# DIABETES MELLITUS AND ASSOCIATED FACTORS IN HUMAN IMMUNODEFICIENCY VIRUS INFECTED INDIVIDUALS AT JIMMA UNIVERSITY SPECIALIZED HOSPITAL, SOUTHWEST ETHIOPIA


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A RESEARCH PAPER SUBMITTED TO THE DEPARTMENT OF MEDICAL LABORATORY SCIENCE AND PATHOLOGY, COLLEGE OF PUBLIC HEALTH AND MEDICAL SCIENCES, JIMMA UNIVERSITY, IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR MASTER OF SCIENCE DEGREE IN CLINICAL LABORATORY SCIENCE, SPECIALTY IN CLINICAL CHEMISTRY.

SEPTEMBER, 2014

JIMMA, ETHIOPIA

JIMMA UNIVERSITY
COLLEGE OF PUBLIC HEALTH AND MEDICAL SCIENCES
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## *ABSTRACT*

***Background**: Over 25 million people in sub-Saharan Africa are infected with HIV, representing nearly 70% of the world's total population of people living with HIV and AIDS (PLWHA). In 2012, 7.5 million Africans were on HAART. However, adults are living longer on HAART; there is increasing concern about rising incidence of insulin resistance, glucose intolerance, type 2 diabetes, and dyslipidemia among PLWHA.*

***Objectives**: To assess the magnitude of diabetes mellitus and associated factors in HIV/AIDS positive individuals.*

***Methods**: An institution based cross sectional study design was conducted from April to May, 2014 at JUSH, comprehensive chronic care and training center in a total of 393 HIV infected individuals. Convenient sampling technique was implemented and the samples were taken consecutively. Socio-demographic and anthropometric data was collected by structured questionnaire. Laboratory analysis of serum glucose, total cholesterol, triglycerides, HDL, LDL and HCV was done according to the manufacturer's instruction. The data was analyzed by SPSS version 20 and descriptive and inferential stastics was applied.*

***Results**: A total of 393 HIV infected individuals of age ranging from 21 to 75 years with mean age of* 37.9 ±11.18 *(mean ±SD) had participated in this study. The total prevalence of diabetes mellitus (DM) in this study was 6.4 % (n=25). Two hundred ninety one (74%), and 77(19.6%) of the study participants had normal glucose value (70-110 mg/dl) and impaired fasting glucose value (111-125 mg/dl) respectively. After adjusting of the other variables, age (AOR=2.98, 95%CI: 1.04-8.51, P=0.042), duration of HAART (AOR=19.48, 95%CI: 2.59-146.44, P=0.004), hypertension (AOR=5.49, 95%CI: 1.88-16.08, P=0.002) and dyslipidemia (AOR=6.07, 95%CI: 2.07-17.83, P=0.001) had strong significance association with diabetes.*

***Conclusion and recommendations:** We conclude that, diabetes was highly prevalent among adult HIV/AIDS patients, at comprehensive chronic care and training center of JUSH. We recommend that all newly diagnosed HIV/AIDS patients should be routinely screened for diabetes, both before and after initiating HIV treatment. All the adult HIV/AIDS patients should have routine checkup for hypertension and lipid profile tests must be routinely done for screening of dyslipidemia*

## ACKNOWLEDGEMENT

I would like to acknowledge my advisors Mr.Waqtola Cheneke and Dr.Tilahun Yemane for their valuable guidance , critical comments and suggestions in the preparation of this paper, Jimma University, department of Medical Laboratory Science and Pathology for starting of Clinical laboratory science (CLS) masters program, the study participants, the staff of comprehensive chronic care and training center of JUSH, Gebretsadik Shawo hospital and its laboratory staffs especially Melkam Tesfaye, my colleagues Mr.Lealem Gedefaw, Mr.Kedir Abdela and Ms.Serkadis Debalke for their advice and suggestion in the preparation of this document and my wife Ekram Ahmed for her support in different aspects to reach me in this event.

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## ABBREVIATIONS AND ACRONYMS

| | |
|---|---|
| 3TC | Lamivudine |
| ABC | Abacavir |
| AIDS | Acquired Immuno-Deficiency Syndrome |
| AOR | Adjusted Odds Ratio |
| ART | Antiretroviral Therapy |
| AZT | Zidovudine |
| BMI | Body Mass Index |
| CD4 | Cluster of Differentiation 4 |
| CHE | Cholesterol esterase |
| CHOD | Cholesterol oxidase |
| CI | Confidence Interval |
| CLS | Clinical laboratory science |
| CM | Centimeters |
| COR | Crude Odds Ratio |
| D4T | Stavudine |
| DBP | Diastolic Blood Pressure |
| DDI | Didanosine |
| DM | Diabetes Mellitus |
| EFV | Efavirenz |
| FPG | Fasting Plasma Glucose |
| GK | Glycerol kinase |
| GPO | Glycerol Phosphate Oxidase |
| HAART | Highly Active Anti-Retroviral Therapy |
| HDL | High Density Lipoprotein |
| HIV | Human Immunodeficiency Virus |
| IDDM | Insulin-Dependent Diabetes Mellitus |
| IDF | International Diabetes Federation |
| IDV | Indinavir |
| IFG | Impaired Fasting Glucose |
| IR | Insulin Resistance |

| | |
|---|---|
| LDL | Low Density Lipoprotein |
| LPV | Lipoinavir |
| LS | Lipodystrophic syndrome |
| NIDDM | Non-Insulin-Dependent Diabetes Mellitus |
| NNRTIs | Non-Nucleoside Reverse Transcriptase Inhibitors |
| NRTIs | Nucleoside Reverse Transcriptase Inhibitors |
| NVP | Nevirapine |
| OR | Odds Ratio |
| PLWHA | People living with HIV and AIDS |
| POD | Peroxidase |
| R | Ritonavir |
| SBP | Systolic Blood Pressure |
| SOP | Standard operating procedures |
| TDF | Tenofovir Disoproxil Fumarate |
| TC | Total Cholesterol |
| TG | Triglyceride |
| UNAIDS | United Nations Programme on HIV/AIDS |
| VLDL | Very Low Density Lipoprotein |
| WHO | World Health Organization |

## OPERATIONAL DEFINITIONS

**Diabetes mellitus-**fasting plasma glucose value ≥ 126 mg/dl and who have the three sign and symptoms of diabetes mellitus.

**Dyslipidemia** -is defining as TC ≥200 mg/dl, HDL-c < 40 mg/dl, LDL-c ≥130 mg/dl, TG ≥150 mg/dl and TC/HDL-c ratio≥5.

**Impaired fasting glucose** - fasting plasma glucose value between 110-125mg/dl.

**Adult** –a person whose age ≥18 years.

**Body mass index -** is calculated as weight in kg divided by height in meters squared and defined as BMI <18.5 $kg/m^2$ is considered to be underweight, between 18.5 – 24.9$kg/m^2$ normal , ≥25-29.9$kg/m^2$ overweight and ≥30$kg/m^2$ obesity.

# CHAPTER ONE: INTRODUCTION

## 1.1 BACKGROUND

Diabetes mellitus (DM) is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action or both [1]. Diabetes mellitus is a group of diseases in which blood glucose levels are elevated and it is the most common set of disorders of carbohydrate metabolism [2]. The disorders of diabetes differ in their etiology and symptoms and in the consequences of disease. Four types of diabetes have been classified. These are type 1, type 2, gestational diabetes and other specific causes of diabetes [3].

Type 1 diabetes mellitus(T1DM) represents approximately 10% of all cases of diabetes. There is an autoimmune destruction of insulin-producing beta cells in the islets of the pancreas, causing an absolute deficiency in insulin production and commonly occurs in childhood and adolescence. The cell mediated response causes infiltration of the pancreas and reduction in the volume of beta cells [1, 3].

T1DM is initiated by an environmental factor or infection in individuals with a genetic predisposition and causes the immune destruction of the β-cells of the pancreas and, therefore, a decline synthesis of insulin. T1DM is characterized by those of having a sudden onset, insulin dependence, and ketosis tendency. One or more of the following markers are found in 85% to 90% of individuals with fasting hyperglycemia: islet cell autoantibodies, insulin autoantibodies [1]. At the latter stage of the disease, there is little or no insulin secretion, as manifested by low or undetectable levels of plasma C-peptide [4].

Type 2 diabetes mellitus (T2DM) is characterized by decline in insulin action due to the resistance of tissue cells to the action of insulin. The problem is intensified by the inability of the beta cells of the pancreas to produce enough insulin to counteract the resistance. It is a disorder of both insulin resistance and relative deficiency of insulin [3].

T2DM constitutes the majority of the diabetes cases. Most patients in this type are obese or have an increased percentage of body fat distribution in the abdominal region. This type of diabetes often goes undiagnosed for many years and is associated with a strong genetic predisposition, with patients at increased risk with an increase in age, obesity, and lack of physical exercise.

Characteristics usually include adult onset of the disease and milder symptoms than in type 1, with ketoacidosis seldom occurring [1].

T2DM accounts 90–95% of all diabetes cases and previously referred to as non–insulin-dependent diabetes mellitus (NIDDM) or adult onset diabetes. At least initially, and often throughout their lifetime, these individuals do not need insulin treatment to survive. There are probably many different causes of this form of diabetes. Although the specific etiologies are not known, autoimmune destruction of beta-cells does not occur, and patients do not have any of the other causes of diabetes. Patients who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal region [4]

Gestational diabetes (GDM) is any degree of glucose intolerance with onset or first recognition during pregnancy. Causes of GDM include metabolic and hormonal changes. Patients with GDM frequently return to normal postpartum. However, this disease is associated with increased perinatal complications and an increased risk for development of diabetes in later years [1]. GDM is similar in etiology to T2DM; however, it is defined as diabetes that is diagnosed in pregnancy. Pregnancy is associated with increased tissue cell resistance to insulin. Most pregnant women will compensate with increased secretion of insulin; those individuals who are unable to compensate may develop gestational diabetes [3].

Other specific causes of diabetes; this form of hyperglycemia may be the secondary result of non–insulin-related events. Blood glucose levels are increased in endocrine disorders, such as Cushing's syndrome; in exocrine disorders, such as cystic fibrosis; and as a response to specific drugs, such as protease inhibitors and glucocorticoids. Other causes of this form of diabetes are the result of genetic defects that affect pancreatic beta cells or the action of insulin [3]. DM is a common disorder affecting individuals of all ages. Similar to general population, DM can also be seen in HIV infected individuals.

Three subgroups of patients with diabetes and HIV can be identified: Patients with preexisting diabetes that contract HIV, those who are diagnosed to have diabetes at onset of HIV infection, and others who develop hyperglycemia after start of highly active antiretroviral therapy (HAART) [5].

HAART is based on the use of a class of drugs which have been used extensively as antiretroviral agents. Benefits of HAART include suppression of viral load, improvement in cluster of differentiation 4($CD_4$) count, decrease in opportunistic infections and length of hospital stay, and reduction in mortality [5]. Nowadays, both the diagnostic tests of HIV and diabetes are improved and become more advanced.

Glucose can be measured from serum, plasma, or whole blood. Most of the glucose measurements are performed on serum or plasma [3].Before the discovery of different enzymes for determination of glucose, reducing agent has been useful in the detection and quantitation of carbohydrates in body fluids. Glucose and other carbohydrates are capable of converting cupric ions in alkaline solution to cuprous ions. The solution loses its deep-blue color and a red precipitate of cuprous oxide forms. Benedict's and Fehling's reagents, which contain an alkaline solution of cupric ions stabilized by citrate or tartrate, respectively, have been used to detect reducing agents in urine and other body fluids [1].

