotensin-aldosterone system (RAAS) plays a major role in the development of hypertension among individuals with HIV. A variety of factors contribute to increased RAAS activity in those with HIV.⁽¹¹⁷⁾ Adults with HIV have shown higher plasma renin levels, even when sodium intake is low, possibly due to the structural similarity between HIV-1 protease and renin.⁽¹³³⁾ The virus has been shown to activate CD4 T cells, which are natural sources of RAAS activation, leading to increased renin production.⁽¹³³⁾

Arterial Stiffness: Arterial stiffness, an independent predictor of hypertension, can be exacerbated by protease inhibitors (PIs). Arterial stiffness has been linked to left ventricular hypertrophy, activation of the sympathetic nervous system, and elevated central pulse pressure.⁽¹²⁹⁾ Patients treated with PIs, specifically lopinavir/ritonavir, were found to have a doubled risk of developing hypertension compared to those on other ART regimens.⁽¹¹⁹⁾

The study conducted by Schillaci et al. indicates that individuals with HIV who are treated with protease inhibitors (PIs) demonstrate a higher degree of aortic rigidity. This is demonstrated by a rise in the velocity of the pulse wave traveling through the aorta and its augmentation when compared to those without HIV. These findings imply that inflammation of the blood vessels may be a direct contributor to the malfunction of the endothelial cells lining the arteries, ultimately resulting in increased arterial rigidity.⁽¹²⁸⁾

Protease Inhibitors: PIs are implicated in several mechanisms that contribute to hypertension in HIV-positive patients, including lipodystrophy, arterial stiffness, endothelial dysfunction, RAAS activation, and dyslipidemia. The PI combinations of lopinavir/ritonavir and atazanavir/ritonavir have been shown to act on fat cells, triggering inflammatory pathways mediated by adipokines, which in turn activate the adipose RAAS.

Risk Stratification

carotid intima-media thickness (cIMT) ⁽¹³⁴⁾

Atherosclerosis is often present in the coronary and carotid arteries. Utilizing ultrasound to measure carotid intima-media thickness (cIMT) and plaque offers a non-invasive, precise, and consistent method for early detection of vascular atherosclerosis and evaluating cardiovascular disease (CVD) risk. Notably, cIMT is a reliable indicator of future cardiovascular events such as myocardial infarction, stroke, and coronary heart disease, even in asymptomatic individuals, acting as an indirect measure of atherosclerosis.

In general populations, the presence of carotid plaque is associated with cardiovascular incidents, and its ability to predict such events is comparable or even superior to cIMT measurements. Specifically, in HIV patients who have not commenced combination antiretroviral therapy (cART), abnormal carotid ultrasound results, including plaque presence or a cIMT exceeding 0.9 mm, show a strong association with all cardiovascular risk factors, as well as with the estimated Framingham risk score.

CARDIAC FUNCTIONING ⁽¹³⁵⁾

In various Asian research initiatives, two-dimensional echocardiograms have been utilized to assess cardiac functionality in individuals living with HIV. Findings from India reveal that a significant proportion, 67%, of those with HIV exhibited irregularities in their echocardiogram assessments. The most common cardiac irregularity identified was diastolic dysfunction, affecting nearly half of the patients at 48.2%. This was followed by LV systolic dysfunction, which was observed in 17.6% of the HIV-positive population.

ASCVD (ACC/AHA) ⁽¹³¹⁻¹⁴³⁾ The parameters considered are gender, age, race, Total Cholesterol, HDL-C, LDL, Diabetes, Smoking status, and SBP (systolic blood pressure).

- **Outcome:** Forecasts the 10-year risk of CVD (cardiovascular disease), intended for individuals aged 40-79.
- **Constraints:** Does not account for HIV-specific factors.
- **Comment:** The risk may be underestimated for certain groups, such as women and people of color. A recalibration is suggested to improve its accuracy in European cohorts.

Current Age [ⓘ] *	Sex *		Race *		
	Male	Female	White	African American	Other
Age must be between 20-79					
Systolic Blood Pressure (mm Hg) *	Diastolic Blood Pressure (mm Hg) *				
Value must be between 90-200		Value must be between 60-130			
Total Cholesterol (mg/dL) *	HDL Cholesterol (mg/dL) *	LDL Cholesterol (mg/dL) [ⓘ] ○			
Value must be between 130 - 320		Value must be between 20 - 100		Value must be between 30-300	
History of Diabetes? *		Smoker? [ⓘ] *			
Yes No		Current [ⓘ] Former [ⓘ] Never [ⓘ]			
On Hypertension Treatment? *		On a Statin? [ⓘ] ○		On Aspirin Therapy? [ⓘ] ○	
Yes No		Yes No		Yes No	

Figure 4 - ASCVD Scoring System

10-year risk levels for atherosclerotic cardiovascular disease (ASCVD) are classified as follows:

- **Low Risk score** include Less than 5%
- **Borderline Risk score** include 5% to 7.4%
- **Intermediate Risk score** include 7.5% to 19.9%
- **High Risk score** include 20% or more

ASCVD is characterized by a gradual development from initial fatty deposits to more complex plaques, and eventually to significant blockages. Early identification and intervention for at-risk individuals, including lifestyle changes and risk factor management, can markedly decelerate disease progression. Given the increasing prevalence of non-communicable diseases due to lifestyle shifts among other reasons, cardiovascular risk assessment has become increasingly vital.

The new risk assessment method proposed by the RAWG, known as the ASCVD Pooled Cohort Equations, marks a significant advancement by providing risk estimates tailored to African American and Caucasian men and women, reflecting the varying prevalence of risk factors.

Moreover, the RAWG expanded its focus from strictly hard coronary heart disease (CHD) events to encompass ASCVD as a whole. Additionally, for patients or clinicians uncertain about initiating long-term statin therapy, the RAWG suggested considering several supplementary factors beyond the equations to help refine or reclassify an individual's risk following the initial quantitative risk assessment.

Limitations:

- Quantitative risk estimation is inherently a probabilistic and imprecise process. Over a decade, an individual's actual risk of ASCVD is either 0% or 100%, not a percentage in between.
- The absence of “novel” risk markers, deemed important by some for risk assessment, is a notable omission in the updated Pooled Cohort Equations.
- Concerns have been raised that the risks for ASCVD may be overstated due to the specific cohorts used to derive the Pooled Cohort Equations.

Framingham Risk Score For Hard Coronary Heart Disease ⁽¹⁴⁴⁻¹⁵²⁾

The Framingham score is a tool recommended in both American and European guidelines for categorizing individuals' primary prevention needs. It incorporates factors such as Total Cholesterol, HDL-C, Gender, Age, Smoking habits, Diabetes, Systolic blood pressure, and use of antihypertensive medication.

Outcome: It provides a forecast of cardiovascular disease risk over a span of 5 to 10 years, and is applicable to people between 30 to 75 years of age.

Considerations: It does not account for factors related to HIV.

Observation: The D:A:D score is preferred by the European AIDS Clinical Society (EACS) for those with HIV/AIDS.

When applied to six ethnically diverse U.S. cohorts, D'Agostino et al. found that the FHS-CVD score performed well for White and Black populations but tended to overestimate risk among Native American women, Japanese American, and Hispanic men. It has been noted to significantly overestimate risk in northern Europeans with a high risk of coronary artery disease (CAD), and even more so in southern Europeans with a lower risk. In the Chinese Multi-provincial Cohort Study, the FHS-CVD score consistently overestimated the absolute CAD risk (CMCS), and it underestimated the risk for Indians by 200% compared to the Chinese.

Limitations of the FHS-CVD Score:

- The FHS-CVD score is recommended for those with more than two risk factors but is not suitable for extreme cases of any single risk factor, such as heavy smoking or very high cholesterol levels.
- It does not include measures for reducing risk factors other than hypertension and uses total cholesterol instead of LDL-C. It omits factors like South Asian ethnicity and family history, which are associated with a doubled risk of future CAD, as well as other known risk factors such as fibrinogen, NON-HDL-C, apolipoprotein- B, C-reactive protein, left ventricular hypertrophy, estimated glomerular filtration rates, physical inactivity, metabolic syndrome, high lipoprotein(a), high triglycerides, and more.
- The FHS-CVD score focuses solely on CAD events and does not consider other vascular events like stroke, which are more prevalent in Asian

populations. The scoring system was developed based on research in a predominantly white, middle-class demographic and may not be as accurate for younger individuals or those from various ethnic backgrounds .

- A significant limitation is that the FHS-CVD score only estimates the likelihood of developing CAD over a 10-year span.

Framingham Risk Score for Hard Coronary Heart Disease ☆

Estimates 10-year risk of heart attack.

INSTRUCTIONS

There are several distinct Framingham risk models. MDCalc uses the 'Hard' coronary Framingham outcomes model, which is intended for use in **non-diabetic** patients age 30-79 years with no prior history of coronary heart disease or intermittent claudication, as it is the most widely applicable to patients without previous cardiac events. See the [official Framingham website](#) for additional Framingham risk models.

When to Use ▼

Pearls/Pitfalls ▼

Age		years
Sex	Female	Male
Smoker	No	Yes
Total cholesterol	Norm: 150 - 200	mg/dL ↵
HDL cholesterol	Norm: 0 - 60	mg/dL ↵
Systolic BP	Norm: 100 - 120	mm Hg
Blood pressure being treated with medicines	No	Yes

Result:

Figure 5 - Framingham Heart score system

The cardiovascular risk scoring system categorizes individuals as follows:

- **Low Risk** score include Below 10%
- **Moderately High Risk** score include Between 10% and 20%
- **High Risk** score include Above 20% This scoring system, while useful for assessing cardiovascular risk in Western populations, may not accurately reflect the risk in the Indian population due to variations in racial, ethnic, and behavioral risk factors.

ASCVD vs. Framingham Score: The ASCVD risk assessment faces limitations due to its variable cutoffs, such as age and cholesterol levels, potentially excluding a significant portion of the population. This is in contrast to the Framingham score, which accommodates a broader range of data, including individuals with high blood pressure, making it more suitable for population studies and assessing cardiac risk in men aged 18 and above. The Framingham Risk Score (FRS) has shown greater efficacy in differentiating patients without coronary artery disease (CAD) from those with CAD than in distinguishing between patients with varying degrees of CAD severity, as per research by Z. Günaydın et al. among Asian populations. While the FRS is valuable for CAD screening, it lacks precision in identifying the severity of lesions.

Prevention and Therapy in HIV Patients: Primary care for HIV patients includes risk stratification and interventions aimed at reducing cardiovascular risk due to the high incidence of traditional risk factors.

Key interventions include:

- Smoking cessation
- Behavioral modification counseling
- Initiating treatment for diabetes, dyslipidemia, and hypertension

Hypertension can be managed with diuretics, beta-blockers, and renin-angiotensin system blockers, which are generally well-tolerated and have minimal drug interactions. Calcium channel blockers may have interactions with protease inhibitors (PIs) and non-nucleoside reverse transcriptase inhibitors (NNRTIs) due to CYP3A4 metabolism. Diabetes management and prevention follow the same protocols as the general population, including annual metabolic assessments.

Aspirin therapy recommendations should be based on cardiovascular risk, determined by one of the previously mentioned risk scores. Aspirin is initiated when its benefits outweigh the risks of gastrointestinal bleeding and if the cardiovascular risk is high. However, aspirin is often underprescribed, possibly due to multiple comorbidities, increased bleeding risk, potential drug interactions, and the risk of developing antiplatelet resistance.

Approach to management of HIV-associated dyslipidemia⁽¹⁵³⁾

1. Dietary and Lifestyle Adjustments:

- a. Trials involving non-pharmacological interventions have shown mixed results.
- b. Recommendations include a wise diet with reduced overall caloric intake, decreased fat-derived calories, minimal saturated and transaturated fat, and consumption of foods with a low glycemic index;
- c. Enhancing physical activity, particularly resistance exercises.

2. Nutritional Guidance:

- a. When dietary needs conflict, consulting a nutrition expert is advisable.

3. Medication Review:

- a. Switching antiretroviral drugs may be necessary, as protease inhibitors are often associated with more significant dyslipidemia, including elevated triglyceride levels.

4. Hierarchy of Hyperlipidemia Induction:

- a. The order of hyperlipidemia induction by protease inhibitors is as follows: Ritonavir > Amprenavir/Nelfinavir > Indinavir/Saquinavir/Lopinavir > Atazanavir.

5. Variability Among Antiretrovirals:

- a. Non-nucleoside reverse transcriptase inhibitors (NNRTIs) show different levels of dyslipidemia, with tenofovir presenting less dyslipidemia compared to stavudine. NNRTIs and nucleoside reverse transcriptase inhibitors (NRTIs) generally induce less dyslipidemia than protease inhibitors.

Statins as a Treatment Option:

- Statins are prescribed for baseline triglycerides below 5 mmol/L as well as Total and LDL cholesterol levels.
- Depending on the specific statin and dosage, reductions in triglycerides may be up to 25% and in LDL cholesterol up to 50%.
- Given that both HAART medications and statins are processed by the enzyme CYP3A4, there's a risk of notable drug interactions.

- Statins like Fluvastatin, Rosuvastatin, and Pravastatin are less reliant on CYP3A4, making them preferable choices in cases involving higher doses or multiple medications.
- These are generally mild to moderate in effect, efficacious, and have good tolerability.
- As a single treatment, they are unlikely to meet lipid level goals.
- Recent guidelines suggest that HIV patients taking pitavastatin may have a reduced risk of significant cardiovascular incidents.

Role of Fibrates:

- Fibrates are considered when baseline triglycerides exceed 5 mmol/L
- They can cut triglyceride levels by as much as 50% and LDL cholesterol by 25%.
- Achieving optimal cholesterol levels solely with fibrates is improbable.
- Combining fibrates with statins requires regular checks of liver enzymes and creatine kinase levels.
- Ezetimibe is generally well-received and can be used alone for mild dyslipidemia or when statins are not tolerated.
- For more severe cases, it can be combined with other drugs to lower LDL cholesterol by up to 25%.
- In the context of protease inhibitor-induced dyslipidemia, niacin should be used cautiously.

Benefits of Fish Oils:

- Omega-3 fatty acids (PUFA) can boost HDL cholesterol by up to 20% and reduce plasma triglycerides by as much as 50%.

Pitavastatin to Prevent Cardiovascular Disease in HIV Infection ⁽¹⁵⁴⁻¹⁵⁹⁾

Over a median follow-up duration of 5.1 years, a study observed that participants with HIV who were treated with pitavastatin were less likely to suffer from significant cardiovascular complications compared to those on a placebo. The selection of pitavastatin calcium for the study was informed by its minimal drug-drug interactions with the antiretroviral medications typically prescribed.

In this third-phase clinical trial, 7,769 HIV-positive individuals at low-to-moderate risk of cardiovascular disease and receiving antiretroviral therapy were randomly allocated to either a placebo group or a treatment group receiving 4 mg of pitavastatin calcium daily. The primary goal was to monitor the incidence of major cardiovascular events, which included conditions such as peripheral arterial ischemia, stroke, myocardial infarction, hospitalization due to unstable angina, revascularization procedures, and death due to cardiovascular causes. The secondary goal was to evaluate the overall mortality rate.

Mean age of 50 years and a median CD4 count of 621 cells/mm³, a substantial majority, 5,250 out of 5,997, had levels of HIV RNA that were undetectable. The research was halted earlier than planned after a median observation period of 5.1 years because the results did not show the expected benefits. The incidence of major cardiovascular problems was lower in the group receiving pitavastatin, with 4.81 events / 1,000 person-years, in relation to 7.32 events / 1,000 person-years in the placebo group. Muscle-related complaints were noted in 2.3% of the pitavastatin group and 1.4% of the placebo group. Additionally, diabetes mellitus developed in 5.3% of those on pitavastatin and 4.0% of those on placebo. Therefore, during the median 5.1 years of follow-up, those with HIV who were administered pitavastatin

experienced a reduced risk of major adverse cardiovascular events compared to the placebo recipients.

Initially, the LDL cholesterol levels of both groups were similar. After 12 months, the median LDL cholesterol levels in the pitavastatin group decreased to 74 mg/dl, while in the placebo group, they reduced to 106 mg/dl.

Beyond the reduction of LDL cholesterol levels, statins are known to diminish immune inflammation and activation in individuals with HIV infection. This suggests that statins might confer additional health advantages to this demographic beyond merely lowering LDL cholesterol levels.

For HIV-positive individuals between the ages of 40 and 75 who have a 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimate of less than 20%, the Panel for the Use of Antiretroviral Agents in Adults and Adolescents with HIV advises the following:

- Begin moderate-intensity statin therapy for those with a 10-year ASCVD risk estimate ranging from 5% to less than 20%.
- The recommended moderate-intensity statin treatments include:
 - 4 mg of pitavastatin OD
 - 20 mg of atorvastatin OD
 - 10 mg of rosuvastatin OD

For individuals with HIV who have a 10-year risk of atherosclerotic cardiovascular disease (ASCVD) under 5%, the Panel recommends initiating at least moderate-

intensity statin therapy . The decision to start statin therapy in this group should take into account HIV-specific ASCVD risk factors, as the overall benefit from statins is relatively modest.

Therefor recommended moderate-intensity statin treatments are the same for those with 10-year ASCVD risk estimates between 5% and 20%.

For individuals younger than 40 YEARS : Current evidence does not support the use of statins for primary prevention of ASCVD in young adults with HIV. Instead, lifestyle changes are encouraged for this age group in the general population, with statin therapy reserved for particular subgroups.

For people between 40 and 75 years old who have a high (20% or greater) 10-year risk of atherosclerotic cardiovascular disease (ASCVD), it is recommended to start treatment with high-intensity statin therapy. Those in the age range of 20 to 75 years with low-density lipoprotein cholesterol (LDL-C) levels at or above 190 mg/dL should also receive high-intensity statin therapy, using the highest dose they can tolerate. Additionally, for individuals aged 40 to 75 with diabetes mellitus, initiating at least moderate-intensity statin therapy is advised, with further evaluation to determine if high-intensity statin therapy may be necessary

Key Consideration :-

- Interactions between certain statins and antiretroviral drugs can lead to severe complications when combined. For additional details, please refer to the section on Drug–Drug Interactions. Depending on the situation, it may be necessary to switch to an alternative statin, modify the dosage of the current

statin, or enhance the monitoring for potential side effects associated with statin use.

- Statin therapy should be discontinued for pregnant individuals with HIV, and initiation of such treatment should be deferred for those at low-to-intermediate risk for ASCVD during pregnancy. It is also recommended to avoid breastfeeding while on statin medication. Research from the University of Montreal Hospital Research Centre (CRCHUM) indicates that people with HIV undergoing antiretroviral therapy might find metformin beneficial in mitigating chronic inflammation, a drug typically used for managing type-2 diabetes.
- While individuals with HIV have experienced health improvements, they continue to be susceptible to chronic inflammation-related conditions such as cardiovascular disease. The ongoing activation of the immune system and the persistence of HIV in the memory T cells are the main contributors to these issues.
- Current antiretroviral drugs prevent the replication of HIV by stopping the virion, the complete virus particle, from exiting infected cells and infecting new ones. However, they do not address the replication of the viral genome within the infected cell itself. This replication leads to persistent immune activation and inflammation inside the cell, even with antiretroviral therapy, potentially leading to comorbidities like cardiovascular disease. Metformin has been utilized in treating non-diabetic individuals with HIV on antiretroviral therapy due to its ability to inhibit the mTOR protein, which plays a role in the intracellular replication of the HIV genome.