Three enzyme systems are commonly used to measure glucose: glucose oxidase, hexokinase and glucose dehydrogenase. These reactions produce an electrical current that is proportional to the initial glucose concentration, or a product that measured spectrophotometrically is proportional to the initial glucose concentration. The assays can be initial rate-of-change assays, where the velocity of the reaction is dependent on the initial glucose, or end-point assays [2].

The peroxidase coupling reaction used in the glucose oxidase method is subject to positive and negative interference. Increased levels of uric acid, bilirubin, and ascorbic acid can cause falsely decreased values as a result of these substances being oxidized by peroxidase, which then prevents the oxidation and detection of the chromogen. Strong oxidizing substances, such as bleach, can cause falsely increased value [1-2].

The hexokinase method is considered more accurate than the glucose oxidase methods because the coupling reaction using glucose-6-phosphate dehydrogenase is highly specific; therefore, it has less interference than the coupled glucose oxidase procedure. The hexokinase method is accepted as the reference method, this method is not affected by ascorbic acid or uric acid. Gross hemolysis and extremely elevated bilirubin may cause a false decrease in results [1, 3].

## 1.2 STATEMENT OF THE PROBLEM

The global burden of diabetes is rising dramatically worldwide and is causing a high health burden in low- and middle-income countries [9]. The International Diabetes Federation (IDF) estimates that 382 million people have diabetes in 2013; by 2035 this will rise to 592 million worldwide. In 2013 diabetes caused 5.1 million deaths globally, every six seconds a person die with diabetes. Almost 80% of diabetes deaths occur in low- and middle-income countries [10].

As the disease progresses, individuals are at increased risk for the development of specific complications including retinopathy (which may lead to blindness), renal failure, neuropathy (nerve damage) and arteriosclerosis. The last condition may result in stroke, gangrene or coronary artery disease[11].

HIV/Acquired Immuno-Deficiency Syndrome (AIDS) is still considered as a disastrous infectious disease with a high impact on quality of life among individuals and communities. It weakens economic and social performances which manifests along with poor mental and/or physical health [12]. According to the report of global AIDS epidemic 2013 by the Joint United Nations Programme on HIV/AIDS (UNAIDS), in the year 2012 an estimated 35.3 million [32.2 million – 38.8 million] people globally were living with HIV, 2.3 million [1.9 million – 2.7 million] people became newly infected with HIV and 1.6 million [1.4 million – 1.9 million] people died from AIDS-related illnesses in the world [13].

In 2013, 12.9 million people living with HIV were receiving antiretroviral therapy (ART) globally, of which 11.7 million were receiving ART in low- and middle-income countries [14].

In 2012, a total of 25 million people in Sub-Saharan Africa were living with HIV, 1.6 million newly infected individuals and 1.2 million were estimated to have died due to AIDS[15]. By killing the economically active population in particular, HIV/AIDS is destroying the very fabric of societies throughout the continent [16]. The cause of death due to DM is increasing in the era of HAART [17].

Introduction of HAART in developing countries with a high prevalence of HIV has been recognized as a public health priority. The number of people with access to HAART in sub-Saharan Africa is estimated to have increased 10-fold over the last three years [10].

As treatment of HIV develops and access to therapy improves, the incidence of HIV-associated diabetes is bound to grow. HAART lead to an increase in metabolic dysfunction, including insulin resistance, diabetes, dyslipidemia, lipodystrophic and cardiovascular disease [5]. HIV infected persons may be at increased risk for developing T2DM because of viral co infection and adverse effects of treatment [6-8].

DM is a common disorder affecting individuals of all ages. Similar to general population, DM can also be seen in HIV infected cases. The prevalence of insulin resistance, glucose intolerance, and diabetes in the HIV-infected population has increased dramatically following the widespread use of HAART [18].

The introduction of HAART revolutionized the prognosis of HIV/AIDS. However, the use of such treatment regimen has coincided with the emergency of body fat distribution modification (lipodystrophy) and alteration in lipid and glucose metabolism, a condition known as lipodystrophic syndrome (LS) . Though the introduction of HAART improved survival and quality of life, early data from those treated raised concerns about a possible increase in both peripheral and coronary arterial disease through lipodystrophy, diabetes mellitus and dyslipidemia [19- 21].

The most important metabolic abnormalities observed in HIV-patients during ART include: insulin resistance (IR) with hyperinsulinemia, glucose intolerance and hypercholesterolemia with low levels of high density lipoprotein (HDL), hypertriglyceridemia, lactic acidemia and hypercoagulopathy. Lipodystrophy also associates fat redistribution: subcutaneous lipid stores wasting (Bichat's fat pad, buttocks and extremities), central adiposity and fat accumulation in the dorsocervical region (lipohypertrophy). Glucose intolerance and DM are becoming more common in HIV-infected patients in the era of HAART [22- 25].

The toxicities associated with HIV therapy are directly influencing the emerging diabetes crisis in Africa. Antiretroviral therapies (ART), Protease inhibitors (PIs) and thymidine analogue Nucleoside Reverse Transcriptase Inhibitors (NRTIs), contribute to dyslipidemia and insensitivity to insulin, and thereby to cardiovascular risk. Interrupting HIV treatment may also contribute to a higher risk for cardiovascular disease, suggesting that HIV infection in itself may contribute to the formation of plaques in the lining of the arteries (atherogenesis) [16, 26].

Metabolic abnormalities including dyslipidemia, insulin resistance, diabetes, excessive loss of fat beneath the skin (lipoatrophy) – resulting in sunken cheeks, indentations, and hollow eyes - and increased inflammatory indexes have been increasingly observed among HIV-infected patients in the current era of HAART. The causes of these abnormalities are complex and multifactorial, likely related in part to the effects of HIV itself, a chronic inflammatory condition, medication effects and changes in body composition with relative losses in subcutaneous fat and gains in central adiposity with the institution of antiretroviral therapy. These changes may place HIV-infected patients at greater risk of cardiovascular disease [16, 27-29].

Over 25 million people in sub-Saharan Africa are infected with HIV, representing nearly 70% of the world's total population of people living with HIV and AIDS (PLWHA) [15]. In 2012, 7.5 million Africans were on HAART [30]. However, as children and adults are living longer on HAART, there is increasing concern about rising incidence of insulin resistance, glucose intolerance, T2DM, and dyslipidemia among PLWHA [31].

In 2011, about 1.2 million people were estimated to be living with HIV in Ethiopia [32] and among them 62% had access to ART [33].

It is difficult to manage co morbidities of the two chronic diseases and it will increase the morbidity and mortality of the HIV/AIDS patients. In Ethiopia, the magnitude of DM in HIV/AIDS individuals is not known and there is inadequate information about DM in HIV infected individuals. Therefore, the aim of this study is to assess the magnitude of diabetes mellitus and associated factors in HIV/AIDS patients.

# CHAPTER TWO: LITERATURE REVIEW

According to the report of global AIDS epidemic 2013 by the Joint United Nations Programme on HIV/AIDS (UNAIDS), in the year 2012 an estimated 35.3 million people globally were living with HIV, 2.3 million people became newly infected with HIV and 1.6 million people died from AIDS-related illnesses in the world. In 2012, 7.5 million Africans were on HAART [30]. However, as children and adults are living longer on HAART, there is increasing concern about rising incidence of insulin resistance, glucose intolerance, T2DM, and dyslipidemia among PLWHA [31].

A study conducted in USA in 2009 showed that 3,327 HIV-infected subjects have 14.9% prevalence of diabetes mellitus at baseline. HIV infected individuals without ART had a lower risk of diabetes but HIV infected individuals who were on ART had higher risk for the development of diabetes mellitus [34]. Also, an increasing age, male gender, high body mass index (BMI), family history of diabetes and smoking were associated with an increased risk for diabetes mellitus [7, 29, 34-35]. Hepatitis C virus (HCV) coinfection was associated with a higher risk of diabetes in HIV infected individuals [12, 29, 34].

According to a study conducted in 2005 in California among 101 patients with HIV infection, patients who were taking PIs had higher serum triglyceride levels than patents that are not taking PIs. Fasting plasma glucose (FPG) was higher in HIV patients taking PIs compared to non-PI-treated. Among those who were taking PIs, 10 (12%) subjects were found to have diabetes. Diabetic subjects on PIs had significantly higher serum triglyceride (TG) levels [36]. On the other side, nucleoside and non-nucleoside reverse transcriptase inhibitor therapy were associated with a higher risk of diabetes in HIV infected veterans [34].

Another study conducted in China in 2013 among a total of 2006 HAART naïve patients indicated that 211 (10.52%) had diabetes [37].

According to a study done in Tamil Nadu India in 2012 among 145 HIV-positive patients on ART and 146 HIV-positive patients not on ART, dyslipidemia was significantly higher in the treatment group than ART-naive. Total duration of ART was significantly associated with dyslipidemia [38].

According to a study done in Malaysia in 2013 among 1,583 adult HIV infected population on antiretroviral medication, dyslipidemia was common in 82.3 % of the subjects. Majority of the studied population were male (81.1%) and aged between30–49 (68.6%). Increased levels of triglyceride (TG) and total cholesterol (TC) were found among 59% and 54.2% of the population. However, declined level of high-density lipoprotein (HDL), raised low-density lipoprotein (LDL) and fasting plasma glucose (FPG) were found among 28.7%, 35.1% and 38.2% of the population, respectively. Protease inhibitor (PI) was a potential risk for elevated triglyceride, high TC and low HDL [12].

A research done in Australia in 2010 among 788 HIV-infected adult showed that there is high prevalence of lipid disturbances [21]. Another study conducted in Nigeria among 130 HIV positive patients showed lipid abnormalities including significant elevation of LDL and reduced level of HDL and TC compared with HIV negative controls. There were significant increase in the mean BMI and mean HDL at 12 months [25].

A total of 384 HIV/AIDS patients on ART at Muhimbili National Hospital, Tanzania in 2010 were tested for diabetes mellitus and the prevalence of diabetes mellitus was 95 (24.7%), from these 49 (51.5%) were males and 46 (48.5%) were females. Females were the group most affected by HIV/AIDS and males were the group most affected by diabetes. HIV/AIDS patients who were using PI's had diabetes [39]. PI usages were significant factors for the development of diabetes mellitus in HIV infected individuals [35].

A study conducted in Senegal in 2012 with 242 patients showed that the prevalence of diabetes mellitus to be 14.5%. The frequency of diabetes was higher in patients' ≥45 years of age and in those with long duration of ART treatment (≥119months). Those who had hypercholesterolemia or elevated triglycerides also had higher prevalence of diabetes. ART, age, BMI at treatment initiation, levels of total cholesterol and triglycerides, and duration of exposure to Stavudine (d4T), Didanosine (ddI), and Indinavir (IDV) have strong association with diabetes mellitus [40].

A study conducted in Thai in 2004, among 278 HIV-infected individuals showed at least one abnormality in either lipid or glucose metabolism. Eighty-eight percent had dyslipidemia, 30% had insulin resistance and 27% had diabetes mellitus [41]. Another study conducted in Kigali,

Rwanda in 2007 with a total of 409 individuals showed that 370 (90%) were on ART regimen and 140 (34%) had lipid abnormalities [42].

A study conducted in Hawassa, Ethiopia in 2012 among 93 (82.3%) of HAART and 87 (76.9%) pre-HAART patients showed hypercholesterolemia among 43.4% of HAART and 15.9% of pre-HAART patients, whereas HDL-cholesterol below 40 mg/dl occurred in 43.4% and in 63.7% respectively. The LDL-cholesterol≥130 mg/dl occurred in 33.6% of HAART and 15% pre-HAART patients, while triglycerides≥150 mg/dl occurred in 55.8% and 31.0% respectively. Receiving of HAART was significantly and positively associated with raised total cholesterol, LDL-cholesterol, and triglycerides. The number of patients on ZDV/3TC/EFV, and ZDV/3TC/NVP regimens were 22 (19.5%) and 36 (31.8%) respectively, while those on d4T/3TC/EFV, and d4T/3TC/NVP regimens were 24(21.2%) and 31 (27.4%) respectively [32].