A 2017 investigation by Flavia Ballocca and colleagues into cardiovascular conditions in HIV patients highlighted that with the extended lifespan due to HAART, those with HIV are now facing additional comorbidities, notably cardiovascular diseases. The complexity of cardiovascular disease (CVD) in the HIV-positive population stems from a mix of conventional risk factors, infection-related elements, and antiretroviral therapy-related factors.⁽¹⁶⁰⁾

The research by Paraskevi C. Fragkou and others in 2023 on cardiovascular disease (CVD) and risk evaluation in HIV positive patients identified HIV infection as a significant risk factor for cardiovascular issues. Given the evolving nature of both CVD and HIV, it appears that people living with HIV (PLWH) may not fully benefit from standard CVD screening and treatment practices used in the general population. A notable concern is the increasing incidence of CVD in the aging PLWH demographic. It is clear that the optimal approach for managing PLWH involves personalized treatment and prevention strategies, along with a holistic plan for HIV-associated CVD.⁽¹⁶¹⁾

In 2013, Su Jin Jeong and colleagues conducted a study on clinical factors related to intima-medial thickness and carotid plaque in HIV-positive individuals. The study found a direct link between the occurrence of carotid plaque and elevated LDL-C levels, hypertension, and advanced age (≥ 40 years) in HIV-positive Koreans. It was also established that both maximum and mean intima-medial thickness (Max-IMT and Mean-IMT) are strongly associated with the occurrence of carotid plaque, and carotid intima-medial thickness (cIMT) is connected with traditional CVD risk factors in this population.⁽¹⁶²⁾

Jisun Oh and others in 2007 studied HIV-associated dyslipidemia, noting an uptick in cases among HIV patients, particularly those undergoing antiretroviral therapy. The link between antiretroviral drug use, HIV infection, and a heightened risk of cardiovascular disease is well-established, despite the complexities involved. Dyslipidemia management may involve dietary changes, exercise, adjusting secondary factors, switching antiviral therapies, and the judicious use of various pharmacological agents. Future research should aim to evaluate and comprehend the role of different drugs in treating dyslipidemia caused due to HIV infection and to explore more deeply the mechanisms of how it happens and optimal management for this significant adverse effect of HIV treatment.⁽¹⁶³⁾

Efforts to merge healthcare services for HIV and non-communicable diseases (NCDs) have been made by various countries, primarily through the inclusion of NCD prevention within HIV treatment frameworks, as highlighted in a 2021 research by Adeyemi O. et al. This approach is expected to enhance the reach of NCD services and potentially improve health outcomes for individuals with NCDs. Nonetheless, the integration poses a risk of disproportionate resource allocation, particularly with the recent reduction in HIV funding, which could compromise HIV service quality and result in poorer health outcomes for HIV patients and minimal gains for those with NCDs. It is crucial to implement thorough and timely monitoring and evaluation of these integrated programs, focusing on clinical outcomes at the patient level. Such measures will generate valuable insights into the benefits, challenges, and cost-effectiveness of the integration, which is particularly pertinent for countries where NCD services are nascent.⁽¹⁶⁴⁾

A 2018 study by Sasha A. Fahme on hypertension among HIV-positive individuals suggests that the condition is common and likely arises from a combination of risk factors, HIV-related factors, and antiretroviral therapy (ART). Unique pathophysiological mechanisms such as movement of microbes, ongoing inflammation, challenges in immune system function and its restoration, the virus's tendency to affect certain cells, fat distribution abnormalities, adipokines, and HIV-associated nephropathy are among the contributing factors to hypertension in those with HIV.⁽¹⁶⁵⁾

The research by Rosan A van Zoest et al., in 2016 suggested that alterations in body composition, including abdominal obesity and stavudine-induced peripheral lipoatrophy could be linked to the increased occurrence of hypertension in individuals infected with HIV-1.⁽¹⁶⁶⁾

The 2021 research by Sucharita Duttagupta et al. on the applicability of Western-developed Framingham and ASCVD risk scores in an Indian context found that both FRS and ASCVD scores are effective in assessing cardiovascular disease risk. The FRS, in particular, shows a significant association with the Syntax Score, which assesses coronary artery lesion complexity and severity, and outperforms the ASCVD score in risk prediction accuracy.⁽¹⁶⁷⁾

A study by Benoit Delabays et al. 2022 underscores the critical need for the development and validation of cardiovascular risk assessment tools for people living with HIV (PLWH), who exhibit twice the incidence rate of ASCVD compared to the HIV negative individuals. The study found that PLWH are less likely to meet LDL-C goals under lipid-lowering treatments compared to their counterparts in HIV negative individuals within the same risk bracket. The use of SCORE2 or PCE for ASCVD

prediction in PLWH is deemed appropriate due to their simpler set of variables, as opposed to more complex scores that include HIV-specific factors. The addition of HIV-specific criteria did not significantly enhance the performance of scores designed for the general populace.⁽¹⁶⁸⁾

The 2010 study by Emanuele Fanales Belasio et al. delves into the intricate processes involved in the cellular infection and subsequent breakdown of the immune system by HIV. The research primarily focuses on the virus's composition, traits, and its interaction with the host. Enhancing our grasp of HIV's structure, its genetic diversity, replication process, and the host's immune response is vital. This ongoing advancement in knowledge is essential for identifying pivotal elements in the virus's lifecycle, which could be instrumental in creating new diagnostic tools, targeted antiviral treatments, and vaccines. The paper provides a comprehensive review of HIV's molecular architecture, replication dynamics, and pathogenesis, emphasizing aspects that are critical for the formulation of diagnostic tests.⁽¹⁶⁹⁾

MATERIALS AND METHODS

Source of Data: Inpatient and outpatient department at DR.Kle Prabhakar Kore Charitable Hospital were the source.

Study Design: Cross sectional

Study Period: 1 YEAR - 1st Jan 2023 to 31st December 2023

Sample Size: Sample size at 95% confidence interval

20% allowable error and 10% attrition

$$n = \frac{Z^2_{1-\alpha/2} \times SD^2}{(20\% \text{ of } SD)^2} \times 1.1$$

$$N = 105.6 = \mathbf{106}$$

N=Sample size

SD = Standard deviation

Z_{2 1-ALPHA/2}= Confidence interval

106 TOTAL SAMPLE SIZE

70 = HIV POSITIVE INDIVIDUALS

36 = NORMAL INDIVIDUALS

Sampling technique: Upon admission, all participants provided their written agreement, confirming they were fully informed before participating.

Inclusion Criteria: All participants with HIV status positive (naive and on treatment) were included in this study.

Exclusion Criteria:

1. People with HIV positive status but age <18
2. Established cases of Myocardial Infarction, Stroke, Peripheral vascular disease.
3. Expectant mothers identified with cardiovascular risk factors linked to pregnancy, like preeclampsia or gestational high blood pressure, are part of the exclusion criteria.

Study protocol: At KLE Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi, individuals underwent a screening process. Those meeting the specified criteria for inclusion and exclusion were selected to participate in the research.

With the approval of the ethical committee and those who provided written informed consent was taken from all the participants enrolled in this study.

Data collection procedure:

- One year cross sectional study
- Patients provided their informed consent for participation in the study.
- Inpatient and outpatient department HIV infected individuals (naïve and on treatment) will be identified
- Detailed history and examination would be done.

History includes:- Age of the patient, Gender, Past history of Diabetes, High blood pressure, and Abnormal lipid levels, along with a Family history of HIV and cerebrovascular accidents (strokes), were included.

Habits like Smoking or Alcoholism.

Treatment history with drugs like Antihypertensives, statin or aspirin.

The examination includes systolic and diastolic blood pressure.

General Physical Examination included Cardiovascular System, Central nervous System, Respiratory system and Per Abdomen system.

Fasting Lipid profile which included Total Cholesterol levels , HDL Cholesterol Levels, LDL Cholesterol levels, Triglyceride along with other Routine investigations were sent.

Calculation of cardiovascular risk by ASCVD and Framingham Score

Data Of Each Patient Will Be Collected On A Proform.

STATISTICAL ANALYSIS

Data entry was conducted using a Microsoft Excel spreadsheet, while analysis was performed with SPSS software, version 22. Categorical data was depicted using frequency counts and percentage distributions. The chi-square test served as the significance test for categorical variables. Continuous data was summarized using the mean and standard deviation. The Paired T-test was utilized to assess the mean difference between the periods before and after the intervention. A statistical P value less than 0.05 is deemed indicative of statistical significance. R-Squared (R^2 or the coefficient of determination) value was used to determine the proportion of variance in the dependent variable that can be explained by the independent variable. The F statistic, or F value was used to compare variance between groups

RESULTS

Table 1 - Baseline Characteristics of the included participants

	HIV Infected (N=70)	Control Population (N=36)	p value
	N (%)	N (%)	
HIV Infected			
Yes	70	-	
No	-	36	
HIV Disease Duration (years) ^a	-	-	
Gender			< 0.05
Male	47 (67.14)	25 (69.4)	
Female	23 (32.86)	11 (30.6)	
Age			1
40-49	40 (57.14)	19 (52.8)	(Not significant)
50-59	25 (35.7)	13 (36.1)	
60-70	5 (7.14)	4 (11.1)	
Diabetes			< 0.05
Present	11 (15.71)	10 (27.8)	
Absent	59 (84.29)	25 (72.2)	
Hypertension			< 0.05
Present	19 (27.14)	16 (44.4)	
Absent	51 (72.86)	20 (55.6)	
Systolic Blood Pressure (mmHg) ^a	119.7 (±16.2)	127.5 (±11.2)	0.1091 > 0.05 – (not significant)
Diastolic Blood Pressure (mmHg) ^a	75.6 (±10.1)	77.5 (±9.2)	
Antihypertensive			< 0.05
Present	28(40)	17(47.2)	
Absent	42(60)	19(52.8)	
Dyslipidemia			< 0.001
Present	0 (0)	0 (0)	
Absent	70 (100)	36 (100)	
Smoking			< 0.001
Present	14(20)	3(8.3)	
Absent	56(80)	33(91.7)	

Alcohol			< 0.05
Present	20 (28.57)	4 (11.1)	
Absent	50 (71.43)	32 (88.9)	
ASCVD score			< 0.05
Low	39(55.7)	34(94.4)	
Borderline	8(11.43)	2(5.6)	
Intermediate	17(24.29)	0	
High	6(8.57)	0	
Framingham score			< 0.05
Low	46(65.71)	22(61.1)	
Medium	9(12.86)	11(30.6)	
High	15(21.43)	3(8.3)	
Total Cholesterol			< 0.05
<200	64(91.43)	31(86.1)	
>200	6(8.57)	5(13.9)	
HDL cholestrol			< 0.05
<40	53(75.71)	17(47.2)	
>40	17(24.28)	19(52.8)	
LDL cholestrol			< 0.05
<100	53(75.71)	20(55.6)	
>100	17(24.29)	16(44.4)	
Triglyceride			0.284 > 0.05
<150	41(58.57)	32(88.9)	
>150	29(41.43)	4(11.1)	

Table 2 - Baseline Characteristics of the included participants

HB (g/dl)	11.52 (±2.4)	11.65 (±2.45)
PCV (%) ^a	36.45 (±5.84)	36.25 (±7.4)
RBC (10⁶/uL) ^a	3.91 (±1.05)	3.94 (±1.15)
Platelet (10⁶/ul) ^a	2.52 (±1.2)	2.46 (±1.16)
Total Count (10³/uL) ^a	9.88 (±5.9)	8.98 (±3.8)
Urea (mg/dl) ^a	30.82(±23.7)	29.96(±21.7)
Creatinine (mg/dl) ^a	1.35 (±1.33)	1.07 (±0.49)
Total Bilirubin (mg/dl) ^a	0.98 (±0.9)	1.35 (±1.49)
Direct Bilirubin (mg/dl) ^a	0.44 (±0.5)	0.62 (±0.5)
Indirect Bilirubin (mg/dl) ^a	0.56 (±0.58)	0.72 (±0.68)
SGOT (U/L) ^a	51.41 (±42.8)	40.17 (±29.8)
SGPT (U/L) ^a	31.85 (±21.1)	23.64 (±11.45)
ALP (U/L) ^a	109.8 (±60.78)	108.8 (±53.62)
Total Protein (gm/dl) ^a	8.34(±9.4)	7.19(±1.12)
Sodium (mEq/L) ^a	133.87(±5.24)	133.8(±4.54)
Potassium (mEq/L) ^a	4.15(±0.64)	3.85(±0.74)
Bicarbonate (m mol/L) ^a	20.7(±12.56)	20.66(±3.8)
Chloride (mEq/L) ^a	100(±5.51)	97.23(±15.53)
FBS (mg/dl) ^a	118.49(±42.5)	109.42(±56.89)

• ^a – Mean (SD)

• N – Sample size

ASCVD vs Framingham Score

Table 3 - Risk stratification in ASCVD AND FRAMINGHAM SCORE

	HIV Infected (N=70)		Control Population (N=36)	
Framingham score	11.38($\pm$ 0.07) %	Moderately high risk	9.18($\pm$ 0.29) %	Moderately high risk
ASCVD score	6.67($\pm$ 0.1) %	Borderline risk	2.11($\pm$ 0.6) %	Low risk

Table 4 - ASCVD and Framingham score with significant p value

	ASCVD	Framingham	P value
HIV Infected	6.67($\pm$ 0.1) %	11.38($\pm$ 0.07) %	< 0.05
Control Population	2.11 ($\pm$ 0.6) %	9.18 ($\pm$ 0.29) %	

ASCVD Score in HIV vs Control group

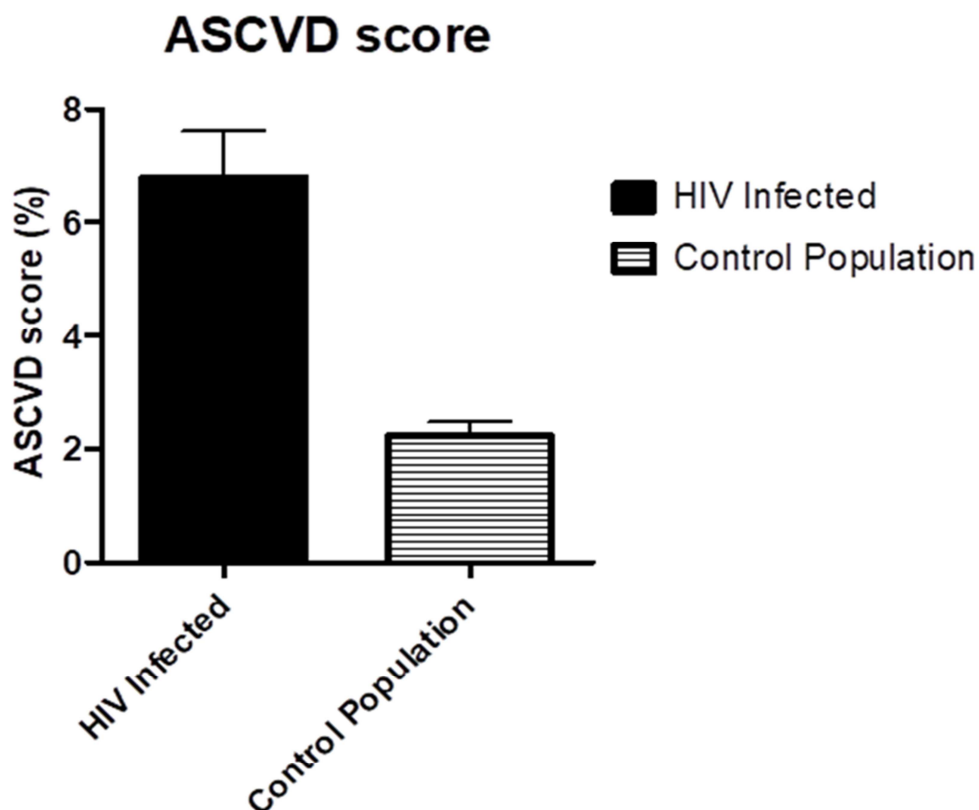


Figure 6 - ASCVD Score in HIV vs Control group

The analysis reveals significant differences in ASCVD Score among the two groups. The Control Population has significantly lower ASCVD score (2.11%) compared to the HIV Infected group (6.67%) with p value of 0.0006 ($P < 0.05$). Despite the R square value indicating that only 6.821% of the variability is explained by group differences. The difference between the Control Population and the HIV Infected Population is even more significant at the 0.1% level.

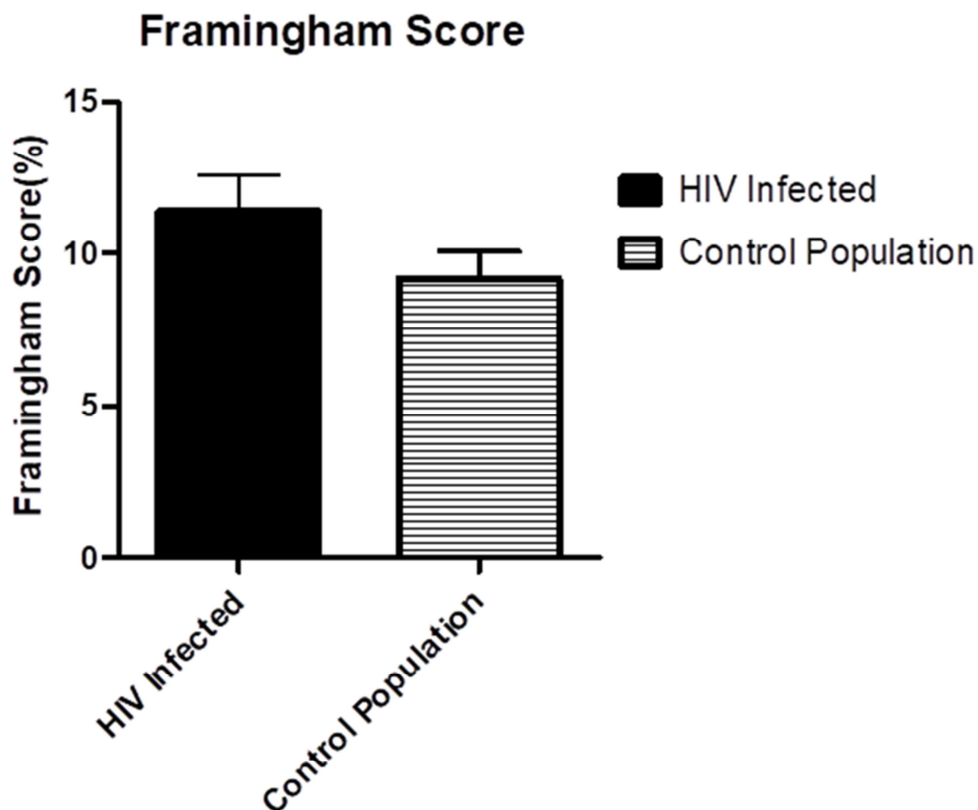
Framingham Score in HIV vs Control group

Figure 7 - Framingham Score in HIV vs Control group

The statistical analysis shows that the Framingham Scores between the two groups have a significant difference overall with P value- 0.0494. The F statistic is 3.1172, suggesting some variation among the group means. The R square value of 0.02121 indicates that about 2.121% of the variability in the Framingham Scores is explained by the differences between the groups. The HIV Infected group has a higher Framingham Score (11.38%) compared to the Control Population (9.18%)

ASCVD vs Framingham Score in HIV vs Control group

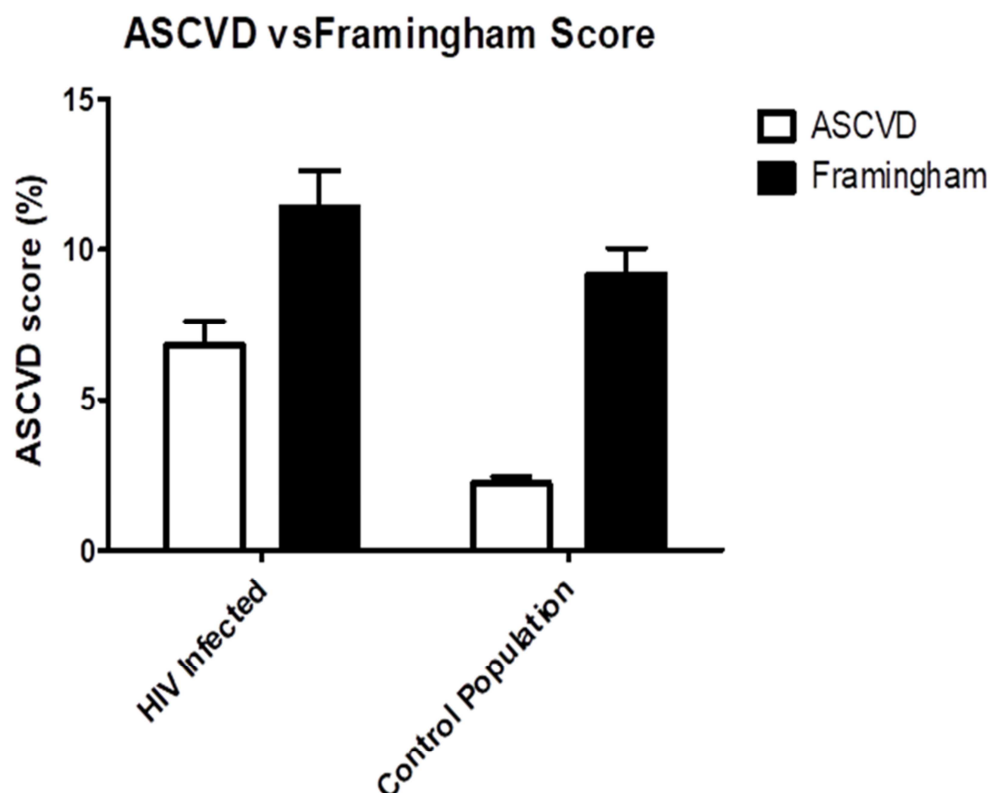


Figure 8 - ASCVD vs Framingham Score in HIV vs Control group

This analysis demonstrates notable differences in ASCVD and Framingham scores among the HIV Infected individuals, and the Control Population. The means of ASCVD and Framingham scores vary significantly across the two populations, as indicated by the P value- 0.004 (<0.05), R square- 0.8987 and the 95% confidence interval. The high R-squared value indicates a strong relationship between ASCVD and Framingham scores, suggesting that Framingham scores can predict a substantial portion of the variability in ASCVD scores across the populations studied. HIV Infected individuals have high ASCVD and Framingham score (6.67% ;11.38%) as compared to the Control Population (2.11% ; 9.18%).