A study done in Welayta Sodo, Ethiopia in 2013 among 176 HIV/AIDS positive individuals indicated the prevalence of diabetes mellitus to be 8% of the subjects. Hypertension was seen in 15.9% of the subjects. Age, duration of HAART, lipoatrophy and lipohypertrophy were significantly associated with diabetes mellitus while only lipohypertrophy remained to be significantly associated with hypertension [19].

Even though, different studies are conducted on HAART experienced HIV/AIDS to explore the magnitude of DM, there is scarcity of literature on magnitude of DM that are done in HAART naïve HIV infected individuals. Therefore, this study will involve both HAART naïve and HAART experienced HIV infected individuals.

## 2.2 CONCEPTUAL FRAME WORK

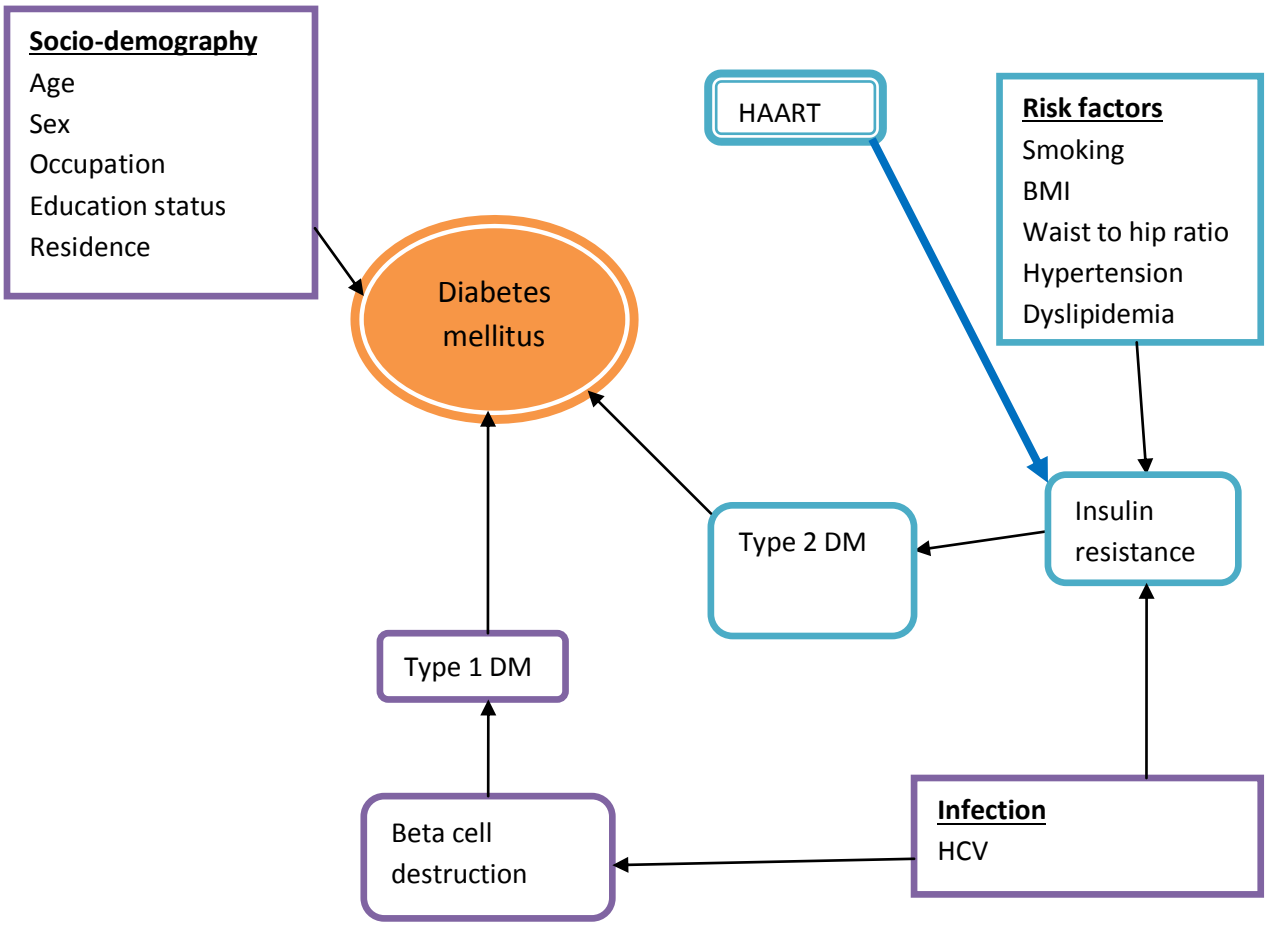


Figure 1. Conceptual framework

## 2.3 SIGNIFICANCE OF THE STUDY

The magnitude of DM in HIV infected individuals is increasing dramatically in the era of HAART. However, as far as our knowledge is concerned there is no published research on DM in both HAART experienced and HAART naïve HIV infected individuals in Ethiopia. Therefore; the finding of this study will provide information about the magnitude of diabetes mellitus in HIV infected individuals. Assist policy makers, programme evaluators and local government to be involved in prevention and control strategies, provide baseline data for further study, provide information to the HIV infected individuals about diabetes mellitus, give information to health worker and other concerned bodies for early detection and treatment of DM in HIV infected individuals patients and to prevent further complications.

# CHAPTER THREE: OBJECTIVES

## 3.1 GENERAL OBJECTIVES

- ✓ To assess the magnitude of diabetes mellitus and associated factors among HIV infected individuals at JUSH, comprehensive chronic care and training center.

## 3.2 SPECIFIC OBJECTIVES

- ✓ To determine the overall prevalence of DM in HIV infected individuals.
- ✓ To determine associated factors of diabetes mellitus in HIV infected individuals.

# CHAPTER FOUR: METHODS AND MATERIALS

## 4.1. STUDY DESIGN AND PERIOD

An institution based cross sectional study design was conducted at JUSH, comprehensive chronic care and training center from April 5- May 30, 2014.

## 4.2. STUDY AREA

The study was conducted in Jimma University Specialize Hospital (JUSH), comprehensive chronic care and training center. Jimma is found in Jimma zone, Jimma town Woreda, the town is located 352 km southwest of capital Addis Ababa. The town is divided into 13 kebeles. According to the 2007 Central Statistical Agency census report the total population of the town is 120, 960 from this 60,824 were males and 60,136 females. The town has a characteristic of tropical high land climate condition, heavy rain fall, warm temperature and long wet period. A total of 7,288 HIV positive individuals are registered/ attending at JUSH, comprehensive chronic care and training center during April 2014. Of these 4277 are on ART [43]. JUSH have one diabetic's clinic that serves for 2000 diabetic patients and one laboratory.

## 4.3. SAMPLE SIZE AND SAMPLING TECHNIQUE

### 4.3.1. SAMPLE SIZE DETERMINATION

The sample size (n) is calculated by using single population proportion formula as follows:

$$n = \frac{(Z_{1-\alpha/2})^2 \times p(1-p)}{d^2}$$

Where: n= minimum sample size

p =an estimate of prevalence

d = accepted margin of error

$Z_{1-\alpha/2}$=standard normal variance with 95% confidence interval

I use p 50% to obtain maximum sample size.

$$n = \frac{(1.96)^2 \times 0.5(1-0.5)}{(0.05)^2}$$

n = 385

The current total number of HIV infected individuals in JUSH, comprehensive chronic care and training center is 7288(N).So, the source population is less than 10,000 and sample size correction is implemented and the sample size become 366 and with 10% contingency for hemolyzed and inappropriate specimens the final sample size is 403.

n=n/1+n/N

n=385/1+385/7288

n=366 and 10% contingency=37

n= 403(Final sample size)

### 4.3.2 SAMPLING TECHNIQUES

Up to April 2014, there are 7,288 HIV infected individuals registered/attending JUSH, comprehensive chronic care and training center, of these 4,277 are on ART and the rest are in pre-ART stage. Convenient sampling technique was implemented and the samples were taken consecutively.

## 4.4 POPULATION

### 4.4.1 SOURCE POPULATION

All HIV positive persons visiting Jimma University specialized hospital, comprehensive chronic care and training center.

### 4.4.2 STUDY POPULATION

All adult HIV positive persons visiting Jimma University specialized hospital, comprehensive chronic care and training center, volunteer to participate in the study and available during the study period.

## 4.5. ELIGIBILITY CRITERIA

### 4.5.1. INCLUSION CRITERIA

All adult HIV-infected individuals who are on HAART for a minimum of greater than 6 months and HAART treated group to have a good ART adherence (adherence rate ≥ 95%). A good adherence is defined by missing < 2 dose of 30 doses or < 3 dose of 60 doses; and it was adopted

from Ethiopian Federal Ministry of Health, HIV Care/ART follow-up form. Additionally pre-HAART individual those who never get HAART yet.

### 4.5.2 EXCLUSION CRITERIA

Patients who had had their therapy regimens changed during follow-up within the last six months would not be included. Participants receiving lipid altering therapies, pregnant women, known diabetes mellitus patients on treatment and renal failures were excluded.

## 4.6. STUDY VARAIBLES

### 4.6.1. DEPENDENT VARIABLES

Diabetes mellitus

### 4.6.2 INDEPENDENT VARIABLES

Age

Sex

Religion

Ethnicity

Residence

Marital status

Occupation

Educational status

BMI

HCV infection

Waist circumference

Hip circumference

Waist-hip ratio

Dyslipidemia

Family history of diabetes mellitus

Smoking

Types of HAART regimen

Duration of HAART

Sign and symptoms

Hypertension

HIV duration

WHO clinical staging of HIV/AIDS

## 4.7 DATA COLLECTION AND PROCESSING

### 4.7.1 SOCIO-DEMOGRAPHIC DATA COLLECTION

The data of the socio-demographic and anthropometric was collected using structured questionnaire by skilled senior clinical nurses.

### 4.7.2 GLUCOSE TEST (GOD-PAP METHOD)

Glucose was determined after enzymatic oxidation in the presence of glucose oxidase at $37^{O}C$ for 5 minutes. The formed hydrogen peroxide reacts under catalysis of peroxidase, POD with phenol and 4-aminophenazone to red-violet quinoemine dye as indicator. The intensity of the pink color was measured at 546 nm.

Glucose was analyzed by Dr.Lang 80 spectrophotometer at Biochemistry laboratory, Jimma University. Glucose liquicolor, Human Gesellschaft fur Biochemica und Diagnostica mbH, Germany reagent were used to analyze the glucose test.

The WHO diagnostic criteria for diabetes is when fasting plasma glucose (FPG) $\geq$126 mg/dl, impaired fasting glucose is when FPG ranges from 110 -125mg/dl) [44]. When non-fasting plasma glucose is $\geq$200 mg/dl it is also considered to be diabetic [6].

Test principle

$$\text{Glucose} + O_2 \xrightarrow{\text{Glucose oxidase}} \text{Gluconic acid} + H_2O_2$$

$$2H_2O_2 + 4\text{AP} + \text{Phenol} \xrightarrow{\text{POD}} 4H_2O + 4(\text{P-benzoquinoneimine phenazone})$$

### 4.7.3 LIPID PROFILE TESTS

The blood specimen was collected with senior medical laboratory technologist, the serum was separated from the whole blood within 30 minutes and the serum was transferred to Nunc tubes and stored in -20 $^{0}C$ refrigerators. After all the specimens were collected the serum was packed with ice and transported to Gebretsadik Shawo hospital (Bonga) and the lipid profile tests were

done at this hospital laboratory with HumaStar 80 (Germany) spectrophotometer. Human Gesellschaft fur Biochemica und Diagnostica mbH, Germany reagent (Cholesterol liquicolor, triglyceride liquicolor and HDL liquicolor) were used to analyze the lipid profile tests.

### 4.7.3.1 TOTAL CHOLESTEROL (CHOD-PAP METHOD)

Cholesterol is enzymatically oxidized by cholesterol esterase (CHOD) after hydrolysis of its esters with a fungal lipase. Hydrogen peroxide released produces the oxidative coupling of phenol with 4-aminophenazone (4AP) by means of a reaction catalyzed by peroxidase (POD) yielding quinoneimine which is read at 505nm.

Test principle

Cholesterol ester $+H_2O \xrightarrow{CHE}$ cholesterol + Fatty acids

Cholesterol $+ O_2 \xrightarrow{CHOD}$ Cholesten-3-one $+ H_2O_2$

$H_2O_2$ + 4AP + Phenol $\xrightarrow{POD}$ 4(P-benzoquinoneimine phenazone) $+ 4H_2O$

### 4.7.3.2 TRIGLYCERIDES TEST (GPO-PAP METHOD)

Triglycerides were determined after enzymatic hydrolysis with lipases. Indicator is quinoneimine formed hydrogen peroxide, 4-aminoantipyrine and 4-chlorophenol under the catalytic influence of peroxidase.

Test principle

Triglyceride $\xrightarrow{Lipase}$ Glycerol + Fatty acids

Glycerol + ATP $\xrightarrow{GK}$ Glycerol-3-phosphate + ADP

Glycerol-3-phosphate $O_2 \xrightarrow{GPO}$ Dihydroxyacetone phosphate $+ H_2O_2$

$H_2O_2$ + 4-Aminoantipyrine $\xrightarrow{POD}$ Quinoneimine + HCL $+ H_2O$ + 4-Chlorophenol

### 4.7.3.3 HIGH DENSITY LIPOPROTEIN CHOLESTEROL TEST

The assay combines two steps. In the first step chylomicrons, VLDL and LDL cholesterol are specifically eliminated and destroyed by enzymatic reactions. In the second step, remaining cholesterol formed from the HDL fraction is determined by well-established specific enzymatic reactions in the presence of surfactants for the HDL.

Reaction principle

**First step**

LDL, VLDL, chylomicrons $\xrightarrow{\text{CHE-CHO}}$ Cholestenone + $H_2O_2$

$2H_2O_2 \xrightarrow{\text{Catalase}} 2H_2O + O_2$

**Second step**

HDL $\xrightarrow{\text{CHE-CHO}}$ Cholestenone + $H_2O_2$

$H_2O_2$ + Chromogen $\xrightarrow{\text{POD}}$ quinine pigment

**Dyslipidemia is defined as TC≥200 mg/dl, HDL-c < 40 mg/dl, LDL-c≥130 mg/dl, TG≥150 mg/dl and TC/HDL-c ratio≥5 by the United States National Cholesterol Education Program, Adult Treatment Panel (NCEP-ATP) III guidelines [45].**

### 4.7.3.4 LOW DENSITY LIPOPROTEIN CHOLESTEROL

LDL was calculate by this formula; LDL= TC - HDL - (TG/5) [46].

### 4.7.4 HCV TEST

**Test principle**

The EUGENE Anti-HCV rapid test is a lateral flow, immunochromatographic assay, screening serum or plasma using recombinant HCV proteins. Recombinant antigens of HCV labeled by gold conjugates are used in test line as capture materials, and anti-rabbit HCV antibody is used in the control line. When a sample is added in to the sample pad of the test strip, it migrates through the membrane strip. If the antibodies to HCV present in the specimen, a complex of antibody-gold conjugate recombinant antigens was formed, which is then captured by antigen immobilize in the test line region of the membrane , producing a visible pink color line of immune complex conjugate on the membrane. The color intensity will depend on the concentration of the anti-HCV present in the sample. Absence of the test line suggests a negative result. The test contains an internal control (control lone) which should exhibit a pink colored line of the immunocomplex conjugate regardless of color development on the test line. Otherwise, the test result is invalid and the specimen must be retested with another test strip.

### 4.7.5 ANTHROPOMETRIC PARAMETERS

#### 4.7.5.1 Weight

Participants were asked to stand, face forward, and place arms on the sides of the body with light clothes. Weight was record in kilograms [47].

#### 4.7.5.2 Height

Height was measure without the participants wearing foot ware or head gear. Before the reading was taken, the participants were requested to have their feet together, heels against the back board, knees straight, and look straight ahead. Height was record in centimeters [47].

#### 4.7.5.3 BMI

The height and body weight were measured to a precision of 0.1 cm and 0.1 kg, respectively, utilizing a digital balance with height measurement attached to the digital balance [19]. Body mass index (BMI), was calculated as weight in kilograms divided by the square of the height in meters and was stratified in to $<18.5 kg/m^2$ (underweight), 18.5–24.9 $kg/m^2$ (normal), 25–29.9 $kg/m^2$ (overweight), and $\geq$ 30 $kg/m^2$ (obese) [6].

#### 4.7.5.4 Waist circumference

This measurement was taken in a private area. The midpoint between the inferior margin of the last rib and the crest of the ilium was marked using a tape measure. With the assistance of the participants, the tape measure was wrapping around the waist directly over the skin or light clothing. Just before the measurement was taken, participants were requested to stand with their feet together, place their arms at their side of their body with the palms of their hands facing inwards, and breathe out gently. The cut of values are 102 cm for male and 88 cm for females and the values greater than this numbers indicates risk factor for obesity [52].

#### 4.7.5.5 Hip circumference

The measurement was taken in a private area immediately after the measurement of waist circumference in centimeters. The measurement was taken at the maximum circumference over the buttocks, after requesting the participants to relax their arms at the sides [47].

#### 4.7.5.6 Waist to hip ratio

It was calculated by dividing waist in centimeters (cm) to hip in centimeters. The cut off values are 0.90 cm to male and 0.85cm for females and values greater than the cut off value are at increased risk for obesity [52].

## 4.8 BLOOD PRESSURE

The blood pressure was taken by skilled clinical nurses. After the patient take rest at least for five minutes the first blood pressure measured and between each measurement the patient rest for five minutes. We chose to take an average of the second and third readings as recommended by World Health Organization.

Systolic blood pressure (SBP) is stratified into four levels: <140 mmHg (normal), 140-169 mmHg (mildly raised), 170-179 mmHg (moderately raised), and 180+ mmHg (severely raised); similarly diastolic blood pressure (DBP) is stratified into four levels: < 90 mmHg (normal), 90-99 mmHg (mildly raised), 100-109 mmHg (moderately raised), and 110+ mmHg (severely raised) [53].

A participant is classified as having normal blood pressure if SBP and DBP was both normal; Meanwhile, a participant is classified as having mild hypertension when SBP or DBP is mildly raised; moderate hypertension when SBP or DBP is moderately raised; and severe hypertension when SBP or DBP is severely raised [53].

## 4.9 PRE-TEST

Pre-test was done in 5% of the total sample size, the result was determined and the necessary measures on the questionnaire and in the test method was taken.

## 4.10 QUALITY ASSURANCE

Blood specimen was collected based on the standard protocol for blood collection that was described in the SOP and training was given for the data collectors prior to the actual work started. The reagents and instruments were checked before the start of the actual work for proper functioning. Both pathological and normal quality control was run for each type of test for detecting of errors and to validate patient result. The anthropometric measurements were done strictly by skilled senior clinical nurse. The questionnaire was translated to local languages and checked for consistency and completeness.

## 4.11 DATA ANALYSIS

Data from the laboratory investigation and structured questionnaires was coded and entered to Epi info version 7 and analyzed using SPSS version 20. Descriptive statistics (tables and figures) and inferential statistical tests like chi-square test, bivariate and multivariate logistic regression tests were implemented. For variables that do not satisfy the assumptions of chi-square test, the Fisher's exact test was used instead of Pearson chi-square test to test the association between the dependent variable and the independent variables. The variables with p-value less than 0.25 in bivariate logistic regression analysis were nominated for multivariate logistic regression analysis.

Before running of multiple binary logistic regressions Hosmer and Lemeshow goodness of fit test was done. In multivariate logistic regression test p-values less than 0.05 was taken as cut off value for significant association among the dependent and independent variables. The result was discussed and compared with different literatures on prevalence of diabetes mellitus and associated factors on fasting blood specimens. Finally, recommendations were forward based on the findings.

## 4.12 ETHICAL CONSIDERATIONS

Ethical clearance was obtained from Jimma university ethical review committee and official permission letter to conduct the study was obtained from Jimma university specialize hospital administrators, written informed consent was taken from each study subjects after clear orientation of the study objective. Positive findings was reported to the clinician working at the comprehensive chronic care and training center for better management and all the results was kept confidential.

## 4.13 DISSEMINATION OF RESULT

The result of the study will be presented to the department of medical laboratory science and pathology, Jimma University as a fulfillment of master's degree in Clinical laboratory science (specialty in clinical chemistry); to JUSH administration, to Jimma University specialized

hospital, comprehensive chronic care and training center staffs and it will be published in scientific journal.

## 4.14 LIMITATIONS

A single fasting serum specimen was analyzed which lead to over estimation of the hyperglycemia and impaired fasting glucose values of the study participants. The time when the HIV/AIDS patients develop the diabetes was not well-known either before or after initiation of HAART and we did not included this data because the study participants were not aware of this and no any test/information concerning diabetes is found in their card. Also, only the three signs and symptom of diabetes were assessed.

# CHAPTER FIVE: RESULTS

## 5.1 Characteristics of the study participants

A total of 403 study participants were involved in this study. Ten of the samples were incomplete and they are excluded from analysis. A 393 HIV infected individuals of age ranging from 21 to 75 years with mean age of 37.9 ±11.18 (mean +SD) had participated in this study. Two hundred eighty four (72.3%) of the study participants were HAART experienced and the rest 109 (27.7%) were HAART naive. The majority (71.2%) of the study participants were less than 40 years. Most of the study participants (66.9%) were females and 348 (88.5%) of the study participants were from urban areas. One hundred eighty six (47.3%) of the study participants were employed. Most of the study participants were literate (78.4%) and married (62.3%). About half of the study participants (51.4%) were orthodox and 153 (38.9%) of the study participants were Oromo (Table 1).

## 5.2 Magnitude of diabetes mellitus in the study participants

The overall prevalence of diabetes mellitus in this study was 6.4 % (n=25). Seven of the DM positive individuals were know about their DM status before our study and they do not start drug. Two hundred ninety one (74%) and 77(19.6%) of the study participants had normal glucose value (70-110 mg/dl) and impaired fasting glucose value (111-125 mg/dl) respectively (Figure 1).

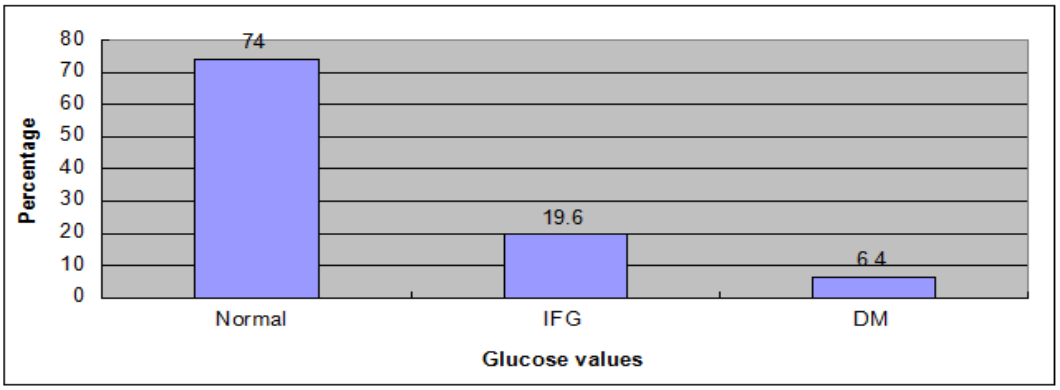


**Figure 2. . Classification of serum glucose test values of HIV infected individuals at JUSH, comprehensive chronic care and training center southwest Ethiopia, 2014.**

### 5.3 Risk factors associated with diabetes mellitus

In this study, several socio-demographic and other predisposing factors to DM were assessed. With regard to the association of DM with age, 15 (13.3%) and 10 (3.6%) of the study participants who were DM positive were >40 and <40 years, respectively. There was a strong statistically significant association between DM and age (P=0. 001). In addition to this, 11(5.9%) and 10(11.4%) of the employed and merchant of the study participants were positive for DM respectively (Table 1).