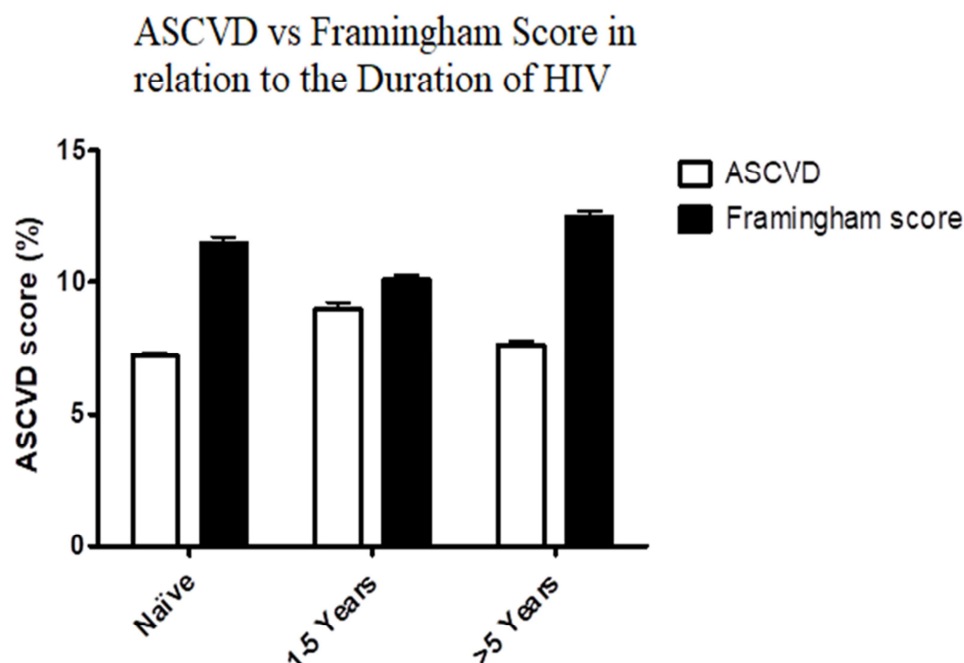
ASCVD vs Framingham Score in relation to the Duration of HIV

Figure 9 - ASCVD vs Framingham Score in relation to the Duration of HIV

The analysis reveals a statistically significant difference between ASCVD and Framingham Scores among HIV-infected individuals, contingent upon the duration of HIV infection. The duration of HIV infection substantially impacts the variance in these scores, as evidenced by a high R-squared value of 0.7939, indicating that approximately 79.39% of the variance in the comparison between ASCVD and Framingham Scores is attributable to the duration of HIV infection. Furthermore, the P-value of 0.0172 signifies a significant difference in scores based on infection duration. When comparing ASCVD Score versus Framingham Score in relation to the duration of HIV infection within the HIV-infected population, it is evident that the score difference between ASCVD and Framingham is significantly higher in individuals with HIV infection lasting more than 5 years.

ASCVD vs Framingham Score in relation to the HIV Treatment

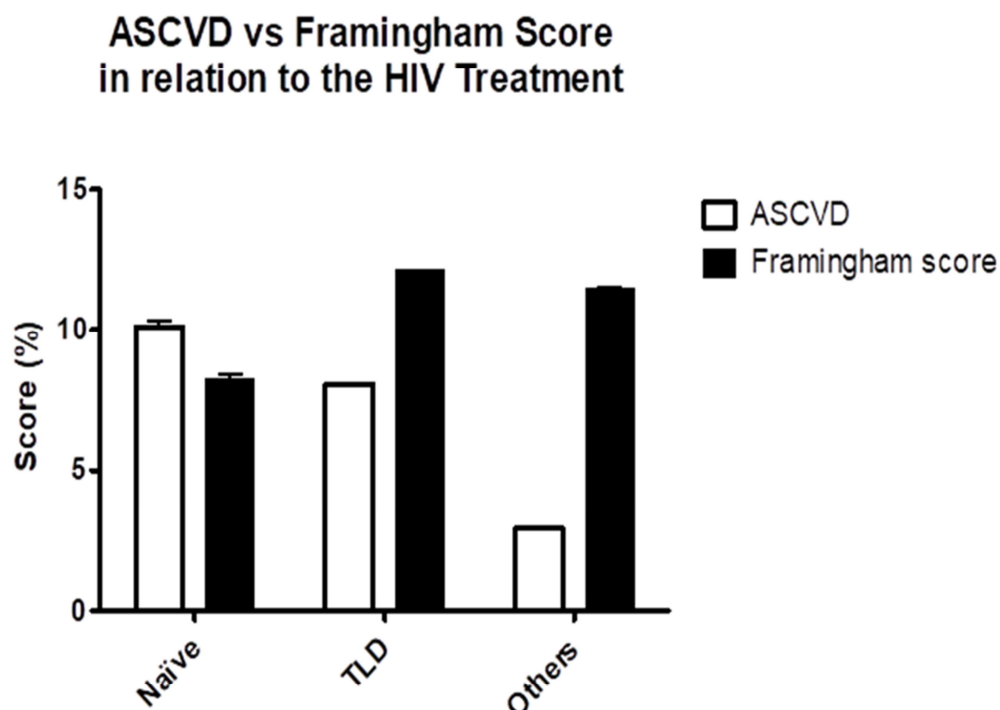


Figure 10 -ASCVD vs Framingham Score in relation to the HIV Treatment

The statistical comparison between ASCVD and Framingham scores among different treatment groups for HIV-infected individuals reveals that the score difference between ASCVD and Framingham is significantly higher in individuals receiving TLD treatment (8.1% ; 12.11%) compared to naive group and other group (10.13% ; 8.25% and 2.95% ; 11.44%). The P-value of 0.0454 indicates a statistically significant difference between ASCVD and Framingham scores ($P < 0.05$) in the context of treatment types for the HIV-infected group. The R-squared value of 0.3436 suggests that approximately 34.36% of the variability in the risk scores can be explained by the difference between ASCVD and Framingham scores.

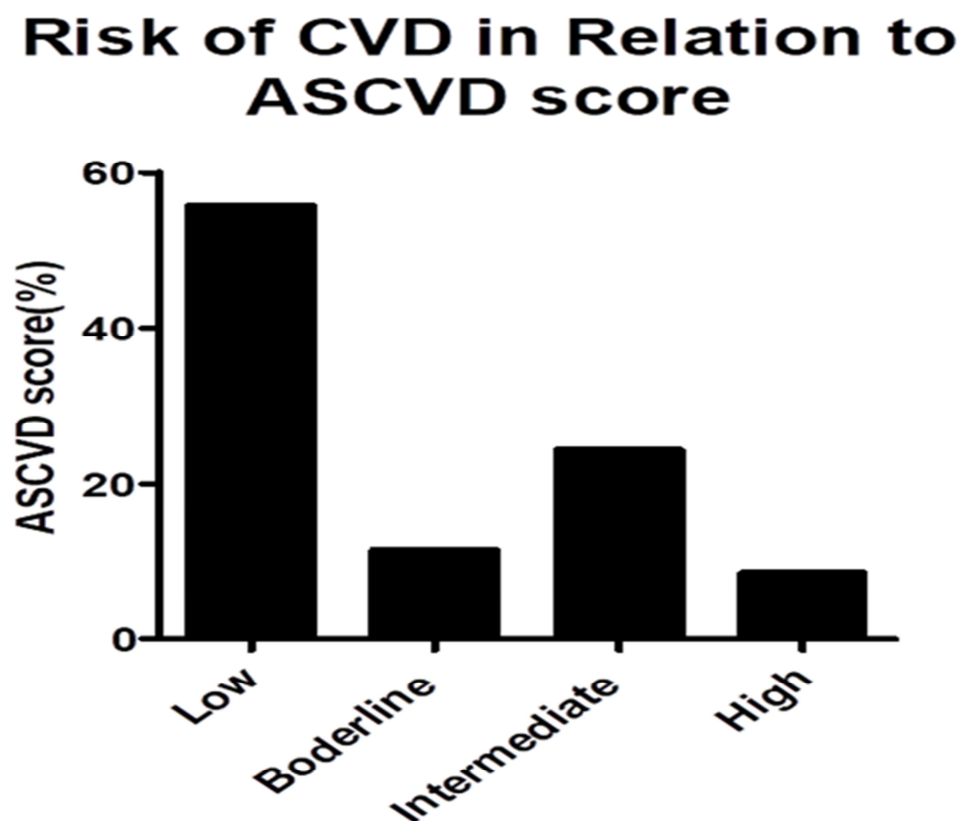
Risk of CVD in Relation to ASCVD score

Figure 11- Risk of CVD in Relation to ASCVD score

When comparing the risk of CVD in relation to the ASCVD score in the HIV-infected population, it is evident that a significant proportion of this population (55.7%) has a low CVD risk. This is followed by those with intermediate risk (24.29%), borderline risk (11.43%), and high risk (8.57%).

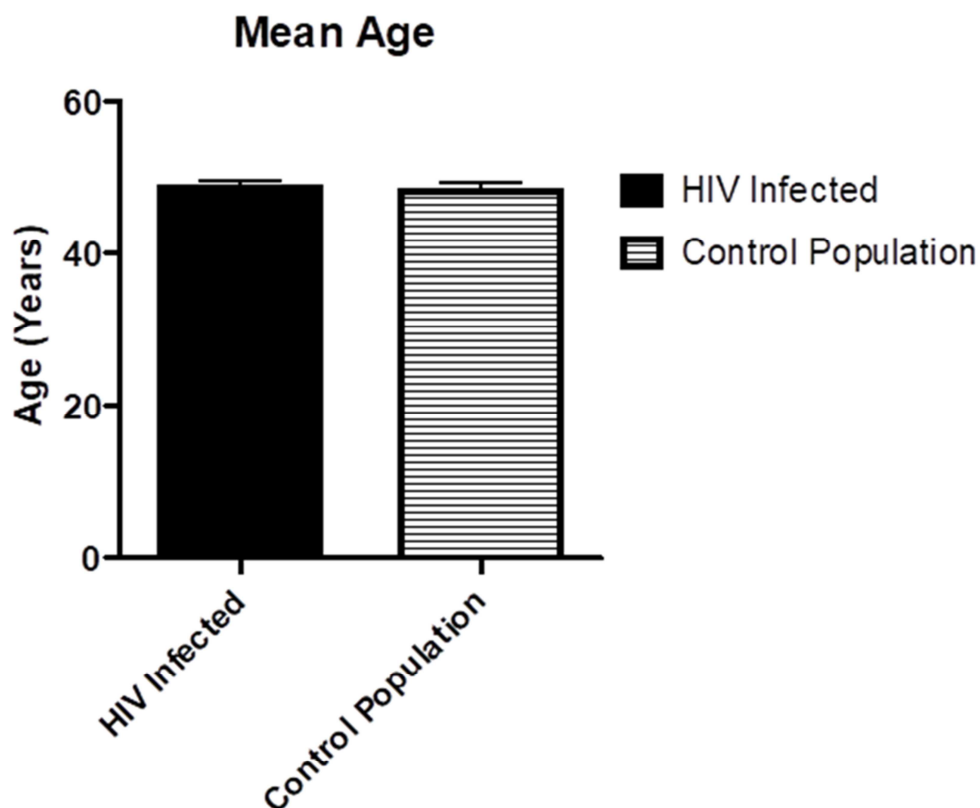
Mean Age in HIV vs Control group

Figure 12 -Mean Age in HIV vs Control group

The analysis shows that there are no statistically significant differences in the mean ages of the HIV-infected individuals, and the Control Population. This suggests that the age distribution is fairly consistent across two groups. The R-squared value of 0.01136 indicates that only 1.136% of the variability in age can be explained by group differences, which is very low. Thus, any observed differences in mean ages are likely due to random variation rather than true differences between the groups.

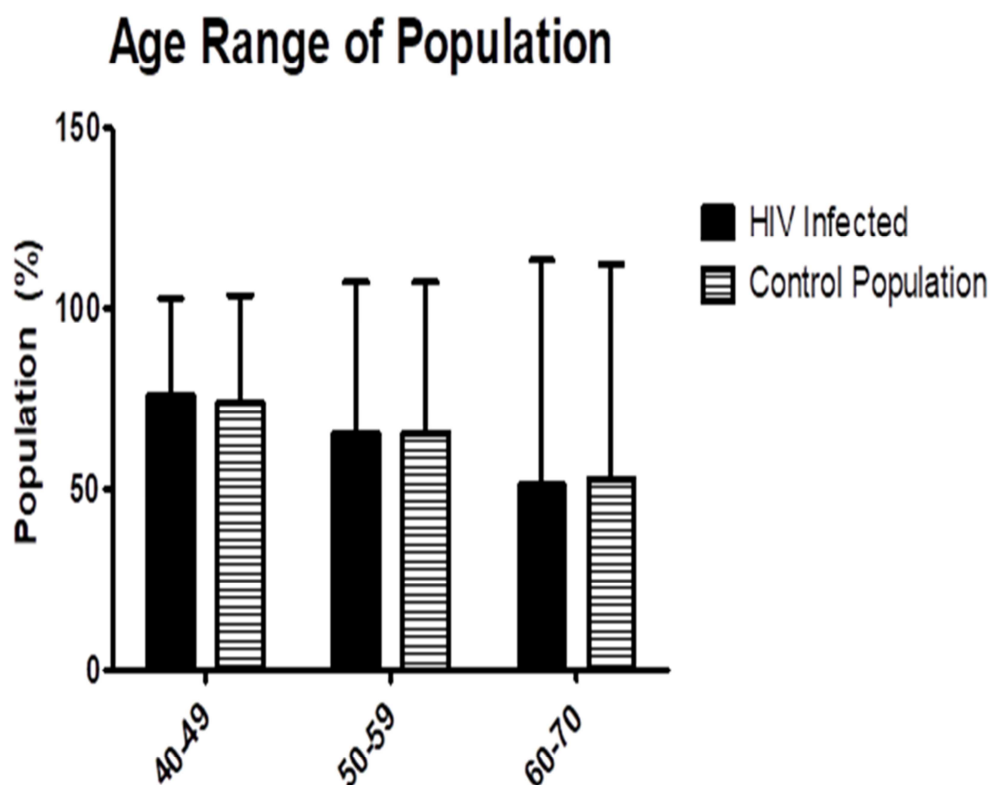
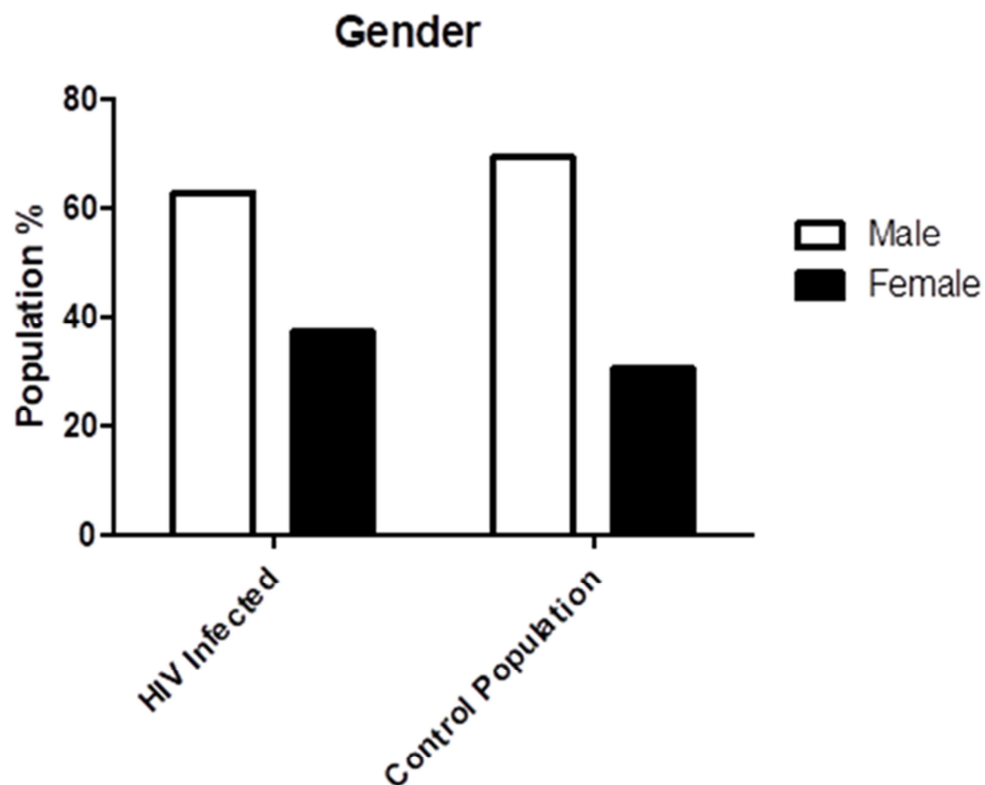
Age Range of Population in HIV vs Control group

Figure 13 - Age Range of Population in HIV vs Control group

The analysis reveals no significant differences in the age ranges of 40-49, 50-59, and 60-70 among the HIV Infected individuals and Control Population. The P value of 1 and extremely low F statistic suggest that any observed differences are not statistically significant and are likely due to random variation.

Gender in HIV vs Control group**Figure 14 - Gender in HIV vs Control group**

The statistical analysis reveals significant differences in gender distribution among the two populations, with a highly significant P-value of 0.0003. Specifically, there is a notable disparity in the proportions of males and females across these groups. Across HIV and control groups, the male proportion is significantly higher (67.14% and 69.4%) than the female proportion (32.86% and 30.60%).

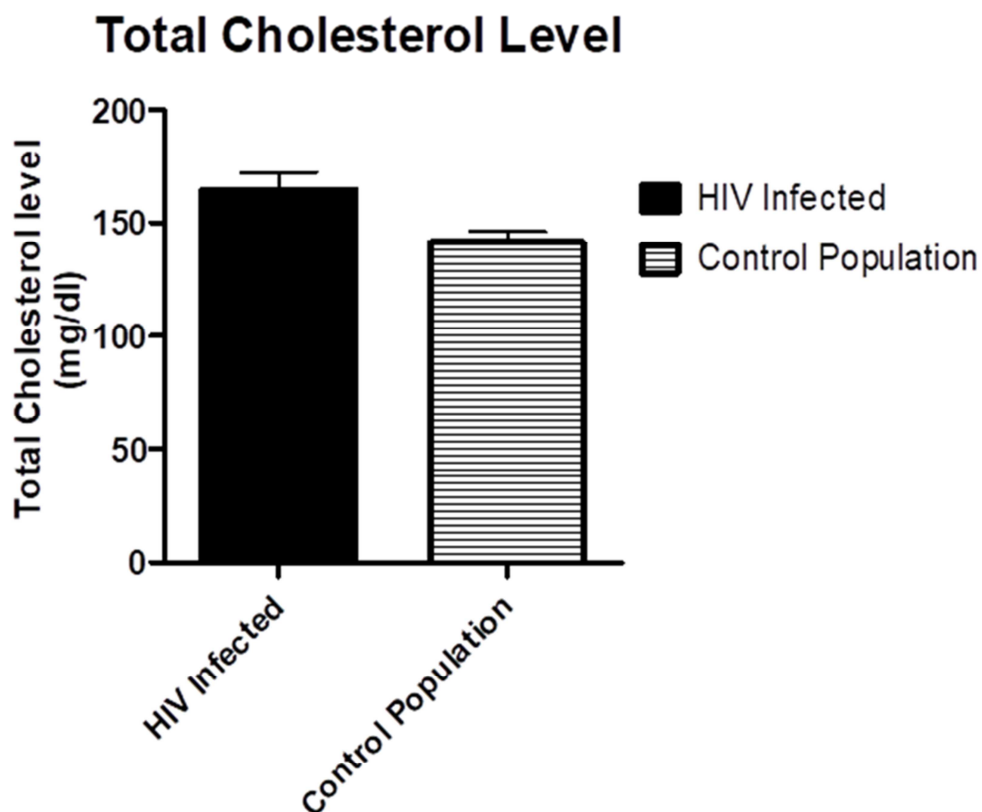
Total Cholesterol Level in HIV vs Control group

Figure 15 - Total Cholesterol Level in HIV vs Control group

The analysis demonstrates significant differences in Total Cholesterol Levels among the two populations, with a P-value of 0.0179. The HIV-infected group exhibits higher cholesterol levels (164 mg/dl) compared to the Control Population (141 mg/dl). The R-squared value of 0.3778 implies that approximately 37.78% of the variability in Total Cholesterol Levels can be attributed to the differences between the groups.

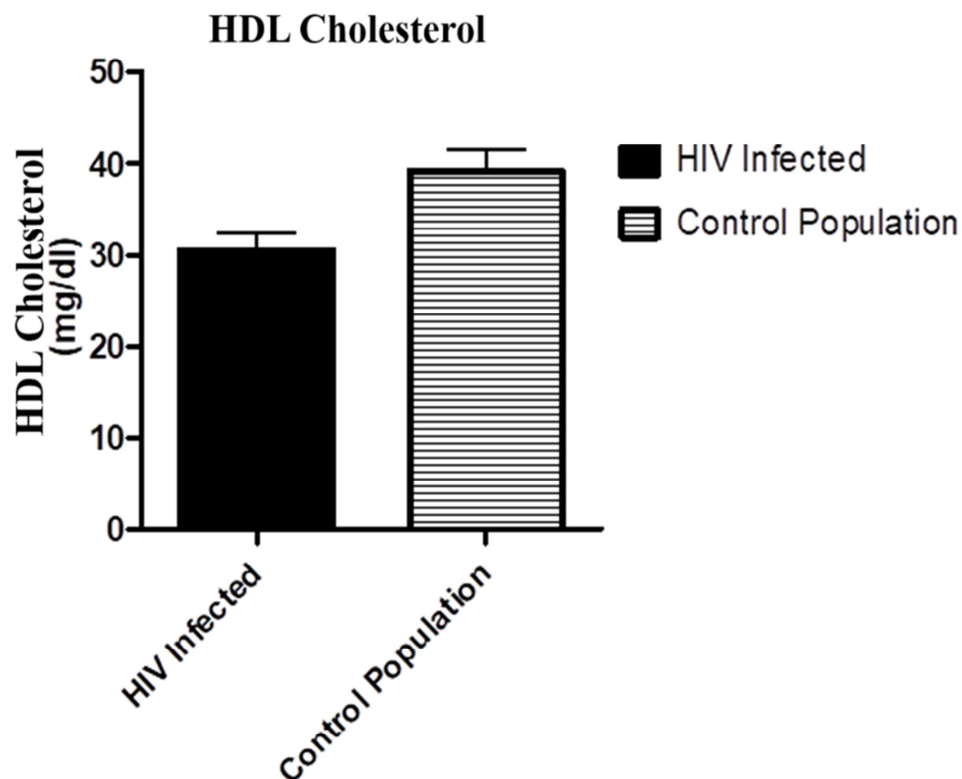
HDL Cholesterol Level in HIV vs Control group

Figure 16 - HDL Cholesterol Level in HIV vs Control group

The analysis demonstrates a significant overall difference in HDL cholesterol levels among the two populations, with a P value of 0.0261. The HDL cholesterol level in the Control Population (39mg/dl) is significantly higher than the HIV Infected group (30mg/dl). The R square value of 0.03429 indicates that approximately 3.429% of the variability in HDL cholesterol levels is explained by the differences among the groups.

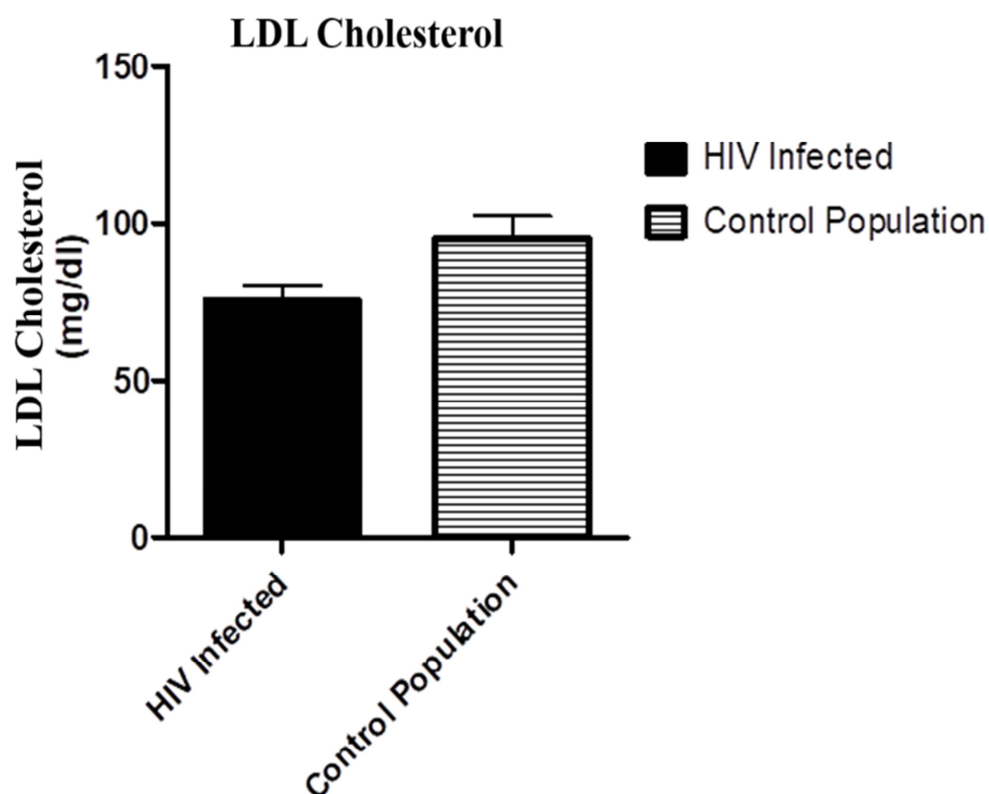
LDL Cholesterol Level in HIV vs Control group

Figure 17 - LDL Cholesterol Level in HIV vs Control group

The analysis demonstrates a significant overall difference in LDL cholesterol levels among the two populations, with a P value of 0.0226. The LDL cholesterol level in the Control Population (95 mg/dl) is significantly higher than the HIV Infected group (75 mg/dl). The R square value of 0.04897 indicates that approximately 4.897% of the variability in LDL cholesterol levels is explained by the differences among the groups.

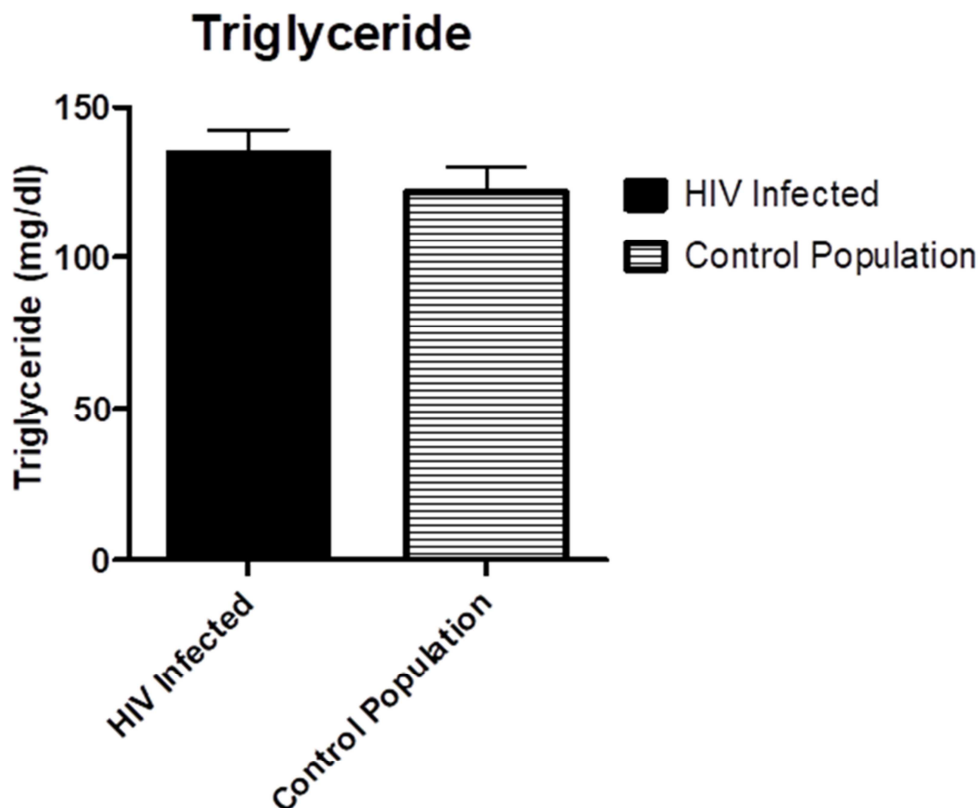
Triglyceride Comparison in HIV vs Control group

Figure 18 - Triglyceride Comparison in HIV vs Control group

The analysis reveals no significant differences in the triglyceride level among the HIV Infected individuals and Control Population. The P value of 0.2843 and extremely low F statistic suggest that any observed differences are not statistically significant and are likely due to random variation.

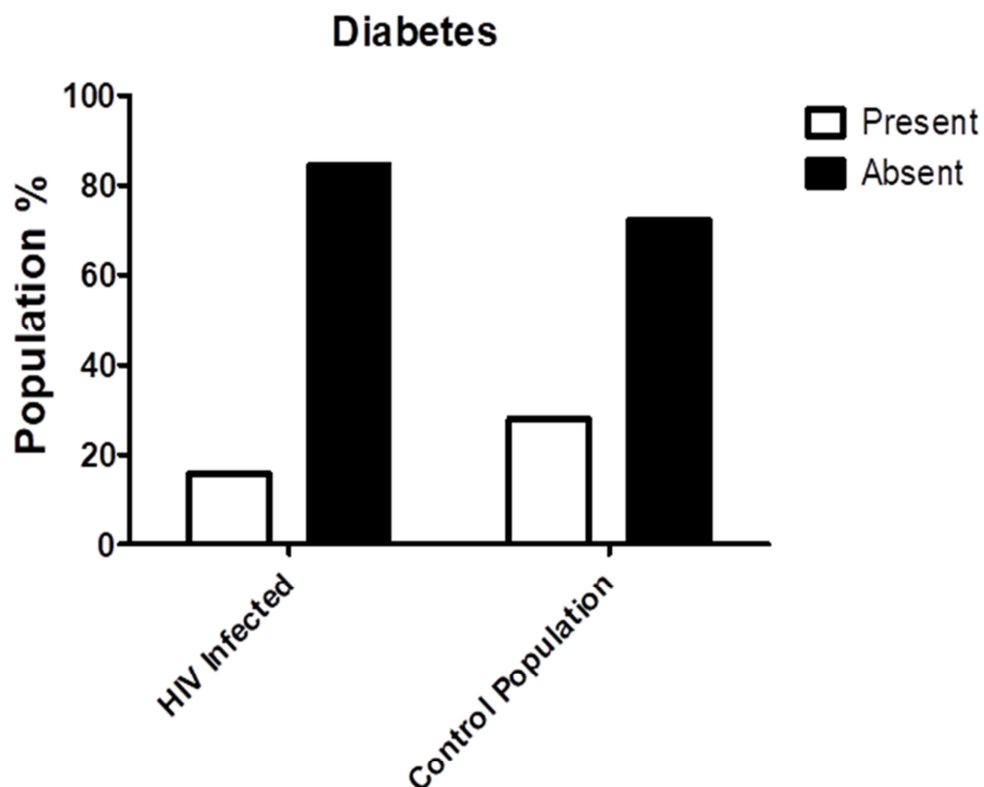
Diabetes in HIV vs Control group

Figure 19 -Diabetes in HIV vs Control group

The statistical analysis reveals notable differences in the prevalence of diabetes across the two populations. While diabetes is present in a substantial portion of each group, the Control Population (27.8%) exhibits the highest prevalence compared to HIV infected population (15.71%). The P-value of 0.0004 suggests a significant difference in the prevalence of diabetes among these populations.

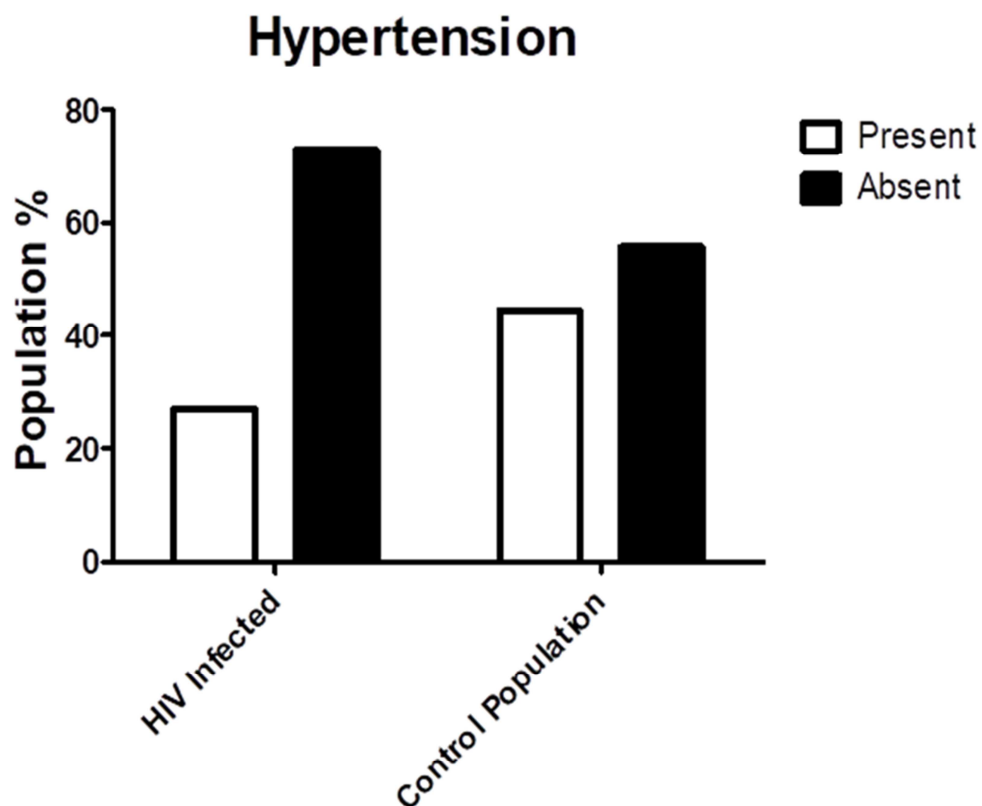
Hypertension in HIV vs Control group

Figure 20 -Hypertension in HIV vs Control group

The study demonstrates notable disparities in the prevalence of hypertension among the two populations. HIV Infected individuals exhibit the lowest prevalence of hypertension at 27.14%, and Control Population shows the highest prevalence at 44.44%. The statistical analysis confirms the significance of these differences, with a P value of 0.0313.

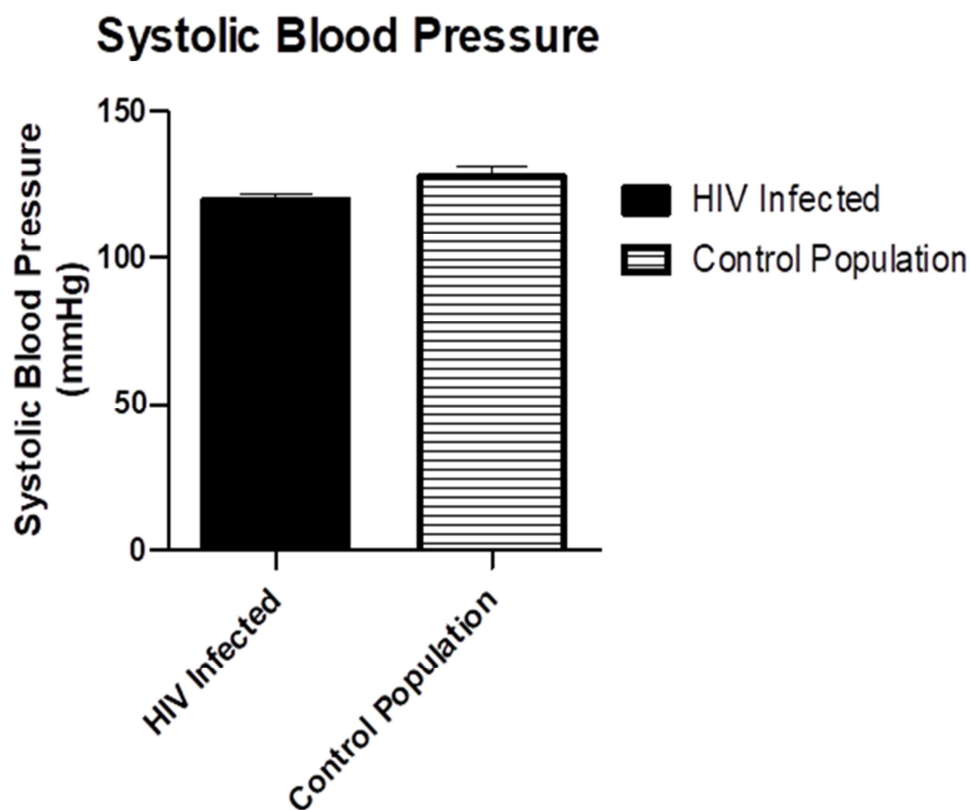
Systolic Blood Pressure in HIV vs Control group

Figure 21 - Systolic Blood Pressure in HIV vs Control group

When comparing systolic blood pressure among different population groups, it is evident that there are no statistically significant differences. Any observed differences in systolic blood pressure are likely due to random variation rather than true differences between the groups.