On the other side, 17(6.5%), 21(6.8%), 12(4.9%), 22(6.3%) and 24(8.5%) of the females, literates, married, urban residents and HAART experienced of the study participants were DM positive respectively. There was no significant association between DM and gender (P= 0. 906), educational status (P=0. 480), but, HAART status had statistically significant association with DM (P=0.006) (Table 1).

**Table 1. Socio-demographic factors versus DM in HIV infected individuals (n=393) at JUSH comprehensive chronic care and training center, Southwest Ethiopia, 2014.**

| Variables | | Glucose result: no (%) | | Total, no(%) | P-value of $X^2$ test |
|---|---|---|---|---|---|
| | | Normal | DM | | |
| Age | <40 years | 270(96.4) | 10(3.6) | 280(71.2) | 0.001 |
| | >=40 years | 98(86.7) | 15(13.3) | 113(28.8) | |
| Sex | Male | 122(93.8) | 8(6.20 | 130(33.1) | 0.906 |
| | Female | 246(93.5) | 17(6.5) | 263(66.9) | |
| Occupation | Farmer | 37(100) | 0 | 37(9.4) | |
| | Merchant | 78(88.6) | 10(11.4) | 88(22.4) | |
| | Employed | 175(94.1) | 11(5.9) | 186(47.3) | |
| | Unemployed | 78(95.1) | 4(4.9) | 82(20.9) | |
| Residence | Urban | 326(93.7) | 22(6.3) | 348(88.5) | |
| | Rural | 42(93.3) | 3(6.7) | 45(11.5) | |
| Educational status | Literate | 287(93.2) | 21(6.8) | 308(78.4) | 0.480 |
| | Illiterate | 81(95.3) | 4(4.7) | 85(21.6) | |
| Marital status | Married | 233(95.1) | 12(4.9) | 245(62.3) | |
| | Single | 50(94.3) | 3(5.7) | 53(13.5) | |
| | Widowed | 44(89.8) | 5(10.2) | 49(12.5) | |
| | Divorced | 41(89.1) | 5(10.9) | 46(11.7) | |
| Drug status | HAART experienced | 260(91.5) | 24(8.5) | 284(72.3) | 0.006 |

| | | | | |
|---|---|---|---|---|
| | HAART naive | 108(99.1) | 1(0.9) | 109(27.7) |

Thirty three (8.4%) of the study participants had the three signs and symptoms (polydipsia, polyuria and polyphagia) of DM and 58 (14.8%) of the study participants had one or two of the sign and symptoms. Among the study participants who have the three signs and symptom, 25(75.8%) of them were positive for DM.

Majority (88.3%) of the study participants had no family history of DM. Of these, 19 (5.5%) had DM. In relation to habit of smoking with DM, 326 (83%) of the study participants reported no habit of smoking. Among those who reported no history of smoking 22 (6.7%) were DM positive. There was no statistically significant association with DM in both family history of DM (P=0. 098) and smoking (P=0. 0.783).

With regard to types of HAART, 126 (32.1%) of the study participants used the drug AZT+3TC+NVP. Of these, 13 (10.3%) had DM. Additionally, 6 (7.1%) of the study participants who were using the drug TDF+3TC+EFV were positive for DM.

Almost greater than half (67.4%) and 44 (11.2%) of the study participants had a normal (18.5-25 $kg/m^2$) and overweight (25-29.9 $kg/m^2$) BMI values, respectively. Thirteen (4.9%) and 10 (22.7%) of the study participants who had normal and overweight BMI values were positive for DM, respectively.

Majority (85.8%) of the study participants were in clinical stage I of WHO classification. Of these, 24 (7.1%) had DM. On the other hand only 5 (1.3%) of the study participants were positive for HCV antibody test and all the study participants who had DM were negative for HCV antibody test. There was no significance, association between DM and hepatitis C virus (P=1. 000).

One hundred eighty nine (66.5%) of the study participants were using HAART for greater than five years. Of these, 22 (11.6%) of them had DM. On the other hand only 2 (2.1%) of the DM positive study participants had HAART duration of less than five years. There was a statistically significant association between DM and duration of HAART (P=0. 006).

Regarding to HIV duration, almost half (51.9%) of the study participants live with HIV for greater than or equal to five years. Of these, 19 (9.3%) of them had DM. There was a statistically significant association between DM and HIV duration (p=0. 013).

In relation to hypertension and dyslipidemia, 135(34.4%) and 126(32.1%) of the study participants had hypertension and dyslipidemia, respectively. Nineteen (14.1%) and 19(15.1%) of the study participants who had hypertension and dyslipidemia were DM positive, respectively. Six (2.3%) of the study participants with DM had normal systolic and diastolic values. There were statistically significant association of both hypertension (p=0. 001) and dyslipidemia (P=0. 001) with DM.

In the anthropometric measurement, 85(32.3%) and 75(28.5%) of the female study participants had an abnormal waist circumference and waist to hip ratio value respectively. On the other hand, 21(16.2%) and 38(29.2%) of the male study participants had an abnormal waist circumference and waist to hip ratio values respectively. One hundred twelve (28.5%) of the study participants had abnormal waist circumference and among them 12(10.7%) were DM positive. There was statistically significant association between DM and waist circumference (P=0. 026). In addition to this, 113(28.8%) of the study participants had abnormal waist to hip ratio. Of these, 5(4.4%) of them had DM. There was no statistically significant association between DM and waist to hip ratio (p=0. 318) (Table 2).

**Table 2. Clinical factors versus DM in HIV infected individuals (n=393) at JUSH comprehensive chronic care and training center, Southwest Ethiopia, 2014.**

| Variables | | Glucose result: no (%) | | Total, no(%) | P-value of $X^2$ test |
|---|---|---|---|---|---|
| | | Normal | DM | | |
| Sign and symptoms | No symptoms | 302(100) | 0 | 302(76.8) | |
| | All symptoms | 8(24.2) | 25(75.8) | 33(8.4) | |
| | Polydipsia or Polyuria or Polyphagia | 58(100) | 0 | 58(14.8) | |
| Family history of DM | Yes | 40(87) | 6(13) | 46(11.7) | 0.098* |
| | No | 328(94.5) | 19(5.5) | 347(88.3) | |
| Smoking | Never smoked | 304(93.3) | 22(6.7) | 326(83) | 0.783* |
| | Former smoker | 64(95.5) | 3(4.5) | 67(17) | |
| Types of HAART | AZT/3TC/EFV | 27(93.1) | 2(6.9) | 29(7.4) | |
| | TDF/3TC/NVP | 38(92.7) | 3(7.3) | 41(10.4) | |
| | AZT/3TC/NVP | 113(89.7) | 13(10.3) | 126(32.1) | |
| | TDF/3TC/EFV | 78(92.9) | 6(7.1) | 84(21.4) | |
| | ABC/DDI/LPV/r/TDF+DDI+LPV/r/DDI+3TC+LPV/r | 4(100) | 0 | 4(1) | |
| | HAART naive | 108(99.1) | 1(0.9) | 109(27.7) | |
| WHO clinical stage of HIV/AIDS | Stage 1 | 313(92.9) | 24(7.1) | 337(85.8) | |
| | stage 2 | 32(1000 | 0 | 32(8.1) | |
| | stage 3 | 21(100) | 0 | 21(5.3) | |
| | stage 4 | 2(66.7) | 1(33.3) | 3(0.8) | |
| HIV duration | <5 years | 183(96.8) | 6(3.2) | 189(48.1) | 0.013 |
| | ≥5 years | 185(90.7) | 19(9.3) | 204(51.9) | |
| Duration of HAART | < 5 years | 93(97.90 | 2(2.1) | 95(33.5) | 0.006 |
| | ≥5 years | 167(88.4) | 22(11.6) | 189(66.5) | |
| Hypertension | Yes | 116(85.9) | 19(14.1) | 135(34.4) | 0.001 |

| | | | | | |
|---|---|---|---|---|---|
| | No | 252(97.70 | 6(2.3) | 258(65.6) | |
| Dyslipidemia | Yes | 107(84.9) | 19(15.1) | 126(32.1) | 0.001 |
| | No | 261(97.8) | 6(2.2) | 267(67.9) | |
| BMI | <18.5 | 77(98.7) | 1(1.3) | 78(19.8) | |
| | 18.5-25 | 252(95.1) | 13(4.9) | 265(67.4) | |
| | 25.1-29.9 | 34(77.3) | 10(22.7) | 44(11.2) | |
| | ≥30 | 5(83.3) | 1(16.7) | 6(1.5) | |
| HCV antibody test | Positive | 5(100) | 0 | 5(1.3) | 1.000* |
| | Negative | 363(93.6) | 25(6.4) | 388(98.7) | |
| Waist circumference (M&F) | Normal | 268(95.4) | 13(4.6) | 281(71.5) | 0.026 |
| | Abnormal | 100(89.3) | 12(10.7) | 112(28.5) | |
| Waist to Hip Ratio (M&F) | Normal | 260(92.9) | 20(7.1) | 280(71.2) | 0.318 |
| | Abnormal | 108(95.6) | 5(4.4) | 113(28.8) | |
| Total cholesterol | <200 | 248(96.5) | 9(3.5) | 257(65.4) | 0.001 |
| | ≥200 | 120(88.2) | 16(11.8) | 136(34.6) | |
| Triglycerides | <150 | 128(96.2) | 5(3.8) | 133(33.8) | 0.131 |
| | ≥150 | 240(92.3) | 20(7.7) | 260(66.2) | |
| LDL-C | <130 | 231(97.9) | 5(2.1) | 236(60.1) | 0.001 |
| | ≥130 | 137(87.3) | 20(12.7) | 157(39.9) | |
| HDL-C | <40 | 328(92.9) | 25(7.1) | 353(89.8) | 0.093* |
| | ≥40 | 40(100) | 0 | 40(10.2) | |

Statistically significant at $P< 0.05$, M: male, F: female,*: P-value of Fisher's exact test

For variables that do not satisfy the assumptions of chi-square test, the Fisher's exact test were used instead of Pearson chi-square test to test the association between the dependent variable (DM) and the independent variables. Variables that are not satisfying the assumptions of chi-square test in both Pearson chi-square and Fisher's exact test such as: sign and symptoms, types of HAART, WHO clinical stage of HIV/AIDS and occupation had no computation for testing the association and thus no p-values were extracted.