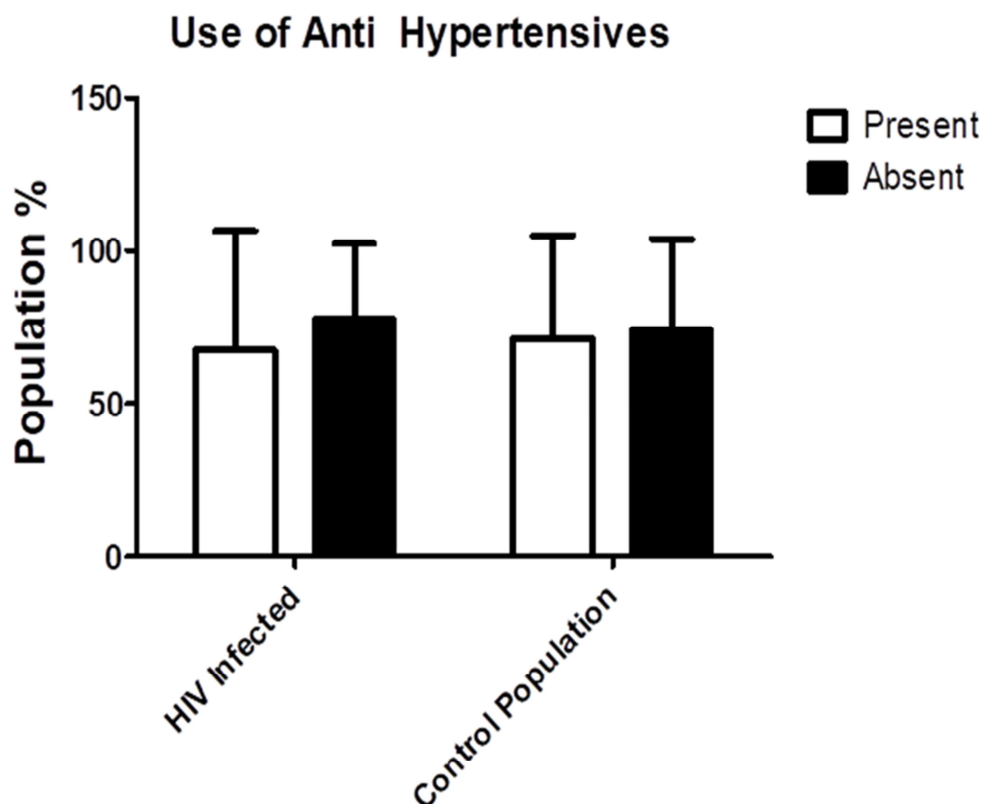
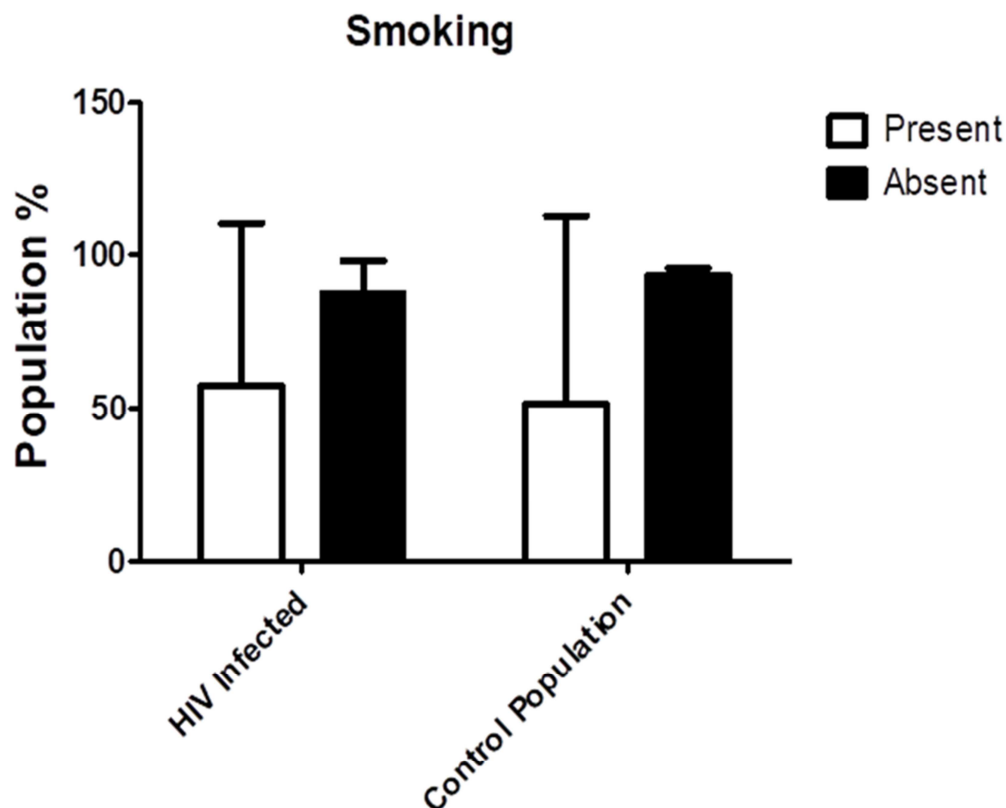
Use of Antihypertensives in HIV vs Control group

Figure 22 -Use of Antihypertensives in HIV vs Control group

The findings indicate that there are discernible differences in the usage of antihypertensive medication among the two populations with P value of 0.0107. Notably, the prevalence of antihypertensive medication usage appears to be highest among HIV Infected individuals, followed by the Control Population.

Smoking in HIV vs Control group

**Figure 23 -Smoking in HIV vs Control group**

The comparison of smoking habits among the two populations highlights notable disparities. The statistical analysis indicates a significant difference in smoking prevalence among the two groups with a p-value of 0.0001, which suggests a highly significant difference in smoking habits across the populations. HIV Infected individuals demonstrate the highest prevalence of smoking (20%) , followed by the Control Population(8.3%).

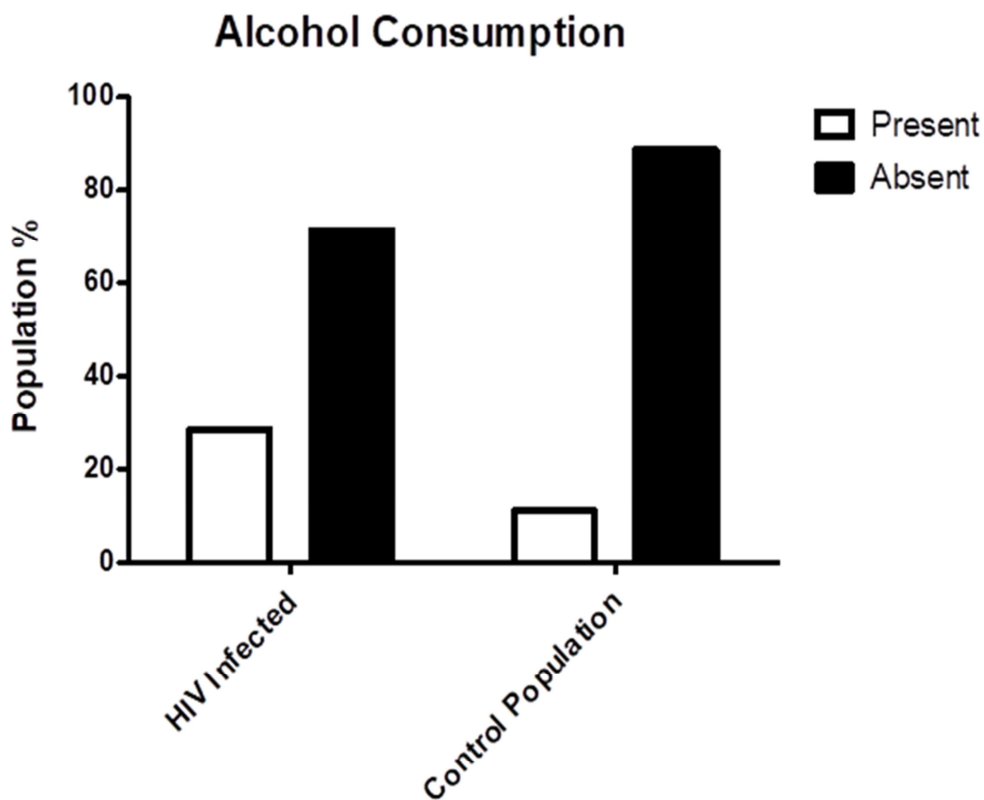
Alcohol Consumption in HIV vs Control group

Figure 24 - Alcohol Consumption in HIV vs Control group

This analysis reveals significant differences in alcohol consumption rates among the two populations with a p-value of 0.0013. It highlights that HIV Infected individuals have the high prevalence of alcohol consumption at 28.57%, followed by the Control Population at 11.11%. The statistically significant P value suggests that these differences are not due to random chance and reflect true disparities in alcohol consumption across these groups.

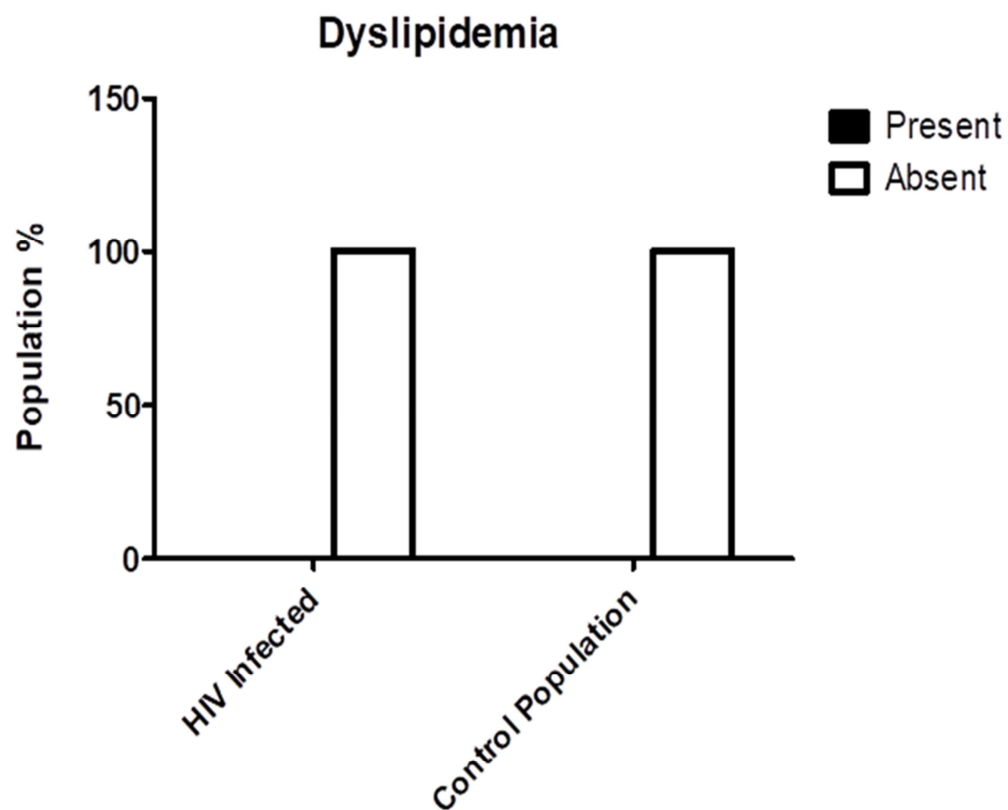
Family history of Dyslipidemia in HIV vs Control group

Figure 25 -Family history of Dyslipidemia in HIV vs Control group

None of the individuals had a family history of dyslipidemia.

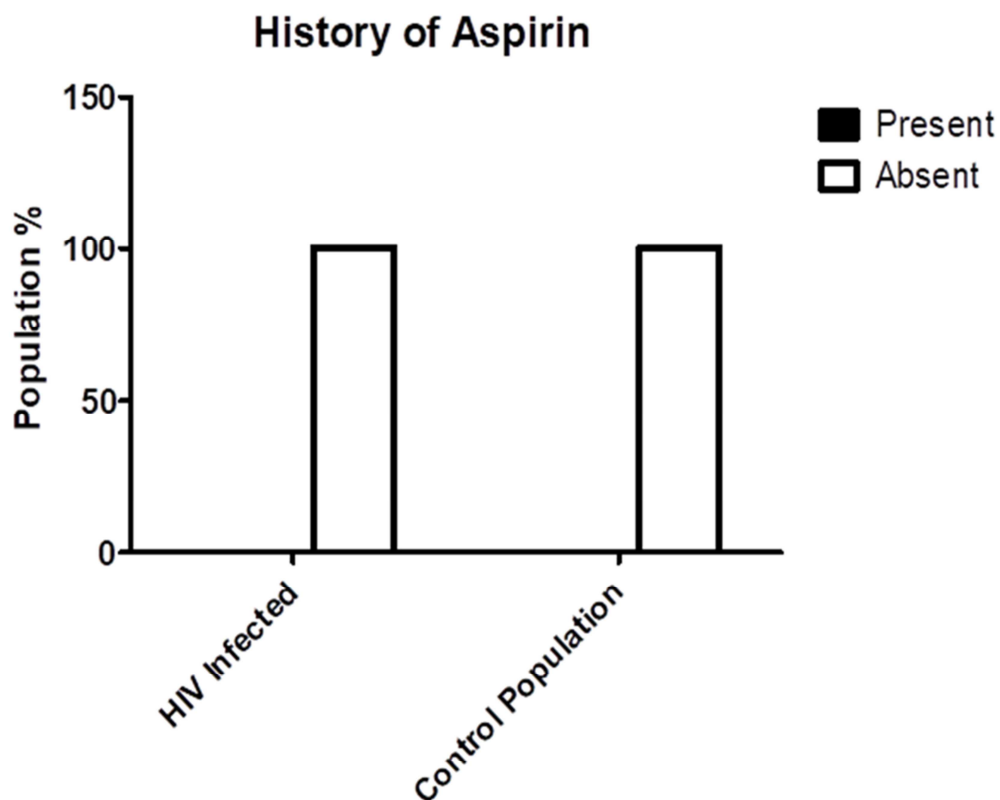
History of Aspirin in HIV vs Control group

Figure 26 - History of Aspirin in HIV vs Control group

None of the individuals were on aspirin in either group.

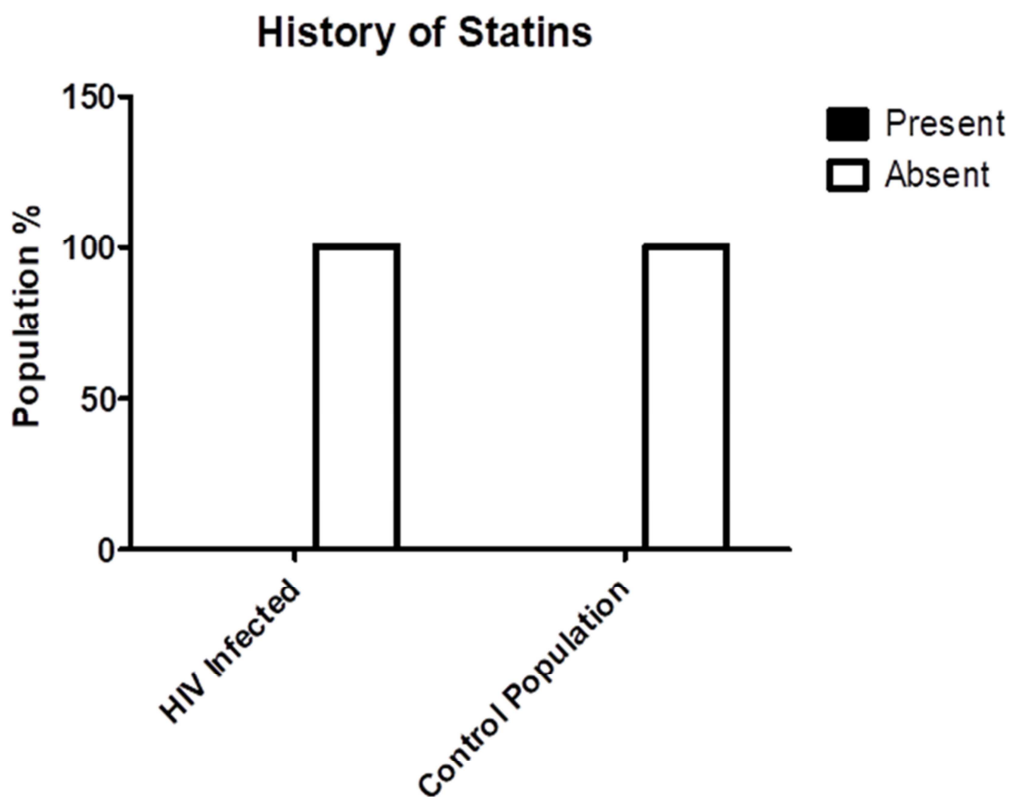
Statins Consumption in HIV vs Control group

Figure 27 - Statins Consumption in HIV vs Control group

None of the individuals were on statins in either group

DISCUSSION

This study was designed to evaluate cardiovascular risk using the ASCVD score within the HIV-positive population and to compare it with the Framingham score. The study included 106 participants, with 70 HIV-positive individuals and 36 in the control group. The average age among both HIV-positive and control groups was 48 years. The gender distribution was 67.14% male and 32.86% female in the HIV group, and 69.44% male and 30.56% female in the control group.

The findings indicated that the control group had a significantly lower ASCVD score, with a p-value of 0.006 ($P < 0.05$), suggesting a low risk at 2.11%, compared to the HIV-positive group's borderline risk score of 6.67%. This suggests a higher prevalence of cardiovascular disease among HIV-positive individuals, potentially due to systemic inflammation. The disparity in cardiovascular risk between the control and HIV-positive groups was even more pronounced at the 0.1% significance level. In individuals with HIV, an elevated ASCVD score may be attributed to several factors. Persistent chronic inflammation and immune system activation can occur leading to atherosclerosis. Additionally, HIV-related risk factors such as specific ART medications, and other comorbid conditions contribute to cardiovascular risk. Moreover, conventional risk factors for ASCVD, including smoking and dyslipidemia, also play a significant role in increasing the risk of cardiovascular disease in HIV-positive patients.

A related study by Robert S et al., conducted in 2019 in the US, found an increased risk of atherosclerotic cardiovascular outcomes in HIV-positive American

adults compared to non-HIV individuals. The ASCVD score was 5.53% in the HIV-positive cohort versus 3.49% in the control group.⁽¹⁷⁰⁾

Table 5 - Comparing our study with Robert S et al., study i/v/o CVD risk using ASCVD scoring in HIV vs Controls

	OUR STUDY	ROBERT S ET AL STUDY
HIV POSITIVE	70	82,426
CONTROLS	36	3,29,704
ASCVD SCORE IN HIV POSITIVE	6.67%	5.53%
ASCVD IN CONTROLS	2.11%	3.49%

In a study conducted by Kingery et al., 2016 in Tanzania reports lifetime and 10 year ASCVD risk and prevalence of metabolic syndrome was higher in HIV individuals compared to Non - HIV individuals. A significantly larger proportion of HIV-positive adults were identified as having a high risk for cardiovascular disease (CVD) over their lifetime (34.7%) compared to their HIV-negative counterparts (17.0%, $p < 0.001$). Moreover, the occurrence of metabolic syndrome was more frequent among the HIV-positive adults on ART (21.3%) than in the HIV-negative group (7.8%, $p = 0.008$), with two predominant patterns of risk factors being noted.⁽¹⁷¹⁾

In our study When comparing the risk of CVD in relation to the ASCVD score in the HIV-infected population, it is evident that a significant proportion of this population (55.7%) has a Low CVD risk. This is followed by those with intermediate risk (24.29%), borderline risk (11.43%), and high risk (8.57%). These study results were similar to a study conducted by Titus Msoka et al., 2021 in Tanzania in which

ASCVD algorithm has shown that a considerable number of individuals are categorized based on their risk levels, with a majority being placed in the Low-risk group (52.9%), followed by intermediate (17.3%) and high-risk (29.8%) categories. This classification helps in identifying individuals who may require more intensive monitoring and preventive strategies for atherosclerotic cardiovascular disease.⁽¹⁷²⁾

Table 6 - Comparing Our study with Titus maika et al., I/v/o risk of CVD in relation to the ASCVD score in the HIV-infected population

ASCVD SCORE	OUR STUDY	TITUS MSOKA ET AL STUDY
LOW	55.7%	52.9%
INTERMEDIATE	24.29%	17.3%
BORDERLINE	11.43%	
HIGH	8.57%	29.8%

In the present study the statistical analysis shows that the Framingham Scores between the two groups have a significant difference overall with P value- 0.0494. The HIV Infected group has a higher Framingham Score (11.38%) compared to the Control Population (9.18%). Prevalence of high CVD risk with framingham score in HIV patients could be due to chronic inflammatory condition of HIV, dyslipidemia being more in HIV group, smoking and alcohol habits and other comorbid conditions.

The research by Kakinami et al., in 2013, conducted in New York, examined the 10-year risk of cardiovascular disease among individuals with HIV (588 participants) using the Framingham score. The findings indicated an increase of 0.88% in risk compared to the non-HIV-infected group (1,764 participants). However, the difference was not deemed statistically significant, with a p-value of 0.24, which

could be attributed to the disparity in the number of participants between the two groups.⁽¹⁷³⁾

The study by B M Bergersen and team in 2000, based in Norway, revealed that when estimating the 10-year risk for cardiovascular disease using the Framingham score, patients undergoing HAART (219 individuals) had a greater than 20% risk at a rate of 11.9%, in contrast to 5.3% in a control group of 438 individuals. The statistical significance of this difference was marked by a p-value of 0.004, indicating a notable disparity, potentially influenced by the smaller size of the sample groups.⁽¹⁷⁴⁾

In a study conducted by Muiru et al., 2018 in Uganda comparing cardiovascular disease risk between HIV infected individuals (N=54) and uninfected individuals (N=50) showed significantly higher cardiovascular disease risk score of 5.3% in HIV uninfected versus 3.6% in HIV infected individuals ($p < 0.001$) using Framingham score. due to small sample size, However our findings differ from this study.⁽¹⁷⁵⁾

Our study demonstrates notable differences in ASCVD and Framingham scores among HIV Infected individuals and the Control Population. The means of ASCVD and Framingham scores vary significantly across the two populations, as indicated by the P value- 0.004 (< 0.05). It also indicates a strong relationship between ASCVD and Framingham scores, suggesting that Framingham scores can predict a substantial portion of the variability in ASCVD scores across the populations studied. HIV Infected individuals have the highest ASCVD and Framingham scores (6.67% and 11.38%) compared to Control Population (2.11% and 9.18%). Primary causes of variation in agreement between cardiovascular disease (CVD) risk prediction

algorithms are due to distinct cut-off points for risk categorization, variability in predictive variables influenced by diverse CVD risk factors across various populations or ethnic groups, different sets of outcomes used by each CVD risk prediction algorithm, and the discrepancy between the populations from which these algorithms were derived and those to which they are applied.

In a study by Titus Msoka and others in 2021 within Tanzania found that the cardiovascular disease risk scores were notably greater in individuals infected with HIV compared to those without the infection. The study reported a 6.1% risk for atherosclerotic cardiovascular disease (ASCVD) and a 9.0% risk according to the Framingham Risk Score (FRS) in the HIV-positive group, versus a 4.6% and 5.6% risk, respectively, in the HIV-negative group.⁽¹⁷⁶⁾

Table 7 - ASCVD score vs Framingham score in HIV vs control in our study compared with Titus Msoka et al., ASCVD vs Framingham Score

	Titus Msoka et al.	Titus Msoka et al.	OUR STUDY	OUR STUDY
	ASCVD	Framingham	ASCVD	FRAMINGHAM
HIV INFECTED NAIVE	4.60%	9.60%	7.19%	11.48%
HIV INFECTED ON ART	5.90%	8.60%	6.79%	11.38%
Control Population	4.60%	9.60%	2.11%	9.18%

In a study conducted by Mosepele et al. 2017 in sub-Saharan Africa showed that ASCVD classified more patients having elevated cardiovascular disease risk than Framingham score compared among diverse general populations beyond the US population.⁽¹⁷⁷⁻¹⁷⁹⁾ The difference in result was due to racial and geographical distribution of the population which affects the above scoring system.

We also compared ASCVD and Framingham scores depending upon duration of HIV infection and it was seen that a significant difference between ASCVD and Framingham Scores among HIV-infected individuals, upon the duration of HIV infection. In the HIV naive group 16 individuals were there and ASCVD and Framingham scores were 7.19% and 11.48% respectively, Naive individuals have high ASCVD and Framingham score due to high viral load. In HIV positive individuals with duration of 1-5 years 27 individuals were there with ASCVD vs Framingham score being 8.95% and 10.09% respectively, after introduction of ART regimen ASCVD score increased and Framingham score was seen to decrease. In HIV positive individuals with a duration of 5-10 years, 27 individuals were there with ASCVD vs Framingham score being 7.63% and 12.49% respectively, could be due to ongoing inflammation.

It was evident that the score difference between ASCVD and Framingham was significantly higher in individuals with HIV infection lasting more than 5 years. HIV is a disease of ongoing inflammatory changes so duration of HIV is directly proportional to the inflammatory changes taking place in the endothelium of the entire body. Ethnicity and race are important parameters which underestimate ASCVD score.

In a study conducted by Olamide O. Todowedo et al., 2019 showed that there is a linear association of older age and people who are taking ART regimen for longer duration and are in high risk score band for CVD mortality.⁽¹⁸⁰⁾

Several Studies have shown that cardiovascular disease risk is associated with duration and regimen of ART than HIV infection.⁽¹⁸¹⁻¹⁸⁴⁾

A study conducted by Islam et al., proved a yearly increasing CVD risk among HIV infected treated patients which establishes that CVD outcomes are strongly associated with duration of HIV infection and ART.⁽¹⁸⁵⁾

A study conducted by Viskovic et al., showed positive correlation among HIV duration and ART and CVD with a median of 8 and 6 years respectively.⁽¹⁸⁶⁾

When we compared ASCVD and Framingham scores among different treatment groups for HIV-infected individuals it revealed that the score difference between ASCVD and Framingham is significantly higher in individuals receiving TLD treatment. The P-value of 0.0454 indicates a statistically significant difference between ASCVD and Framingham scores ($P < 0.05$) in the context of treatment types for the HIV-infected group. Number of HIV naive patients were 13 and ASCVD and Framingham score in this group were 10.13% and 8.25% respectively. Number of HIV patients on TLD regimen were 52 and ASCVD and Framingham score in this group were 8.1% and 12.11%. HIV infected patients on regimen other than TLD regimen (NNRTI and PI) include 5 individuals and ASCVD and Framingham score in them were 2.95% and 11.44% respectively.

The result could be biased because patients would be on different regimens in the past and currently shifted to TLD regimen. In the government set up most of the naive HIV individuals are being started with TLD regimen and even chronic HIV patients are being shifted to TLD regimen because of the least side effect and better efficacy of the drug towards HIV And less drug to drug interactions.⁽¹⁸⁰⁻¹⁸⁶⁾

In our study there are no statistically significant differences in the mean ages of the HIV-infected individuals and the Control Population. This suggests that the age distribution is fairly consistent across two groups. Thus, any observed differences in mean ages are likely due to random variation rather than true differences between the groups.

In a study conducted by Minyahi Woldu et al., 2021 showed that Age leads to increased ASCVD risk. Participants with 40-64 years of age had low risk for ASCVD compared to 65-79 years⁽¹⁸⁷⁾

On the other hand, many other studies have also reported high prevalence of elevated ASCVD risk at 40 to 65 years of age that tended to decline with age above 65 years.⁽¹⁸⁸⁻¹⁸⁹⁾

Our study revealed significant differences in gender distribution among the two populations, with a highly significant P-value of 0.0003. Specifically, there is a notable disparity in the proportions of males in HIV and Controls (67.9% and 69.44%) and females in HIV and controls (32.1% and 30.56%) across these groups. Across two populations, the male proportion is significantly higher than the female proportion. Male are associated with habits like smoking and alcohol which lead to impaired lipid profile leading to increase in ASCVD and Framingham scores.

In a study conducted by Minyahi Woldu et al., 2021 showed there is difference in lifetime risk for cardiovascular disease based on sex based difference, being 50% for men and 33.3% for women at 40 years of age.(187). Females are considered to have protective role for ASCVD because of the sex hormones.⁽¹⁹⁰⁾

In our study the analysis demonstrates significant differences in Total Cholesterol Levels among the two populations , with a P-value of 0.0179. Specifically, the HIV-infected group exhibits higher cholesterol levels (164 mg/dl) compared to Control Population (141 mg/dl).

There was also a significant overall difference in HDL cholesterol levels among the two populations, with a P value of 0.0261. Specifically, the HDL cholesterol levels in the Control Population (39.11mg/dl) are significantly higher than those in the HIV Infected group (30.54%).

Our study compared LDL cholesterol levels among the two populations, with a P value of 0.0226. The LDL cholesterol level in the Control Population (95.11 mg/dl) is significantly higher than the HIV Infected group (75.52 mg/dl).

There were no significant differences in the triglyceride level among the HIV Infected individuals and Control Population with P value of 0.2843.

Deranged lipid profile in HIV infected people is due to ongoing inflammation and ART regimen. HIV is a state of continuous inflammation which leads to reduced HDL level leading to decrease in anti-inflammatory and antioxidant properties of HDL causing atherosclerotic state. ART specially protease inhibitors cause metabolic abnormalities like dyslipidemia, hyperinsulinemia and redistribution of adipose tissue.⁽¹⁸³⁾ Lopinavir/ritonavir (LPV/r) and fosamprenavir/ritonavir (FPV/r) are associated

with increased plasma lipid levels.⁽¹⁹¹⁾ Nucleoside/Nucleotide reverse transcriptase inhibitors (NRTIs) like stavudine, abacavir and zidovudine are also associated with dyslipidemia, while tenofovir disoproxil fumarate (TDF) or tenofovir alafenamide fumarate (TAF) favors lipogenesis.⁽¹⁹²⁾

Prevalence of dyslipidemia in our study was 100% compared to 76% in a study conducted by Dar es salaam et al., tanzania. The difference in prevalence could be because in Tanzania only HAART naive patients were taken while in our study both HAART naive and HAART experienced patients were taken.⁽¹⁹³⁾

In a study conducted in Latin America among HIV individuals showed prevalence of dyslipidemia as 80%.⁽¹⁹⁴⁾

In our study the prevalence of decreased HDL-c in our study was 75.71% was lower than the South Africa HIV cohort where it was reported to be high.⁽¹⁹⁵⁾

Our study demonstrates notable disparities in the prevalence of hypertension among the two populations. HIV Infected individuals exhibit the lowest prevalence of hypertension at 27.14% while Control Population shows the highest prevalence at 44.44%. The statistical analysis confirms the significance of these differences, with a P value of 0.0313. This difference could be because of more male individuals and low female individuals in the control group so proper comparison could not happen.

When comparing systolic blood pressure among different population groups, it is evident that there are no statistically significant differences. Any observed differences in systolic blood pressure are likely due to random variation rather than true differences between the groups.

Our study shows that there are discernible differences in the usage of antihypertensive medication among the two populations with P value of 0.0107. Notably, the prevalence of antihypertensive medication usage appears to be highest among HIV Infected individuals compared to Control Population. This could have led to resolution of hypertension in most of the HIV infected individuals leading to decrease in prevalence of hypertension compared to control groups.

There is limited data on hypertension in HIV patients. According to few studies there is an increased prevalence of hypertension in patients who are receiving ART regimen.

A study done by Gazzaruso et al., found no association between hypertension and ART regimen but their study only took NRTI and PI into consideration not the duration of treatment exposure.⁽¹⁹⁶⁾

The prevalence of hypertension in HIV patients in our study was 27.14% compared to Study done by Mawulorm K. I. Denu et al., 2024 showed the prevalence of hypertension among HIV patients to be 17.4%.⁽¹⁹⁷⁾ Ethiopia and Tanzania reported lower prevalence of hypertension being 14% and 8% respectively.⁽¹⁹⁸⁻¹⁹⁹⁾

Mawulorm K. I. Denu et al., 2024 analyzed that several factors are associated with development of hypertension which included age of 50 year or more , history of comorbid medical conditions like diabetes and dyslipidemia , body mass index (BMI) falling in category of overweight and obese.⁽¹⁹⁷⁾

The statistical analysis reveals notable differences in the prevalence of diabetes across the two populations. While diabetes is present in a substantial portion of each group, the Control Population exhibits the highest prevalence of 27.8%

compared to HIV infected population 15.71%. The P-value of 0.0004 suggests a significant difference in the prevalence of diabetes among these populations. Incidence of diabetes in the Control population being more than HIV population could be because of the prevalence of male population with 50 or more in age which could be associated with different habits or additional comorbidities. In our study most of the HIV individuals were on TLD regimen making them less prone for diabetes. Study done by Manuthu et al., found diabetes to be more prevalent in HIV patients on HAART compared to HAART naive. NRTI and PI are the main culprits of diabetes among HIV patients. PI inhibits uptake of glucose by insulin-sensitive tissue like skeletal muscle and fat. ⁽²⁰⁰⁾

In the present study, comparison of smoking habits among the two populations highlights notable disparities. The statistical analysis indicates a significant difference in smoking prevalence among the two groups with a p-value of 0.0001. Which suggests a highly significant difference in smoking habits across the populations. HIV Infected individuals demonstrate high prevalence of smoking (20%) compared to Control Population (8.3%)

Study done by Boettiger D et al 2016 compared smoking status in Asian population on ART with that of Thailand population which showed that prevalence of smoking in India and Philippines was much lower than in other countries like Vietnam , Hong Kong, Malaysia, Japan, South Korea and Singapore ⁽²⁰¹⁾

Study done by Lapp P et al 2016 showed that current smoking prevalence was higher in men and when compared in HIV status individuals in India it showed that people living with HIV are more likely to smoke. ⁽²⁰²⁾

In our study analysis reveals significant differences in alcohol consumption rates among the two populations with a p-value of 0.0013. It highlights that HIV Infected individuals have high prevalence of alcohol consumption at 28.57% while Control Population has low prevalence at 11.11%. The statistically significant P value suggests that these differences are not due to random chance and reflect true disparities in alcohol consumption across these groups.

Study conducted by Samet et al., 2004 showed that 40-50% people living with HIV have a history of alcohol dependence or alcohol abuse.⁽²⁰³⁾

Study conducted by Blagopal et al., 2008 and Brenchley proved that acute hepatitis secondary to alcohol can increase the rate of microbial translocation and immune activation which result in end organ damage like Myocardial Infarction and death.⁽²⁰⁴⁾

SUMMARY

The present cross sectional study was conducted among the patient more than 18 years of age fulfilling the inclusion criteria admitted in KLE DR.Prabhakar Kore Hospital, Belagavi during the period of January 2023 to December 2023 were analysed. Patients were assessed for physical biochemical examination after obtaining informed consent, the blood of the patient were analysed for dyslipidemia which include fasting lipid profile. All patients' data were collected. Patients redesigned performa and entered in excel sheet. The entire study was carried out using SPSS software, version 22 with a p-value threshold of 0.05 being considered statistically significant.

Present study aimed to estimate risk of CVD in patients living with HIV by using ASCVD score and to compare ASCVD score with Framingham score in estimating the CVD risk.

- In the present study, a total of 106 patients (70 HIV positive and 36 Controls) fulfilled inclusion criteria with mean age of 48 years.
- Among the participants 67.14% males and 32.86% females in the HIV group while 69.44% males and 30.56% females in the Control group.
- The ASCVD score was higher in the HIV group (6.67%) compared to the control group (2.11%).
- When comparing the risk of CVD in relation to the ASCVD score in the HIV-infected population, it was evident that a significant proportion of this

population (55.7%) has a Low CVD risk. This is followed by those with intermediate risk (24.29%), borderline risk (11.43%), and high risk (8.57%).

- The HIV Infected group has a higher Framingham Score (11.38%) compared to the Control Population (9.18%).
- HIV Infected individuals have the high ASCVD and Framingham scores (6.67% and 11.38%) compared to Control Population (2.11% and 9.18%).
- On comparing ASCVD score with Framingham score depending upon duration of HIV, It was evident that the score difference between ASCVD and Framingham was significantly higher in individuals with HIV infection lasting more than 5 years.
- When we compared ASCVD and Framingham scores among different treatment groups for HIV-infected individuals it revealed that the score difference between ASCVD and Framingham is significantly higher in individuals receiving TLD treatment.
- Risk factors associated with development of CVD in our study include Dyslipidemia (Cholesterol,HDL,LDL), Hypertension, Diabetes, Duration of HIV, ART regimen, Smoking, Alcohol. Age and Triglycerides were not statistically significant in our study.

CONCLUSION

- Populations with HIV tend to have a higher cardiovascular disease (CVD) risk when assessed using the ASCVD score compared to those without HIV, with many falling into the low-risk category.
- The Framingham score is often more predictive of CVD risk as compared to ASCVD score.
- The risk of CVD in HIV-positive individuals is multifaceted, influenced by the duration of HIV, the specific antiretroviral therapy regimen, and other risk factors such as gender, cholesterol levels, coexisting conditions like diabetes and hypertension, and lifestyle choices including smoking and alcohol consumption.
- The investigation establishes that both the Framingham Risk Score (FRS) and the Atherosclerotic Cardiovascular Disease (ASCVD) risk scores serve as proficient predictors for coronary artery disease (CAD). The FRS exhibits enhanced sensitivity in the prediction of CAD, while the ASCVD score is superior in its capacity to discern between patients with CAD.

STRENGTH AND LIMITATIONS

Strength of the study -

- Limited study has been conducted in HIV patients to predict the cardiovascular risk using non-invasive methods like ASCVD score and its correlation with Framingham score.

Limitations of the Study: -

- Additionally, the absence of a standard measure, like inflammatory biomarkers including carotid intima-media thickness (cIMT), pulse wave velocity (PWV), or flow-mediated dilation (FMD), meant we couldn't correlate these with the predicted scores to assess the precision of the algorithms in forecasting cardiovascular disease (CVD) risk.

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ANNEXURES

ANNEXURE I : CONSENT FORM

KAHERs JNMC BELAGAVI

INFORMED CONSENT FORM

“TITLE OF THE PROJECT/STUDY” “Estimation Of Cardiovascular Disease Risk Prediction By Atherosclerotic Cardiovascular Disease (ASCVD) Score In Human Immunodeficiency Virus (HIV) Infected Patients Which Is Cross Sectional Study”

Name of Student/Principal Investigator: _____

Name of Guide/Co Investigators: _____

Objective:

- 1) To estimate the risk of cardiovascular disease in patients living with HIV by using atherosclerotic cardiovascular disease (ascvd) score.
- 2) To compare atherosclerotic cardiovascular disease score with framingham score in estimating the cardiovascular diseases risk.

Introduction:

1) people living with HIV are presenting with less of infective complications, but on long term basis these patients are developing non-communicable diseases including diabetes , chronic obstructive pulmonary disease , kidney disease , hypertension , cardiovascular disease and cancer because HIV being a low grade inflammatory disease while cardiovascular and cerebrovascular disease being most important non infectious causes of mortality in HIV infected patients .

Immune dysfunction and persistent inflammation features of HIV infection have a possible role in pathogenesis of cardiovascular risk in addition to traditional risk factors such as cigarette smoking, elevated blood pressure, total cholestrol.

Some studies related this increase cardiovascular risk to the adverse effect of antiretroviral therapy (art) due to dyslipidemia.

With the increase in successful antiretroviral (art) coverage, the favorable effects of art on life expectancy of HIV infected individuals bring on a new challenge: the potential for an increasing incidence of cardiovascular disease(cvd) even a decreased risk was found in treated HIV infected person compared to uninfected controls.

The framingham risk score was established in a predominantly white population. Studies have revealed that in non-white racial group the risk score tends to considerably underestimate cardiovascular risk.

Conversely atherosclerotic cardiovascular disease score (ascvd) incorporates race in the prediction of cardiovascular risk of an individual.

Explanation of procedure:

* an informed consent will be obtained from the patients

*in patient and out patient department HIV infected individual(on treatment and naïve) will be identified.

* detailed history and examination would be done . History including =age , sex, h/o smoking , h/o diabetes , h/o hypertension treatment , on treatment with drugs like statin or aspirin ?

Examination including = systolic and diastolic blood pressure)

Lipid profile of the patient would be send

Calculation of cardiovascular risk by ascvd score

Withdrawal from participation in the study: Participation in this study is voluntary.