**Table 3. Binary logistic regression analysis of variables considered to be associated with DM in HIV infected individuals (n=393) at JUSH comprehensive chronic care and training center, Southwest Ethiopia, 2014.**

| Variables | | Glucose result: no (%) | | COR | 95% CI | P-value |
|---|---|---|---|---|---|---|
| | | Normal | DM | | | |
| Age | <40 years | 270(96.4) | 10(3.6) | 1 | | 0.001 |
| | ≥40 years | 98(86.7) | 15(13.3) | 4.13 | 1.797-9.505 | |
| Sex | Male | 122(93.8) | 8(6.20) | 1 | | 0.906 |
| | Female | 246(93.5) | 17(6.5) | 1.05 | 0.442-2.510 | |
| Education status | Literate | 287(93.2) | 21(6.8) | 1.48 | 0.495-4.439 | 0.482 |
| | Illiterate | 81(95.3) | 4(4.7) | 1 | | |
| Marital status | Married | 233(95.1) | 12(4.9) | 1 | | 0.131 |
| | Single/widowed/Divorced | 135(91.2) | 13(8.8) | 1.87 | 0.829-4.215 | |
| Drug status | HAART experienced | 260(91.5) | 24(8.5) | 9.97 | 1.332-74.621 | 0.025 |
| | HAART naive | 108(99.1) | 1(0.9) | 1 | | |
| HIV duration | < 5 years | 183(96.8) | 6(3.2) | 1 | | 0.017 |
| | ≥5 years | 185(90.7) | 19(9.3) | 3.13 | 1.223-8.021 | |
| Duration of | ≤5 years | 93(97.90 | 2(2.1) | 1 | | 0.016 |

| | | | | | | |
|---|---|---|---|---|---|---|
| HAART | >5 years | 167(88.4) | 22(11.6) | 6.13 | 1.409-26.631 | |
| Hypertension | Yes | 116(85.9) | 19(14.1) | 6.88 | 2.677-17.678 | 0.001 |
| | No | 252(97.70 | 6(2.3) | 1 | | |
| Dyslipidemia | Yes | 107(84.9) | 19(15.1) | 7.72 | 3.002-19.87 | 0.001 |
| | No | 261(97.8) | 6(2.2) | 1 | | |
| Waist circumference (M&F) | Normal | 268(95.4) | 13(4.6) | 1 | | 0.030 |
| | Abnormal | 100(89.3) | 12(10.7) | 2.47 | 1.092-5.603 | |
| Waist to hip ratio | Abnormal | 108(95.6) | 5(4.4) | 1.66 | 0.608-4.541 | 0.322 |
| | Normal | 260(92.9) | 20(7.1) | 1 | | |
| Total cholesterol | <200 | 248(96.5) | 9(3.5) | 1 | | 0.003 |
| | ≥200 | 120(88.2) | 16(11.8) | 3.67 | 1.578-8.555 | |
| Triglycerides | <150 | 128(96.2) | 5(3.8) | 1 | | 0.139 |
| | ≥150 | 240(92.3) | 20(7.7) | 2.13 | 0.782-5.818 | |
| LDL-C | <130 | 231(97.9) | 5(2.1) | 1 | | 0.001 |
| | ≥130 | 137(87.3) | 20(12.7) | 6.75 | 2.475-18.380 | |

Statistically significant at *P*< 0.05, COR: crude odds ratio, CI: confidence interval, M: male, F: female

In the bivariate binary logistic regression analysis, variables such as: age (COR=4.13, 95%CI: 1.79-9.51, P=0.001), drug status(COR=9.97, 95%CI: 1.33-74.62, p=0.025), HIV duration (COR:3.13, 95%CI:1.22-8.02, P=0.017), duration of HAART (COR:6.13, 95%CI:1.41-26.6, P=0.016), hypertension (COR:6.88, 95%CI:2.68-17.68,P=0.001), dyslipidemia (COR=7.72,95%CI:3.00-19.87, P=0.001), waist circumference (COR=2.47, 95%CI:1.09-5.60, P=0.030), total cholesterol (COR:3.67, 95%CI:1.58-8.56, P=0.003) and LDL-c (COR=6.75, 95%CI:2.48-18.38, P=0.001) had statistically significantly association with DM.

Additionally, those variables which had a p-value less than 0.25 were candidate for multivariate logistic regression analysis (Table 3).

**Table 4. Multivariate logistic regression analysis of variables associated with DM in HIV infected individuals (n=393) at JUSH comprehensive chronic care and training center, Southwest Ethiopia, 2014.**

| Variables | | Glucose result, no (%) | | AOR | 95%CI | P-value |
|---|---|---|---|---|---|---|
| | | Normal | DM | | | |
| Age | <40 years | 270(96.4) | 10(3.6) | 1 | | |
| | ≥40 years | 98(86.7) | 15(13.3) | 2.98 | 1.042-8.512 | 0.042 |
| Duration of HAART | <5 years | 93(97.90 | 2(2.1) | 1 | | |
| | ≥5 years | 167(88.4) | 22(11.6) | 19.48 | 2.591-146.439 | 0.004 |
| Hypertension | Yes | 116(85.9) | 19(14.1) | 5.49 | 1.878-16.079 | 0.002 |
| | No | 252(97.70 | 6(2.3) | 1 | | |
| Dyslipidemia | Yes | 107(84.9) | 19(15.1) | 6.07 | 2.069-17.830 | 0.001 |
| | No | 261(97.8) | 6(2.2) | 1 | | |

Statistically significant at $P< 0.05$, AOR: adjusted odds ratio, CI: confidence interval

After adjusting of the other variables, age (AOR=2.98, 95%CI: 1.04-8.51, p=0.042), duration of HAART (AOR=19.48, 95%CI: 2.59-146.44, P=0.004), hypertension (AOR=5.49, 95%CI: 1.88-16.08, P=0.002) and dyslipidemia (AOR=6.07, 95%CI: 2.07-17.83, P=0.001) had strong statistically significant association with DM (Table 4).

# CHAPTER SIX: DISCUSSION

Ethiopia is one of the countries of the IDF Africa region. The national prevalence of diabetes mellitus was estimated 2-3% in the general population [50]. In addition to this, the total prevalence of diabetes mellitus in Ethiopian adult population (20-79 years) was 4.36% [51], which is lower than this study (6.4%). People with diabetes have an increased risk of developing a number of serious health problems. High blood glucose levels can lead to serious diseases affecting the heart, blood vessels, eyes, kidneys and nerves. On the other side, HIV/AIDS is not a curable and it is disastrous disease that compromises the immune system of the patient. ART drugs can also have impact on the cause of diabetes. So, the combination of the two diseases will increase the burden of morbidity and mortality of the HIV/AIDS patients. This could be the probable reason why the present study is having higher prevalence of DM than the general and the adult population of Ethiopia.

The prevalence of this study was lower than reports from Senegal, 14.5% [40], Tanzania, 24.7% [39], Thai, 27% [41], Israel, 22.9% [49], USA, California, 12% [36] and Romania, 13% [48]. On the other hand, the prevalence of diabetes mellitus in this study was comparable to the study conducted in Welayta soda, Ethiopia, 8% [19] and the prevalence of diabetes mellitus in this study was higher than the study conducted in Zambia, 2.7% [47]. In this study, higher prevalence of impaired fasting glucose was seen, which is inconsistent with a study done in Romania, 4.34% [48] and Zambia, 1.3% [47]. The observed difference in the prevalence of diabetes mellitus could be due to variation in sample size, study design, lifestyle, HAART regimens and age distribution of the HIV/AIDS patients.

In this study, most of the DM positive individuals had equal or greater than forty years of age. When age is increased the prevalence of diabetes mellitus also increase, which was consistent with the studies done in New York, USA [7], Welayta Sodo, Ethiopia [19], Texas, USA [29], USA [34], and Zambia [47].

The majority of the study participants and most of the diabetes mellitus positive individuals in this study were females (66.9%), which was consistent with the studies done in Tanzania, Muhabi national hospital, 56% [39], Welayta Sodo, Ethiopia 51% [19], southern India, 54% [38] and inconsistent with the studies done in Malaysia 18.9%[12] and China, 24.3% [37].

In this study (11.7%) of the study participants had a family history of diabetes mellitus, which is inconsistent with the study done in Texas USA, 60% [29]. Whereas, majority of the study participants (83%) in this study had no smoking habit in their life and there was no association between diabetes mellitus and smoking, which was consistent with the study done in Malaysia [12].

In this study, most of the study participants (99%)were using the combination of Nucleoside reverse transcriptase inhibitors (NRTIs) and Non-nucleoside reverse transcriptase inhibitors (NNRTIs) and types of HAART had a high risk of causing diabetes mellitus, which was consistent with the studies done in Malaysia, 90.7%[12], and inconsistent with the studies done in New York, USA 71%[35] and Texas, USA 77.9%[29], in which most of the study participants were used protease inhibitors (PIs). The observed difference could be due to variation in types and availability of HAART regimens.

Greater than half of the study participants(66.5%) in this study stay on HAART for a long time (≥5years), and most of the diabetes mellitus positive study participants 22(11.6%) had long duration of HAART, which is consistent with the studies done in Welayta Sodo, Ethiopia, 12.68% [19] and Senegal, 32.8% [40]. In addition to this, long duration of HAART is a potential risk factor for diabetes mellitus, which is consistent with the studies done in Welayta Sodo, Ethiopia 6.8% [19] and Senegal 32.8% [40]. The above observed difference could be due to variation in the types of HAART regimens.

In this study, 34% of the study participants showed hypertension, which was comparable to the study done in Senegal, 28.1% [40], higher than a study conducted in Ethiopia, 15.9% [19] and Malaysia, 19.7% [12] and lower than a study conducted in Tanzania, 48% [39]. Moreover, in this study, less than half of the study participants (32.1%) had dyslipidemia, which is inconsistent with the studies done in Malaysia, 82.3% [12], and Thai, 88% [41]. The observed difference

could be due to variation in sample size, study design, types of HAART, duration of HIV infection itself, stage of HIV infection, gender, lifestyle and age difference of the study participants.

Only 5 (1.3%) of the study participants in this study were positive for hepatitis C virus (HCV) antibody test and no diabetic case was having HCV leading to lack of association with diabetes mellitus, which is inconsistent with the studies done in Malaysia, 14.4% [12], Texas USA, 25% [29] and USA, 31.2% [34]. The above observed difference could be due to difference in the laboratory diagnosis method for HCV antibody test.

Whereas, most of the diabetes mellitus positive study participants in this study had higher total cholesterol, triglyceride, low density lipoprotein cholesterol and low level of high density lipoprotein cholesterol, which was consistent with the studies done in Malaysia, [12], Nigeria [25] and Australia [21]. The above similarity may be due to the similar risk factors of DM.

In this study, almost half of the diabetes mellitus positive individuals had abnormal waist circumference and showed significant association with diabetes mellitus in the bivariate binary logistic regression analyses.

Most of the diabetes mellitus positive study participants in this study were from urban and their occupation were merchant and employed because the study is conducted in urban area. Despite this, all of the diabetes mellitus positive study participants had the three signs and symptoms (polyphagia, polyuria and polydipsia) of DM. Finally, almost all of the study participants were in WHO clinical stage I and had normal BMI values. This could be because, most of the study participants in this study were HAART users and they would give care for themselves.

# CHAPTER SEVEN: CONCLUSION AND RECOMMENDATIONS

## 7.1 CONCLUSION

We conclude that, the magnitude of diabetes mellitus among adult HIV infected individuals, at comprehensive chronic care and training center of JUSH were higher than the national estimated prevalence of DM in Ethiopia. Age, duration of HAART, hypertension and dyslipidemia had a strong significant association with diabetes mellitus.

## 7.2 RECOMMENDATIONS

It is necessary to routinely screen HIV/AIDS patients who are using HAART for diabetes mellitus. All newly diagnosed HIV/AIDS patients should be routinely screened for diabetes mellitus, both before and after initiating of HIV treatment. All the adult HIV/AIDS patients should be routinely checked for hypertension, and lipid profile tests must be routinely done for screening of dyslipidemia. Moreover, based on our results; hypertension and dyslipidemia were the potential risk factor for the development of diabetes mellitus in HIV/AIDS patients. So, the health professionals who are working in the comprehensive chronic care and training center should make awareness to their HIV/AIDS patients about the prevention, diagnosis and treatment of hypertension, dyslipidemia and diabetes mellitus to prevent further complication.