You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: “Name of student/PI, mobile number, email ID” If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **“Estimation Of Cardiovascular Disease Risk Prediction By Atherosclerotic Cardiovascular Disease (ASCVD) Score In Human Immunodeficiency Virus (HIV) Infected Patients Which Is Cross Sectional Study In Kle Dr.Prabhakar Kore Charitable Hospital For 1year Of Duration.”**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II :

PERFORMA

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

OCCUPATION:

COMPLAINTS AT PRESENTATION:

Past history:

DIABETES

HYPERTENSION

DYSLIPIDEMIA

Family history:

HIV

CVA

Personal history:

SMOKING

ALCOHOL

Treatment history:

HYPERTENSIVE

STATIN

ASPIRIN

HIV HISTORY :-

- DURATION OF ILLNESS
- NAIVE
- TREATMENT
- DURATION OF TREATMENT

Framingham Risk Score for Hard Coronary Heart Disease ☆

Estimates 10-year risk of heart attack.

INSTRUCTIONS
 There are several distinct Framingham risk models. MDCalc uses the 'Hard' coronary Framingham outcomes model, which is intended for use in **non-diabetic** patients age 30-79 years with no prior history of coronary heart disease or intermittent claudication, as it is the most widely applicable to patients without previous cardiac events. See the official Framingham website for additional Framingham risk models.

When to Use ▼ Pearls/Pitfalls ▼

Age years

Sex

Smoker

Total cholesterol mg/dL ↗

HDL cholesterol mg/dL ↗

Systolic BP mm Hg

Blood pressure being treated with medicines

Result:
 Please fill out required fields.

ASCVD Risk Estimator :

Current Age * Age must be between 20-79	Sex * <table border="1"> <tr> <td>Male</td> <td>Female</td> </tr> </table>	Male	Female	Race * <table border="1"> <tr> <td>White</td> <td>African American</td> <td>Other</td> </tr> </table>	White	African American	Other	
Male	Female							
White	African American	Other						
Systolic Blood Pressure (mm Hg) * Value must be between 90-200	Diastolic Blood Pressure (mm Hg) * Value must be between 60-130							
Total Cholesterol (mg/dL) * Value must be between 130 - 320	HDL Cholesterol (mg/dL) * Value must be between 20 - 100	LDL Cholesterol (mg/dL) * Value must be between 30-300						
History of Diabetes? * <table border="1"> <tr> <td>Yes</td> <td>No</td> </tr> </table>	Yes	No	Smoker? * <table border="1"> <tr> <td>Current</td> <td>Former</td> <td>Never</td> </tr> </table>		Current	Former	Never	
Yes	No							
Current	Former	Never						
On Hypertension Treatment? * <table border="1"> <tr> <td>Yes</td> <td>No</td> </tr> </table>	Yes	No	On a Statin? * <table border="1"> <tr> <td>Yes</td> <td>No</td> </tr> </table>	Yes	No	On Aspirin Therapy? * <table border="1"> <tr> <td>Yes</td> <td>No</td> </tr> </table>	Yes	No
Yes	No							
Yes	No							
Yes	No							

Ascvd score

Framingham score

RISK OF CVD DISEASES :

ON GENERAL EXAMINATION:

SYSTOLIC BLOOD PRESSURE

DIASTOLIC BLOOD PRESSURE

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

C.N.S.:

P.A.:

INVESTIGATIONS

INVESTIGATIONS	
HB	
PCV	
RBC	
PLATELET	
TOTAL COUNT	
UREA	
CREATININE	
TOTAL BILIRUBIN	
DIRECT BILIRUBIN	
INDIRECT BILIRUBIN	
SGOT	
SGPT	
ALP	
T.PROTEIN	
SODIUM	
POTASSIUM	
BICARBONATE	
CHLORIDE	
FBS	

LIPID PROFILE

T.CHOLESTEROL	
TRIGLYCERIDES	
HDL	
LDL	

ANNEXURE : III**MASTER CHART KEY**

F/H/O CVA	-	Family history of Cerebrovascular accident
CVD	-	Cardiovascular disease
SBP	-	Systolic blood pressure
DBP	-	Diastolic blood pressure
Hb	-	Hemoglobin
FBS	-	Fasting blood sugar
ALP	-	Alkaline phosphate
SGOT	-	Aspartate aminotransferase
SGPT	-	Alanine aminotransferase

ANNEXURE III : MASTER CHART

Sr No	Case No	Age	Race	Sex	IP No.	Address(Urban/Rural)	Occupation	Diabetes	Hypertension	Dyslipidemia	F/H/O HIV	F/H/O CVA	Smoking(Current/Former /Never)	Alcohol	Antihypertensive	Statins	Aspirin	HIV Duration	HIV naïve	HIV treatment	HIV duration of treatment	Total Cholesterol	HDL cholesterol	LDL cholesterol	Triglyceride	SBP	DBP	Framingham score	Risk of CVD	ASCVD score	Risk of CVD	Respiratory System	Cardiovascular System	Central Nervous System	Per Abdomen	HB (g/dl)	PCV (%)	RBC (10 ⁶ /ul)	Platelet (10 ⁶ /ul)	Total Count (10 ³ /ul)	Urea (mg/dl)	Creatinine (mg/dl)	Total Bilirubin (mg/dl)	Direct Bilirubin (mg/dl)	Indirect Bilirubin (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALT (U/L)	Total Protein (gm/dl)	Sodium (mEq/L)	Potassium (mEq/L)	Bicarbonate (m mol/L)	Chloride (mEq/L)	FBS(mg/dl)	
1	1	58 Years	Other	Male	6527714	Rukmini Nagar	Worker	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	10 Years	No	T. TLD	7 Years	128 mg/dl	22mg/dl	87mg/dl	94 mg/dl	130mmHg	90mmHg	27.30%	High	16.38%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36.9	3.97	1.99	9.13	35	0.9	0.9	0.2	0.7	43	24	172	8.7	141	3.7	24	102	100	
2	2	40 Years	Other	Male	1112000	Kittur	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	9 Years	No	T. TLD	9 Years	174 mg/fl	38mg/dl	91mg/dl	227 mg/dl	130mmHg	80mmHg	4.70%	Low	1.30%	low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	42.3	4.93	2.86	11.9	56	0.79	0.4	0.16	0.24	18	19	88	7.3	139	3.82	26	101	126	
3	3	51 Years	Other	Male	1112306	Belgaum	Teacher	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T.Telma Am	Absent	Absent	27 Years	No	T.ZLE	20 Years	165 mg/dl	27mg/dl	85mg/dl	264 mg/dl	136mmHg	84mmHg	18.21%	Medium	7.10%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36	2.86	2.82	9	26	0.85	0.48	0.21	0.27	21	20	92	7.1	136	4.01	29	102	99
4	4	40 Years	Other	Male	1112105	kiniye	Labour	Absent	Newly Diagnosed	Absent	Absent	Absent	Absent	Absent	Absent	On T Clidivin 5mg	Absent	Absent	2.5 Years	No	T. TLD	2.2 Years	181 mg/dl	99mg/dl	68mg/dl	70 mg/dl	140mmHg	80mmHg	3%	Low	0.5 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	42	3.73	0.97	9.5	27	1.17	0.37	0.16	0.21	16	44	159	5.9	134	3.54	18	103	80
5	5	56 Years	Other	Male	111281	Ramdev gali	Worker	Absent	Absent	Absent	Absent	Absent	Present (15 Years)	Present (15 Years)	Absent	Absent	Absent	Absent	4 Years	No	Absent	Absent	105 mg/dl	17mg/dl	65mg/dl	114 mg/dl	126mmHg	70mmHg	25.77%	High	13%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.3	44	3.61	2.29	8.7	16	0.65	0.49	0.22	0.27	30	10	100	7.5	129	3.84	26	97	79
6	6	43 Years	Other	Female	1112882	Bauxite Road	Cook	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	Absent	Absent	195 mg/dl	39mg/dl	111mg/dl	223 mg/dl	110mmHg	70mmHg	2.58 %	Low	0.9 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	40	3.38	3.59	6.6	18	0.66	0.48	0.18	0.3	45	71	294	7.4	144	4.19	24	107	141	
7	7	54 Years	Other	Male	1112888	Kakati	Worker	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T Telma 40mg	Absent	Absent	4 Years	No	T. TLD	4 Years	189 mg/dl	33mg/dl	124mg/dl	158 mg/dl	120mmHg	80mmHg	16.48%	Medium	7.20%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	33.2	4.04	3.1	12.6	15	0.9	1.6	0.5	1.1	23	11	164	6.3	134	3.5	30	101	90
8	8	40 Years	Other	Male	1114585	Vantmuri	Student	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Yes	Absent	Absent	101 mg/dl	16mg/dl	62mg/dl	117 mg/dl	100mmHg	60mmHg	3.52%	Low	1%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	35	4.9	2.52	4.6	21	0.77	0.54	0.13	0.41	576	19	217	9.6	136	4.53	21	99	90	
9	9	40 Years	Other	Male	1117856	Sankeshw ar	Farmer	Absent	Absent	Absent	Absent	Absent	Present (10 Years)	Present (10 Years)	Absent	Absent	Absent	Absent	4 years	No	T. TLD	3 Years	158 mg/dl	26mg/dl	108mg/dl	120 mg/dl	100mmHg	60mmHg	6.99%	Low	3.9 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	39.4	4.34	2.44	5.45	25	1.1	0.3	0.1	0.2	24	13	84	8.1	139	4.7	27	104	70
10	10	53 Years	Other	Male	1117474	Belgaum	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Present (7 Years)	Absent	Absent	Absent	Absent	Yes	Absent	Absent	100 mg/dl	19mg/dl	27mg/dl	86 mg/dl	120mmHg	80mmHg	9.66 %	Low	4.40%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.3	30.3	3.35	6.02	3.56	14	0.9	0.8	0.3	0.5	45	16	76	6.7	137	3.9	26	102	89	
11	11	54 Years	Other	Male	1118176	Kittur	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Present (8 Years)	Absent	Absent	Absent	Absent	No	T. TLD	2 Years	269 mg/dl	10mg/dl	205mg/dl	271 mg/dl	110mmHg	80mmHg	39.53 %	High	22.8 %	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36	4.18	1.97	23.9	115	5.1	5.2	3.1	2.1	367	41	371	7.5	130	6.2	21	110	80	
12	12	58 Years	Other	Male	1120100	Sadashiv Nagar	Worker	Absent	6 Months	Absent	Absent	Absent	Ocassionally	Present (15 Years)	Absent	On T Amllokint 5mg	Absent	Absent	18 Years	No	T. TLD	10 Years	108 mg/dl	20mg/dl	61mg/dl	135 mg/dl	130mmHg	70mmHg	34.98 %	High	13.8 %	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	43	4.15	2.52	5.1	19	1.2	1	0.2	0.8	41	37	243	7.6	136	3.4	18	103	110
13	13	42 Years	Other	Male	1128438	Shree Nagar	Teacher	Absent	4 Months	Absent	Absent	Absent	Absent	Absent	Present	On T Telma 40mg	Absent	Absent	14 Years	No	T. TLD	14 Years	125 mg/dl	26mg/dl	82mg/dl	79 mg/dl	130mmHg	90mmHg	7.4 %	Low	1.7 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	39.3	4.42	0.39	7.71	24	1	2.2	0.5	1.7	55	51	104	8.8	133	4.2	19	104	101
14	14	56 Years	Other	Male	1117933	Belgaum	Cook	Absent	Newly Diagnosed	Absent	Absent	Absent	Present (20 Years)	Present (20 Years)	Absent	On Tab Amlony 2.5mg	Present	Present	5 Years	No	T.TLE	5 Years	175 mg/dl	43mg/dl	98mg/dl	128 mg/dl	160mmHg	100mmHg	38.94 %	High	18.1 %	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	15	45	5.32	225	7.7	15	1.21	0.81	0.26	0.55	39	23	89	8.4	136	3.03	22	100	90
15	15	41 Years	Other	Male	5758612	Rakaskop	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	T. TLD	4 Years	171 mg/dl	48mg/dl	102mg/dl	103 mg/dl	120mmHg	80mmHg	3.5 %	Low	0.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	34	4.1	2.7	6.4	13	0.8	0.21	0.1	0.11	21	16	77	8.2	137	4.08	22	107	87	
16	16	40 Years	Other	Female	1129329	Belgaum	Cook	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	3 Moths	No	Absent	Absent	100 mg/dl	34mg/dl	32mg/dl	42 mg/dl	100mmHg	60mmHg	0.83 %	Low	0.2 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	4.8	16.7	0.7	1.2	4.3	116	5.1	0.5	0.2	0.3	42	49	124	6.1	139	3.6	24	116	86
17	17	40 Years	Other	Male	1142590	Shivbasav Nagar	Professo r	Absent	Absent	Absent	Absent	Absent	Present (10 Years)	Present (10 Years)	Absent	Absent	Absent	Absent	1 Months	No	T. TLD	1 Months	121 mg/dl	25mg/dl	85mg/dl	56 mg/dl	150mmHg	90mmHg	11.48 %	Medium	1.5 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	15	46.4	4.63	2.79	5.1	14	0.92	0.96	0.39	0.57	1.6	243	89	8	128	3.89	18	93	159
18	18	48 Years	Other	Male	1142712	Darbar Gali	Worker	Absent	Absent	Absent	Absent	Absent	Present (25 Years)	Present (25 Years)	Absent	Absent	Absent	Absent	1 Years	No	T. TLD	7 Months	109 mg/dl	63mg/dl	31mg/dl	77 mg/dl	160mmHg	90mmHg	8.67 %	Low	3.10%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	40	3.98	2.16	8.7	10	0.56	0.9	0.18	0.72	37	15	92	7.1	131	4.3	24	96	127
19	19	43 Years	Other	Male	1142298	Sambra	Professo r	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	T. TLD	Absent	150 mg/dl	23mg/dl	91mg/dl	180 mg/dl	136mmHg	80mmHg	8.61 %	Low	3%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36	3.72	2.05	15.2	30	0.89	1.05	0.68	0.37	105	38	97	7	131	5.16	18	98	123	
20	20	69 Years	Other	Male	1142601	sambra	Worker	Absent	5 Years	Absent	Absent	Absent	Absent	Absent	Absent	On T. Telma 40mg	Absent	Absent	Newly Diagnos ed	Yes	T. TLD	Absent	120 mg/dl	24mg/dl	61mg/dl	175 mg/dl	128mmHg	70mmHg	29.57 %	High	20.80%	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	37.8	4.24	2.61	10	15	1.2	0.42	0.18	0.24	16	12	82	7.4	138	4.43	23	104	38
21	21	40 Years	Other	Female	1144099	Vantmuri	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	8 Years	No	T. TLD	8 Years	130 mg/dl	38mg/dl	67mg/dl	106 mg/dl	120mmHg	80mmHg	1.7 %	Low	0.4 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	36	4.32	2.71	9.9	43	0.85	0.2	0.1	0.1	28	36	169	7.3	137	3.75	28	104	90
22	22	57 Years	Other	Female	1144353	Sankeshw ar	Professo r	5 Years	2 Years	Absent	Absent	Absent	Absent	Absent	Absent	On T.Prolome t XL 25mg	Absent	Absent	2 Month	No	T. TLD	2 Months	146 mg/dl	10mg/dl	13mg/dl	62 mg/dl	110mmHg	70mmHg	21.93 %	High	13.8 %	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	37	4.5	1.02	9.4	76	1.73	1.46	0.29	1.17	16	6	78	7.6	132	4.74	17	96	137
23	23	42 Years	Other	Male	1144495	Vantamuri	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	8 Years	No	T.LZI	Absent	146 mg/dl	37mg/dl	75mg/dl	75 mg/dl	150mmHg	90mmHg	6.11 %	Low	1.6 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	35	4.57	2.41	6.9	17	0.94	0.61	0.19	0.42	30	27	101	8.5	139	4.08	27	102	74
24	24	40 Years	Other	Male	1144991	Hukkeri	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	8 Years	No	T.TLD	1 year	149 mg/dl	36mg/dl	100mg/dl	63 mg/dl	108mmHg	80mmHg	3.7 %	Low	0.7 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	40.5	4.67	1.26	3.93	15	0.9	0.2	0.1	0.1	36	43	95	8.5	133	4.1	26	102	101
25	25	55 Years	Other	Female	1146217	Belgaum	Teacher	Absent	5 Years	Absent	Absent	Absent	Absent	Absent	Absent	On Amlong 5mg	Absent	Absent	12 Years	No	T. TNZ	12 Years	233 mg/dl	28mg/dl	167mg/dl	188 mg/dl	130mmHg	70mmHg	14.37 %	Medium	6.10%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	40	3.19	1.5	5.3	26	0.83	0.29	0.1	0.19	29	23	65	7	131	4.3	22	100	108
26	26	50 Years	Other	Female	1146709	sambre	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	13 Years 10 Years	No	T. TLD	13 Years	149 mg/dl	45mg/dl	88mg/dl	79 mg/dl	110mmHg	70mmHg	2.39 %	Low	0.80%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	35.9	4.23	2.36	11.5	20	0.6	0.6	0.3	0.3	20	10	95	6.8	138	3.2	23	101	160
27	27	45 Years	Other	Female	1148254	Dharwad	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	T. TLD	10 Years	109 mg/dl	46mg/dl	53mg/dl	52 mg/dl	118mmHg																													

Sr No	Case No	Age	Race	Sex	IP No.	Address(Urban/Rural)	Occupation	Diabetes	Hypertension	Dyslipidemia	F/H/O HIV	F/H/O CVA	Smoking(Current/former /Never)	Alcohol	Antihypertensive	Statins	Aspirin	HIV Duration	HIV naïve	HIV treatment	HIV duration of treatment	Total Cholesterol	HDL cholesterol	LDL cholesterol	Triglyceride	SBP	DBP	Framingham score	Risk of CVD	ASCVD score	Risk of CVD	Respiratory System	Cardiovascular System	Central Nervous System	Per Abdomen	HB (g/dl)	PCV (%)	RBC (10 ⁶ /uL)	Platelet (10 ³ /uL)	Total Count (10 ³ /uL)	Urea (mg/dl)	Creatinine (mg/dl)	Total Bilirubin (mg/dl)	Direct Bilirubin (mg/dl)	Indirect Bilirubin (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALP (U/L)	Total Protein (gm/dl)	Sodium (mEq/L)	Potassium (mEq/L)	Bicarbonate (n mol/L)	Chloride (mEq/L)	FBS(mg/dl)
38	38	57 Years	Other	Male	1166143	Shree Nagar	Worker	Absent	2 Years	Absent	Absent	Absent	Absent	Absent	On T. Telma 40mg	Absent	Absent	5 Years	No	T.TLD	5 Years	100 mg/dl	16mg/dl	38mg/dl	88 mg/dl	110mmHg	70mmHg	15.77%	Medium	7.30%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	31.5	3.18	1	3.2	14	0.52	7.34	4.79	2.55	60	19	96	5.3	130	4.04	20	101	110
39	39	49 Years	Other	Male	1167835	Shree Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Present (35 Years)	Absent	Absent	Absent	Newly Diagnos ed	Yes	T.TLD	Absent	217 mg/dl	45mg/dl	145mg/dl	134 mg/dl	100mmHg	60mmHg	5.83%	Low	2.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	37.5	4.57	3	7.6	42	1.37	0.64	0.22	0.42	33	19	84	7.3	138	3.98	21	105	99
40	40	57 Years	Other	Female	1170228	Mantesh Nagar	Worker	4 Years	4 Years	Absent	Absent	Absent	Absent	Absent	On T.Prolome t 50mg	Absent	Absent	7 Years	No	T.TLD	7 Years	151 mg/dl	18mg/dl	54mg/dl	394 mg/dl	100mmHg	60mmHg	12.18%	High	7.40%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36.5	5	4	9	36	1	1	0.5	0.5	20	35	110	6	137	4.06	19	102	217
41	41	56 Years	Other	Female	1171870	Railway 3rd Gate	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	15 Years	No	T.TLD	15 Years	135 mg/dl	39mg/dl	81mg/dl	115 mg/dl	120mmHg	80mmHg	3.86%	Low	1.80%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	43.8	4.82	4	8.3	79	4.41	0.3	0.11	0.19	22	11	130	9.7	130	4.11	12	98	162
42	42	59 Years	Other	Male	1172643	Kiniye	Teacher	10 Years	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	5 Years	No	T.TLD	5 Years	100 mg/dl	22mg/dl	49mg/dl	55 mg/dl	100mmHg	70mmHg	14.23%	High	9.40%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	38	3.63	2	7.3	37	1.75	0.48	0.25	0.23	15	12	66	8.3	136	5.47	22	104	162
43	43	56 Years	Other	Female	1173019	Kiniye	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	T.TLD	Absent	134 mg/dl	29mg/dl	65mg/dl	198 mg/dl	120mmHg	80mmHg	4.70%	Low	2.30%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.9	36	4.85	5	9.8	26	1.08	0.23	0.11	0.12	25	26	87	8.2	126	4.17	17	96	107
44	44	40 Years	Other	Male	1181581	Kiniye	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	2 Years	No	T.TLD	2 Years	192 mg/dl	30mg/dl	123mg/dl	197 mg/dl	140mmHg	100mmHg	7.56%	Low	2.50%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	15	45	5.48	4	30.5	34	0.73	0.94	0.29	0.65	24	35	154	6.5	120	4.19	19	84	270
45	45	40 Years	Other	Male	1180246	Hanuman Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	1 Months	No	T.TLD	1 Months	161 mg/dl	12mg/dl	106mg/dl	217 mg/dl	110mmHg	70mmHg	9.07%	Low	4.40%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	32.9	3.53	1	9.7	28	0.75	1.36	1.04	0.32	90	41	281	6.9	128	3.88	15	91	60
46	46	40 Years	Other	Male	1180253	Bauxite Road	Worker	Absent	Absent	Absent	Absent	Absent	Absent	7 Years	Absent	Absent	Absent	2 Years	No	T. TLD	2 Years	125 mg/dl	26mg/dl	82mg/dl	79 mg/dl	110mmHg	70mmHg	3.42%	Low	0.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	31.2	3.5	2	2.95	20	0.6	0.5	0.2	0.3	60	61	69	6.2	139	3.5	16	101	90
47	47	46 Years	Other	Female	1180270	Kakati	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	5 Years	No	T.TLD	5 Years	113 mg/dl	40mg/dl	51mg/dl	106 mg/dl	110mmHg	60mmHg	1.54%	Low	0.40%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.2	40	3.02	2	4.7	26	1.44	0.3	0.12	0.18	12	10	137	85	136	3.94	16	106	128
48	48	40 Years	Other	Female	10007465	Kakati	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	Absent	Absent	220 mg/dl	83mg/dl	164mg/dl	112 mg/dl	110mmHg	70mmHg	1.48%	Low	0.20%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.6	42	4	2	3.8	16	0.68	0.2	0.1	0.1	26	46	106	8	130	3.82	16	102	130
49	49	52 Years	Other	Female	10007248	Killa Lake	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	8 Years	No	T.TLD	8 Years	100 mg/dl	11mg/dl	10mg/dl	108 mg/dl	110mmHg	70mmHg	4.35%	Low	2.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	32	3.08	1	4	16	0.92	1.56	0.66	0.9	72	32	98	8.8	131	4.11	12	108	101
50	50	50 Years	Other	Female	10006174	Shree Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	6 Months	No	T.TLD	6 Months	100 mg/dl	9mg/dl	8mg/dl	76 mg/dl	100mmHg	70mmHg	2.71%	Low	2.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	7.9	40	2.68	2	6.2	20	0.69	0.79	0.59	0.2	10	17	190	5.6	124	3.84	17	93	116
51	51	40 Years	Other	Male	10004146	Sahu Nagar	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	20 Years	No	T.TLD	15 Years	106 mg/dl	26mg/dl	50mg/dl	233 mg/dl	110mmHg	70mmHg	2.85%	Low	0.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	32	2.97	1	4.3	16	0.6	0.37	0.12	0.25	36	53	93	8.6	134	4.1	20	106	98
52	52	49 Years	Other	Male	10008864	Sahu Nagar	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	5 Years	No	T.TLD	5 Years	100 mg/dl	20mg/dl	12mg/dl	82 mg/dl	100mmHg	60mmHg	5.21%	Low	2.00%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	8.1	38	2.65	2	16.6	12	5.87	1.31	0.84	0.47	47	21	112	5.6	130	4.51	10	100	120
53	53	63 Years	Other	Male	10010072	Hanuman Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	5 Years	No	T.TLD	5 Years	100 mg/dl	15mg/dl	24mg/dl	116 mg/dl	130mmHg	80mmHg	22.19%	High	13.90%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	36	2.7	2.2	10	10	6.3	0.54	0.98	0.8	0.2	18	12	6	134	3.69	20	100	184
54	54	51 Years	Other	Male	10011354	Nehru Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	6 Months	No	T.TLD	6 Months	100 mg/dl	26mg/dl	12mg/dl	136 mg/dl	100mmHg	60mmHg	4.63%	Low	1.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	5.7	19.4	1.87	1	2.4	11	0.6	1.42	0.53	0.89	105	18	311	5.5	121	4.5	12	91	66
55	55	55 Years	Other	Male	10012041	Kakti	Worker	Absent	4 Years	Absent	Absent	Absent	Absent	12 Years	Present	Absent	Absent	Newly Diagnos ed	Yes	T. TLD	Absent	190 mg/dl	24mg/dl	121mg/dl	194 mg/dl	110mmHg	70mmHg	19.49%	Medium	9.00%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	38	5.79	1	4.2	24	0.98	0.42	0.19	0.23	74	61	105	7.5	139	3.2	23	99	81
56	56	40 Years	Other	Male	10012541	Kiniye	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	T. TLD	Absent	110 mg/dl	31mg/dl	58mg/dl	76 mg/dl	120mmHg	70mmHg	2.98%	Low	0.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	40	2.8	1.5	9	24	0.89	0.35	0.22	0.13	43	41	77	6.6	138	3.74	20	105	84
57	57	40 Years	Other	Male	10027492	Vantamuri	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	Absent	Absent	100 mg/dl	9mg/dl	30mg/dl	187 mg/dl	140mmHg	80mmHg	10.96%	Medium	3.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	16	38	5.86	3	11.3	48	0.9	1.97	1.41	0.56	125	51	107	6.8	131	4.35	28	86	65
58	58	45 Years	Other	Female	10027119	Vantamuri	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	5 Years	No	T.TLD	5 Years	205 mg/dl	42mg/dl	135mg/dl	153 mg/dl	100mmHg	70mmHg	2.20%	Low	0.80%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	4	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90
59	59	40 Years	Other	Male	10029180	Rakaskop	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Newly Diagnos ed	Yes	Absent	Absent	102 mg/dl	32mg/dl	42mg/dl	215 mg/dl	100mmHg	70mmHg	1.88%	Low	0.30%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	40	4.92	3	14.9	28	0.82	0.53	0.32	0.21	69	44	57	6	135	4.15	20	103	166
60	60	40 Years	Other	Male	10030076	Pangul Gali	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	8 Years	Absent	Absent	Absent	Newly Diagnos ed	Yes	Absent	Absent	119 mg/dl	38mg/dl	68mg/dl	106 mg/dl	126mmHg	80mmHg	2.96%	Low	0.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	42	3.89	1.16	5.2	17	0.6	3.73	2.23	1.5	76	35	110	8.2	132	3.67	19	101	109
61	61	48 Years	Other	Male	10030089	Darbar Gali	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	9 Years	No	Absent	Absent	127 mg/dl	26mg/dl	80mg/dl	137 mg/dl	130mmHg	80mmHg	8.19%	Low	3%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	45	4.42	1.68	8.4	31	1.13	0.69	0.3	0.39	25	16	59	9.1	128	3.81	22	9.6	119
62	62	43 Years	Other	Male	10031054	Khade Bazar	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	11 Years	No	T.LZI	Present	143 mg/dl	39mg/dl	85mg/dl	146 mg/dl	130mmHg	80mmHg	4.67%	Low	1.20%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	37.2	4.66	1.95	5.9	23	1.04	0.32	0.18	0.14	26	30	67	16.9	141	4.04	22	106	71
63	63	52 Years	Other	Male	10032771	Bapat Gali	Teacher	Absent	Present	Absent	Absent	Absent	Absent	Absent	On T. Telma 40mg	Absent	Absent	10 Years	No	T.TLD	Absent	100 mg/dl	24mg/dl	51mg/dl	153 mg/dl	100mmHg	60mmHg	7.07 %	Low	2.70%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	32.7	3.43	2.55	12.4	85	1.22	0.5	0.13	0.37	79	50	296	8.6	116	6.41	119	94	79
64	64	40 Years	Other	Female	10033728	Shree Nagar	Farmer	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	9 Years	No	T.TLD	Absent	152 mg/dl	49mg/dl	81mg/dl	179 mg/dl	120mmHg	80mmHg	1.75%	Low	0.40%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	38	3.94	2.32	8.68	12	0.8	0.5	0.2	0.3	20	40	52	6	135	3.6	19	98	100
65	65	52 Years	Other	Female	10031526	Shivbasav Nagar	Worker	Absent	Present	Absent																																												

Sr No	Case No	Age	Race	Sex	IP No.	Address(Urban/Rural)	Occupation	Diabetes	Hypertension	Dyslipidemia	F/H/O HIV	F/H/O CVA	Smoking(Current/former /Never)	Alcohol	Antihypertensive	Statins	Aspirin	HIV Duration	HIV naïve	HIV treatment	HIV duration of treatment	Total Cholesterol	HDL cholesterol	LDL cholesterol	Triglyceride	SBP	DBP	Framingham score	Risk of CVD	ASCVD score	Risk of CVD	Respiratory System	Cardiovascular System	Central Nervous System	Per Abdomen	HE (g/dl)	PCV (%)	RBC (10 ⁶ /uL)	Platelet (10 ³ /uL)	Total Count (10 ³ /uL)	Urea (mg/dl)	Creatinine (mg/dl)	Total Bilirubin (mg/dl)	Direct Bilirubin (mg/dl)	Indirect Bilirubin (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALP (U/L)	Total Protein (gm/dl)	Sodium (mEq/L)	Potassium (mEq/L)	Bicarbonate (n mol/L)	Chloride (mEq/L)	FBS(mg/dl)	
75	75	61 Years	Other	Male	10026982	sambre	Worker	Absent	Present	Absent	Absent	Absent	Absent	Absent	On T. Amlong 5mg	Absent	Absent	Absent	No	Not Known	Absent	165mg/dl	20mg/dl	130mg/dl	174mg/dl	150mmHg	80mmHg	42.85%	High	22.0 %	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	18	40	5.24	2	6.1	19	0.86	1.13	0.61	0.51	45	49	76	7.2	138	4.1	22	102	117	
76	76	40 Years	Other	Male	7286228	Sahu Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	100 mg/dl	19mg/dl	42mg/dl	74 mg/dl	110mmHg	70mmHg	3.56%	Low	0.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	4	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90	
77	77	40 Years	Other	Male	10027323	Sankeshwar	Farmer	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	154 mg/dl	74mg/dl	81mg/dl	70 mg/dl	120mmHg	80mmHg	2.64%	High	0.30%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	8.4	31.5	5.07	7	30.4	25	0.55	0.8	0.4	0.4	35	30	105	7	134	4.58	18	98	220	
78	78	55 Years	Other	Female	2805838	Hukkeri	Worker	Present	Present	Present	Absent	Absent	Absent	Absent	Absent	On T.Telma 40mg	Absent	Absent	Absent	No	Not Known	Absent	198 mg/dl	48mg/dl	127mg/dl	159mg/dl	130mmhg	80mmhg	8.33%	Low	3.10%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	4	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90
79	79	54 Years	Other	Male	10025358	Laxmi Nagar	Worker	Absent	Present	Absent	Absent	Absent	Present	Present	On T.Telma Am	Absent	Absent	Absent	No	Not Known	Absent	108 mg/dl	42mg/dl	58mg/dl	96 mg/dl	110mmHg	70mmHg	11.66%	Medium	5.00%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	36	3.37	1.15	11.4	46	0.97	0.8	0.4	0.4	29	37	108	6.6	135	3.9	19	96	90	
80	80	46 Years	Other	Male	10027115	Hubli	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	No	Not Known	Absent	230 mg/dl	40mg/dl	187mg/dl	134 mg/dl	110mmHg	70mmHg	12.75%	Medium	7.30%	Boderline	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	16	37	5.78	4	15.2	14	0.9	0.92	0.46	0.46	22	10	62	6.5	135	3.67	19	94	136
81	81	46 Years	Other	Male	10027317	Hubli	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	No	Not Known	Absent	105 mg/dl	26mg/dl	53mg/dl	190 mg/dl	110mmHg	70mmHg	4.29%	Low	1.30%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	14	34	4.64	3	7.9	17	0.8	0.6	0.33	0.27	97	30	96	5.4	135	3.8	14	91	94	
82	82	68 Years	Other	Male	10026891	Hukkeri	Farmer	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T. Telma 40mg	Absent	Absent	Absent	No	Not Known	Absent	110 mg/dl	37mg/dl	55mg/dl	102 mg/dl	170mmHg	90mmHg	30.09%	High	23.80%	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	8.6	26.7	2.8	2	18.2	107	5.12	1.13	0.61	0.51	45	49	76	7.2	134	4.58	14	100	106
83	83	60 Years	Other	Female	10027021	Railway 3rd Gate	Worker	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T.Telma Am	Absent	Absent	Absent	No	Not Known	Absent	192 mg/dl	45mg/dl	157mg/dl	47 mg/dl	100mmHg	70mmHg	4.99 %	Low	3.6 %	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	29	4.07	3	9.5	30	0.98	0.92	0.46	0.46	22	10	62	6.5	137	3.53	17	104	108
84	84	64 Years	Other	Male	10026749	Ramdev gali	Farmer	Present	Present	Absent	Absent	Absent	Absent	Present	Absent	On T. Telma 40mg	Absent	Absent	Absent	No	Not Known	Absent	197 mg/dl	48mg/dl	123mg/dl	165 mg/dl	120mmHg	70mmHg	32.70%	High	22.70%	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	17	50.4	5.16	3	12.3	18	0.79	0.92	0.29	0.63	29	23	117	7.6	137	4.9	21	98	227
85	85	60 Years	Other	Male	10026846	Rakaskop	Worker	Present	Present	Absent	Absent	Absent	Absent	Absent	Absent	On. T.starmlo	Absent	Absent	Absent	No	Not Known	Absent	225 mg/dl	41mg/dl	132mg/dl	132 mg/dl	110mmHg	70mmHg	30.82 %	High	18.20%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	41.9	5.38	3	13.5	17	0.79	0.92	0.29	0.63	29	23	117	7.6	132	4.74	20	95	172
86	86	55 Years	Other	Female	2805838	Bapat Gali	Worker	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On.T.Nicar dia R	Absent	Absent	Absent	No	Not Known	Absent	198 mg/dl	48mg/dl	127mg/dl	159 mg/dl	130mmHg	80mmHg	8.33%	Low	3.10%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	4	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90
87	87	60 Years	Other	Female	7293776	Bauxite Road	Worker	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	133 mg/dl	13mg/dl	73mg/dl	188 mg/dl	130mmHg	70mmHg	21.83%	High	12.2 %	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	45	3.55	2.04	11	18	0.82	1.25	0.1	1.15	37	23	105	5.6	123	4	19	90	108	
88	88	65 Years	Other	Male	10029191	Belgaum	Worker	Present	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T.Telma 40mg	Absent	Absent	Absent	No	Not Known	Absent	112 mg/dl	35mg/dl	66mg/dl	76 mg/dl	120mmHg	80mmHg	25.66 %	High	20%	High	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	49	3.55	2	9.6	21	0.7	0.48	0.22	0.26	127	40	97	5.7	128	3.39	19	96	120
89	89	67 Years	Other	Male	10029168	Shivbasav Nagar	Farmer	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	112 mg/dl	35mg/dl	66mg/dl	66 mg/dl	120mmHg	80mmHg	22.69%	High	12.5 %	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	39.9	4.73	3	20.4	45	1.9	2.02	0.96	1.06	22	18	75	6.3	134	1.78	20	97	171	
90	90	48 Years	Other	Male	10029800	Shree Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	183 mg/dl	39mg/dl	123mg/dl	117 mg/dl	110mmHg	70mmHg	6.19%	Low	2.50%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	15	39	5.53	3	9.7	22	0.86	2	1	1	20	18	85	6.5	138	4.1	23	102	110	
91	91	70 Years	Other	Male	10029791	Mantesh Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	129 mg/dl	32mg/dl	81mg/dl	120 mg/dl	110mmHg	80mmHg	27.17%	High	16.80%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	13	40	4.43	2	10.4	33	0.81	1.39	0.52	0.87	24	18	92	7.4	139	4.42	20	106	174	
92	92	70 Years	Other	Female	10030416	Shree Nagar	Teacher	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	106 mg/dl	43mg/dl	49mg/dl	127 mg/dl	140mmHg	70mmHg	13.15%	High	18%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	5.4	16	1.89	5	9	77	1.91	0.88	0.47	0.41	29	37	89	7.7	137	5.15	22	100	22	
93	93	40 Years	Other	Male	7305755	Nehru Nagar	Worker	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	238 mg/dl	43mg/dl	183mg/dl	114 mg/dl	130mmHg	70mmHg	10.42%	High	3.90%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	12	35	2.5	1.8	8.5	22	24.5	0.84	0.42	0.42	25	40	80	6.3	139	4.66	24	100	144	
94	94	40 Years	Other	Male	7305746	Sadashiv Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	222 mg/dl	41mg/dl	156mg/dl	192 mg/dl	120mmHg	80mmHg	5%	Low	1.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	29	5.5	2.5	7.4	35	27.7	0.78	0.35	0.43	20	35	86	6	136	5.06	17	101	97	
95	95	70 Years	Other	Female	10031522	kinije	Teacher	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T.Telma40 mg	Absent	Absent	Absent	No	Not Known	Absent	198 mg/dl	41mg/dl	137mg/dl	103 mg/dl	140mmHg	90mmHg	18.96%	Medium	15.70%	Intermediate	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	3	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90
96	96	62 Years	Other	Female	10026340	Kittur	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	119mg/dl	63mg/dl	46mg/dl	89mg/dl	130mmHg	80mmHg	2.70%	Low	3.73%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	3	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90	
97	97	45 Years	Other	Female	10031951	Mantesh Nagar	Teacher	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	214 mg/dl	41mg/dl	147mg/dl	151 mg/dl	130mmHg	80mmHg	4.85%	Low	1.50%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	11	32	4.11	3.23	7.9	32	0.71	0.24	0.13	0.11	18	25	87	6.6	141	3.89	22	108	101	
98	98	40 Years	Other	Male	10031547	Bapat Gali	Worker	Present	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T.Telma 40mg	Absent	Present	Absent	No	Not Known	Absent	164 mg/dl	46mg/dl	109mg/dl	58 mg/dl	200mmHg	100mmHg	19.87%	High	4.20%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	17	49.6	5.43	3.46	12.2	33	1.22	0.74	0.34	0.4	29	42	176	7.7	134	4.46	19	99	242
99	99	50 Years	Other	Male	10031033	Khade Bazar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not Known	Absent	144 mg/dl	44 mg/dl	85mg/dl	78 mg/dl	130mmhg	90mmhg	6.60%	Low	2.60%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	10	35.9	4.64	3.85	13	65	0.82	0.35	0.06	0.24	26	10	103	7.6	128	4.96	17	95	97	
100	100	60 Years	Other	Female	10031659	Rukmini Nagar	Worker	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	On T. Telma 40mg	Absent	Absent	Absent	No	Not Known	Absent	100mg/dl	22mg/dl	51mg/dl	53mg/dl	140mmHg	90mmHg	9.51%	Low	5.70%	Low	B/L Air Entry	S1S2 Present	Consious Oriented	Soft NT	9.7	30.7	2.52	2	10.6	17	0.64	2.88	0.59	2.29	32	12	84	6.6	136	3.77	19	98	90
101	101	59 years	Others	Male	10032066	Nehru Nagar	Worker	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	No	Not known	Absent	192mg/dl	43mg/dl	127mg/dl	170mg/dl	130mmhg	70mmhg	14.79%	Medium	8.80%	Intermediate	B/L Air Entry	S1S2 present	Conscious Oriented	Soft NT	15	42.7	4.73	2.81	9.1	15	0.74	0.53	0.28	0.25	23	27	93	7.8						