Further research is needed to discover the impact of HAART (the new WHO recommended) usage and its duration on the development of diabetes mellitus in HIV/AIDS patients with large sample size and with longitudinal study.

# ANNEXES

## ANNEX I: REFERENCE


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# ANNEX II. LABORATORY PROCEDURE

## VENOUS BLOOD COLLECTION AND PROCESSING

1. A sterile, dry, preferably plastic syringe of the capacity required 5 ml were used.
2. A soft tubing tourniquet to the upper arm of the patient was applied and the tourniquet not applied for no longer than 2 minutes.
3. Using the index finger feel for a suitable vein, selecting a sufficiently large straight vein that does not roll and with a direction that can be felt.
4. The puncture site cleanses with 70% ethanol and allows to drying and the cleansed area is not re-touched.
5. With the thumb of the left hand holding down the skin below the puncture site, make the vein puncture with the bevel of the needle directed upwards in the line of the vein. Steadily withdraw the plunger of the syringe at the speed it is taking the vein to fill. Avoid moving the needle in the vein.
6. When sufficient blood has been collected, release the tourniquet and instruct the patient to open his or her fist. Remove the needle and immediately press on the puncture site with a piece of dry cotton wool. Remove the tourniquet completely. Instruct the patient to continue pressing on the puncture site until the bleeding has stopped.
7. Remove the needle from the syringe and carefully fill the container(s) with the required volume of blood. Discard the needle safely.
8. Check that bleeding from the venipuncture site has stopped. Cover the area with a small dressing.
9. Centrifuge at 2000 RPM for five minutes, separate the serum and analyze the sample in HumaStar 80(Germany) spectrophotometer( a machine can perform 20 different tests once and 80 sample/hour) for total cholesterol, HDL-cholesterol and triglycerides. Despite this, the glucose tests were analyzed by Dr.Lang 80 spectrophotometer at Biochemistry laboratory, Jimma University. Also, from the serum HCV antibody test was done.

**ANTI-HCV RAPID TEST**

Test device, patient samples and control should be brought to room temperature (15-30$^0$C) prior to testing. Do not open pouches until ready to perform the assay.

1. Remove the test strip from its protective pouch and label the strip with patient or specimen number.
2. Use the dropper or a pipette to withdraw specimen from the specimen collection container and add 1 drop (about 25 micro liters) on to the sample pad. Then add 2 drops (100 micro liters) of assay buffer as well.
3. Start the timer and wait for the red line(s). The result should be read at 10-20 minutes. Result read after 20 minutes is invalid.

   Interpretation of result

   **Negative result**: if only the control line is developed, the test indicates that no detectable antibodies to HCV present in the specimen. The result is negative.

   **Positive result**: if both control and test lines are developed, the test indicates for the presence of antibodies to HCV in the specimen. The result is positive.

   **Invalid:** if no control line is developed, the assay is invalid regardless of color development on the T line**.**

**GLUCOSE TEST (GOD-PAP METHOD)**

After at least 10 hours of overnight 5 ml fasting venous blood sample was drawn by senior medical laboratory technologist using serum separator vacuum tubes with clotting enhancers**,** from consent subjects after adequate disinfection of the area. Then the specimen was centrifuged and the serum was separated for analysis within 30 minutes. 1000 micro liter of the reagent and 10 micro liter of the serum was mixed and incubated for 5 minute at 37 $^{O}$C and the test was performed with Dr.Lang 80 spectrophotometer.

**Contents**

RGT 4X100 ML

Phosphate buffer

4-aminophenazone

Phenol

Glucose oxidase

Peroxidase

Mutarotase

Stabilizers

STD 3ml standard

Glucose 100mg/dl

**Reagent preparation**

RGT and STD are ready for use.

**Reagent Stability**

The reagents are stable up to the given expiry date when stored at 2-8$^0$ c.

When opened contamination must be avoided. RGT is stable for 2 weeks at 15-25 $^0$c

**Specimen**

Whole blood, serum, plasma

The glucose is stable for 5 days at 20-25$^0$ c, if deproteinisation and centrifugation of the whole blood is performed promptly after collection.

The glucose is stable for 24 hours at 2-8 $^0$c, if serum or plasma is prepared with in 30 minute after collection.

**Assay**

Wavelength: Hg 546 nm

Optical path: 1 cm

Temperature: 20-25 $^0$c or 37 $^0$c

Measurement: Against reagent blank, only one reagent blank per series is required.

**Performance characteristics**

Linearity

The test is linear up to glucose concentration of 700mg/dl.

Normal values

Serum, plasma (fasting): 70-110mg/dl

## TOTAL CHOLESTEROL (CHOD-PAP METHOD)

**Contents**

RGT 4x100 ml reagent

Phosphate buffer (PH 6.5)

4-Aminophenazoane

Phenol

Peroxidase

Cholestrolesterase

Cholestroloxidase

Sodium azide

STD 3ml standard

Cholesterol 200mg/dl

**Reagent preparation**

The RGT and STD are ready for use.

**Reagent stability**

The reagents are stable up to the given expiry date, even after opening, when stored at 2-8$^0$C. The open reagent is stable for 2 weeks at 15-25$^0$C. Contamination must be avoided.

**Specimen**

Serum, heparinized or EDTA plasma

**Assay**

Wavelength: 500 nm, Hg 546 nm

Optical path: 1 cm

Temperature: 20-25 $^0$c or 37 $^0$c

Measurement: Against reagent blank, only one reagent blank per series is required.

**Performance characteristics**

**Linearity**

The test is linear up to a cholesterol concentration of 750 mg/dl. Dilute samples with a higher cholesterol concentration 1+2 with physiological saline (0.9%) and repeat the determination. multiply the result by 3.

**Clinical interpretation**

Suspect over 220 mg/dl

Elevated over 260 mg/dl

**TRIGLYCERIDES (**GPO-PAP METHOD)

**Contents**

RGT 250 ML monoreagent

PIPES Buffer (PH 7.5)

4-Chlorophenol

4-aminoantipyrine

Magnesium ions

ATP

Lipases

Peroxidase

Glycerol kinase

Glycerol-3-phosphate oxidase

STD 3 ml standard

Triglycerides 200 mg/dl

**Reagent preparation and stability**

The RGT and STD are ready for use.

The reagents are stable up to the given expiry date, even after opening, when stored at 2-8$^0$C. At 20-25$^0$C the reagent is stable for 4 weeks. Contamination must be avoided. Protect from light.

**Specimen**

Serum, heparinized plasma or EDTA plasma

Stability: 3 days at 2-8$^0$C

4 months at -20$^0$C

NOTE: lipemic specimens usually generate turbidity of the sample reagent mixture which leads to falsely elevated results. The TRIGLYCERIDES LIQICOLOR $^{mono}$ test avoids these falsely elevated results through its built-in Lipid-Clearing Factor (LCF). The LCF clears up totally a turbidity caused by lipemic specimens.

**Assay**

Wavelength: 500 nm, Hg 546 nm

Optical path: 1 cm

Temperature: 20…25 $^0$c or 37 $^0$c

Measurement: Against reagent blank, only one reagent blank per series is required.

**Performance characteristics**

Linearity

The test is linear up to a triglyceride concentration of 1000 mg/dl. Samples with a higher concentration have to be diluted 1+ 4 with physiological saline (0.9%) and retested. Multiply the result by 5.

**Clinical interpretation**

Suspect: over 150 mg/dl

Increased: over 200 mg/dl

**HDL CHOLESTEROL**

Contents

ENZ 1x60 ml enzyme (white cap)

Good's buffer PH 7.0(20$^0$C)

Cholesterol esterase

Cholesterol oxidase

Catalase

N-(2-Hydroxy-3-sulfopropyl)-3, 5-dimethoxyaniline (HDAOS)

SUB 1X20 ml substrate (green cap)

Peroxidase

4-aminoantipyrine (4-AA)

Good's buffer PH 7.0(20$^0$C)

Sodium azide

Detergents

CAL 1x4 ml calibrator

Cholesterol concentration see vial label

**Reagent preparation and stability**

ENZ and SUB are ready for use.

Stability: after opening the reagent are stable up to 1 month when stored at 2-8$^0$C. Avoid contamination. Do not freeze. Do not mix caps.

CAL: reconstitute the content of the vial with exactly 4 ml distilled germ free water, close the vial and swirl carefully to dissolve all lyophilisate. Avoid foaming. Let stand for 30 minutes before use.

Stability: 10 days at 2-8$^o$C. If required, freshly prepared calibrator can be divided in to portions and kept frozen at -20$^0$C for maximum 30 days. Freeze and thaw only once, mix carefully after thawing.

**Specimen**

Serum, plasma

Stability: we recommend to test directly after sampling, otherwise store the serum at -20$^0$C (up to several weeks; avoid repeated freezing and thawing).

If plasma following concentration of the anticoagulant should not be exceeded: EDTA-2Na<200 mg/dl; Na-citrate <1000 mg/dl; heparin <50 mg/dl; NaF <2000 mg/dl.

**Assay**

Wavelength: 578 nm, Hg 593 nm, (570 to 610 nm)

Optical path: 1 cm

Temperature: 37 $^0$c

Measurement: Against reagent blank, only one reagent blank per series is required.

**Performance characteristics**

Linearity: up to 150 mg/dl HDL

Linearity concentration depends on the analyzer-specific application. If the serum concentration of HDL exceeds the measuring range, dilute the sample 1+ 1 with saline (0.9%); repeat the test and multiply the result by 2.

**Reference values**

<35 mg/dl risk factor for CHD

>60 mg/dl reduced risk for CHD

## ANNEX III: EQUIPMENTS, MATERIALS AND REAGENTS

- ✓ Vacuum test tubes
- ✓ Surgical Gloves
- ✓ Glucose reagent
- ✓ Total cholesterol reagent
- ✓ HDL-cholesterol reagent
- ✓ Triglyceride reagent
- ✓ HCV reagent strip test
- ✓ 70% alcohol
- ✓ Cotton
- ✓ Measuring tape
- ✓ Paper
- ✓ Permanent marker
- ✓ Pen
- ✓ Pencil
- ✓ Flash disk
- ✓ Syringe
- ✓ HumaStar 80 spectrophotometer
- ✓ Dr. Lang 80 spectrophotometer
- ✓ Centrifuge
- ✓ Calibrator
- ✓ Cleaner
- ✓ Sample cup

## ANNEX IV. CONSENT FORM

Consent form to study the magnitude of Diabetes mellitus and associated factors among HIV infected individuals at JUSH, comprehensive chronic care and training center, Southwest Ethiopia

(≥18 years of age)

**Principal investigator**: Abdurehman Eshete Mohammed

**Organization**: Jimma University

**Sponsor**: Jimma University

**Project Title**: Diabetes mellitus and associated factors among HIV infected individuals at JUSH, comprehensive chronic care and training center, Southwest Ethiopia.

This informed consent has two parts

- Informed sheet, and
- Certificate of Consent

*Read and give a copy of the full informed consent form to the respondent.*

**Part I – Statement**

**Introduction:**

Hello! My name is _____and am working with researchers from Jimma University .We are doing a study to assess the magnitude of Diabetes mellitus and associated factors of HIV/AIDS positive individuals at JUSH, Southwest Ethiopia

**Purpose and length of the study**

The prevalence of Diabetes mellitus is not well known in Ethiopia in HIV/AIDS positive individuals. It has not been the focus of intense study nor of active control programs. This neglect is likely a function of the relatively new onset of diabetes mellitus in HIV/AIDS infected individuals.

This study will designed to provide information on the magnitude of Diabetes mellitus and its relationship with socio-demographic / epidemiological factors, biological factors for the effective planning of control programs geared towards reducing the risks associated with its co infection among HAART naïve and HAART experienced HIV/AIDS positive individuals at JUSH, totally

in Ethiopia and it was the base line for other study for the co infection of diabetes mellitus and HIV/AIDS. The study will take about two months.

Procedures

We invite you to take part in this study. If you are willing, you will required to provide fasting venous blood specimen, weight, height, waist and hip measurement. You will then visit the principal investigator on the second day to be advised about your test results.

**Risks and Discomfort**

There is no discomfort associated with this study.

**Benefits**

Any finding from your blood specimen was taken care of by our study doctor.

**Incentives**

We will not pay you for taking part in this study. However, we will thank you for your participation.

**Confidentiality**

The data that we will collect from venous blood, weight, height, hip and waist was kept confidential and don't contain any information that may lead to your identity. The patient name will not be recorded, rather coding was implemented, and test result will only be known by the principal investigator to keep confidentiality of test result.

**Right to refuse or withdraw**

You do not have to take part in this research if you do not wish to do so, and refusing to participate will not have any effect in your treatment**.** You do have full right to withdraw from this study at any point if you wish. Your withdrawal will have no influence what so ever on your further treatment.

**Whom to contact**

If you have any questions you may ask the nurse/doctor now or later. If you wish to ask questions later, you may contact Abdurehman Eshete (0934580372), aeshete8@gmail.com.

**Part II – Informed consent**

I have been requested to give my venous blood, weight, height, hip and waist measurements for diagnosis of Diabetes mellitus and its associated factors and to take part in the research. The information in part I have been read to me. I have had the opportunity to ask questions about it

and any questions I have asked have been answered to my satisfaction. I consent voluntarily to give venous blood, weight, height, hip and waist measurements for Diabetes mellitus and its associated factors diagnosis.

*Print name of subject, date and signature or thumb impression of subject*

_____ ____/____/____ (dd/mm/yy)

**If illiterate:** Print name of independent literate witness, date and signature of witness (if possible, this person should be selected by the participant and should have no connection to the research team) _____, ____/____/____ (dd/mm/yy)

Print name of researcher, date and signature of researcher

Abdurehman Eshete_____, ____/____/____ (dd/mm/yy)

**Annex-IIIB Amharic Version**

**ለጥናቱ መረጃና ተሳታፊነት መግለጫ ቅጽ**

ተመራማሪ አብዱረህማን እሸቴ

ተቋም ጅማ ዩኒቨርሲቲ

እስፖንሰር ጅማ ዩኒቨርሲቲ

**የጥናቱ ርዕስ** "የሰኳረ በሽታ ና ተያያዥ መንስኤዎች በ HIV/AIDS በሽተኞች ላይ ያለው ክስተት "

ክፍል 1.

መግቢያ

ከጅማ ዩኒቨርሲቲ

ጤ ና ይስጥልኝ፡ እኔ _____ እባላለሁ፡፡ ይህንን ጥናት የምንሰራው "የሰኳረ በሽታ ና ተያያዥ መንስኤዎች በ HIV/AIDS በሽተኞች ላይ ያለው ክስተት ለማወቅ ነዉ፡፡

**የጥናቱ ዓላማና የሚፈጀዉ ጊዜ**

"የሰኳረ በሽታ ና ተያያዥ መንስኤዎች በ HIV/AIDS በሽተኞች ላይ ያለው ክስተት ለማወቅ ሲሆን ይህ ጥናት ሁለት ወራትነን ይፈጃል፡፡

**የአሰራሩ ሂደት**

በዚህ ጥናት ዉስጥ ይሳተፉ ዘንድ እንጠይቆታለን፡፡ለመሳተፍ ደም ናሙና የወገብ ልክ አና የዳሌ ልክ መስጠት ይጠበቅቦታለ፡፡

**በጥናቱ ስለመሳተፍ**

በዚህ ጥናት መሳተፍ በሙሉ ፈቃደኝነት የተመሰረተ ነው፡፡ ስለሆነም በጥናቱ እንዲሳተፉ ፈቃደኛነትዎን እንጠይቃለን፡፡ ለመሳተፍ ከፈቀዱ የደም ናሙና ተወስዶ የላብራቶሩ ምርመራ ያደርግልዎታል፡፡ የደም ናሙናው የሚወሰደው ንፅህናው በተጠበቀ መሳሪያ ነው።

**በጥናቱ ሊከሰቱ የሚችሉ ተያያዥ ችግሮች**

ምንም አይነት **ችግር አያስከትልም፡፡**

**በጥናቱ በመሳተፍ የሚ ገኝ ጥቅም**

የደም ናሙናው ውጤት የስኳር በሽታ የሚያሳይ ከሆነ መዲሃኒት አንዳገኙ ወደ ስኳር ህክምና ክሊኒክ አንዲሄዱ ይደርጋል።**የጥናቱ መረጃዎች ሚስጥራዊነት**

በጥናቱ ውስጥ የተሰበሰቡ ማናቸውም ግላዊ መረጃዎች ሚስጥራዊነታቸው የተጠበቀ ይሆናል፡፡ ከማንነትዎ ጋር በቀጥታ ተያያዥነት ያላቸው መረጃዎች በሙሉ በዋና ተመራማሪው ሚስጥራዊ በሆነ የመረጃ ጥንቅር ዘዴ ከተቀየሩ በኋላ ብቻ ለምርምር ሂደቱ የሚውሉ ይሆናሉ፡፡

**የጥናቱን ውጤት ስለማሳወቅ**

የዚህ ጥናት ውጤት በተለያዩ የህትመት ውጤቶች የሚቀርብ ሲሆን ይህ ከማንነትዎ ጋር የተያያዘ ምንም አይነት መረጃን አያካትትም፡፡ ስለዚህም የጥናቱን ውጤት በሪፖርት እናቀርበው ዘንድም ፈቃድዎን እንጠይቃለን፡፡

**ከጥናቱ ስለመውጣትና ስለማቋረጥ**

ይህ ጥናት በፈቃደኝነት ላይ የተመሰረተ እንደመሆኑ መጠን በማንኛውም ወቅት በፈቃድዎ ከጥናቱ መውጣት ይችላሉ፡፡ ከጥናቱ ቢወጡም እንኳን የተለመደውን የህክምና እርዳታ በጤና ተቋሙ ውስጥ በማንኛውም ጊዜ የማግኘት መብት አለዎት፡፡

**ከጥናቱ ጋር በተያያዘ ማናቸውም ጥያቄ ቢኖርዎት በሚከተው አድራሻ ጥያቄዎን ማቅረብ ይችላሉ፡-**

አብዱረህማን እሸቴ

አድራሻ፡- ጂማ ዮንቨርስቲ ህክምና ላቦራቶሪ • አና ፓቶሎጂ ት/ት ክፍል

ጂማ

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ፓ.ሳ.ቁ. 409

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**<u>ስለስምምነቱ ማረጋገጫ ፊርማዎች /አዋቂዎች/</u>**

እኔ ስሜ ከታች የተገለፀው በዚህ ጥናት ተሳታፊ ለመሆን ስወሰን የጥናቱን አላማዎች አሰራሮችና ቅድመ ሁኔታዎች በግልጽ በመረዳትና እንዲሁም ከጥናቱ ተሳታፊነት ፈቃደኛነቴን በማንኛውም ደረጃ የማስወገድ መብቴን በማረጋገጥ ነው::

እኔ ---------- በጥናቱ ተሳታፊ መሆኔን በፊርማዬ እያረጋገጥሁ ይህንን ስወስን በጥናቱ ሳቢያ ሊከሰቱ የሚችሉ አደጋዎች በሚገባ የተረዳሁና ከጥናቱ በማንኛውም ደረጃ እራሴን ለመሰረዝ ብወስን ተገቢ የሆኑ ህክምናዎችና እገዛዎች ሁሉ እንደማይነፍጉኝ በማመን ነው:: እነዚህ መረጃዎች ሁሉ በሚገባ በምረዳው ቋንቋ የተገለጹልኝ መሆኑን በፊርማዬ አረጋግጣለሁ::

የበሽተኛው ሙሉ ስም ---------- ፊርማ ----------

የተመራማሪው ሙሉ ስም፣ ----------  ፊርማ ----------

የምስክር ሙሉ ስም ---------- ፊርማ ---------- ቀን ----------

**Booca 3C (Annex-III C Afaan Oromoo Version): Booca 2):**

Billeettaa qo'annaa hangaa"Diabetes mellitue" fi saaxila-baasota issaa kan dhibamtoota HIV/AIDS, kibaalixa Itiyophiya kessetti.

Itti gaafatama qorannoo kana – Abduramaan Isheete

---

Dhabhata qorachiisu: Yuniversitii Jimmaa

Dhabhata qorannoo kanaaf arjoomu- Yuniversitii Jimmaa

**Billeettaan kun kutaa lama of kessa qabo.**

1. Waa'ee dhimma kana ilaalchisee ragaa kan kennu.
2. Hirmatootin Waliigaltee kanitti mallatteessan (Billeettaa Waliigaltee)

Waligalttee kana hirmatootaf dhiyaate egaa dubifame (ibsii itti kennamee) boda akka mallatesaan tasisii.

**Kutaa A Ibsa**:-

Akkam jirtuu, Maqaan Koo _____ Jedhama. Bakkan hojeedhu oggessota qorannoofi qo'annoo Yuniversitii Jimmaatti. Yeroo ammaa yuniversitiin Jimma qo'anna hangaa DM fi saaxila –basoo isaa ka dhibamtoota HIV/AIDS JUSH , kibba-lixa Itiyophiyaa.

---

**Kaayyoo fi dheeerina Qorannicha**

Faffaca'insi 'DM' ykn dhibeen sukkaraa dhibamtoota HIV/AIDSii Itiyoophiyaa keessatti hinbeekamu ture. Xiyyeffanoon qo'annafi to'annon hindeemsifamnee.Kunimmoo babalina 'DM' dhibamtota HIV/AIDS ttif sababa ta'eera.

Qo'annan kunis oddeeffannoo 'DM' fi walitti dhufeenyi hawasumma fi faffaca'inssi to'anna dhibeekana irrati fidu, akkasumas dhibamtoota HIV/AIDS JUSH keessa fi , walumagala Itiyoophiyaa keesaaf bu'uura ta'a. Qo'annichis ji'oota sadiif geggeefama.

**Adeemsaa Hojiichaa**

Qoranno kana irratti akka hirmaattanu isin gaafatna. Fedhii keessan yoota'e sooman dhigga keesan ulfaatina, hojjaaa, bal'ina qaamakessa safaruuf.Akkasumas beellama docktorrii isnii laatuun dhuftanii gorsa fudhatani ilaalamuun barbaachisaadha.

**Sodaa YKN Shakkii Hirisuuf**

Yemuu kun hundi ta'u ,ogeessan waan ta'eef kan isin hubu miti.

**Qorannoo irratti hirmachuun fayyidda inni qabu**

Bu'aan qorannoo laboratorii dhangala'a qaama hormaataa kana irraa argamu qorannoo doktoraa keenyaan ni'ilaalama. Halli fayyaa keessan baroo yoo hin fakkaatne doktora keenya beellamaan alallee argachuu nidandeessan.

**Onnachiistuu**

Qo'anno kana irratti hirmmachuu kessaniif galatoma isiinin jechuun alaa qarshii isniii kaffallu hinjiru.

**Icciitummaa lsaa**

Qorannoo qo'anna kana irraa ragaan argamu iciitummanisaa akka egamuu ni taasifama. Itti dabalees mallatoon gara maqaa keessaniitti geessu danda'u hundi akkanni hinjiraanne nigodhama.

**Mirga Hirmaachuu, Hirmaachuu dhisuu fi hordooffii kutuu**

Qoroannoo irraatti hirmaachuuf fedhii yoo hinqabane dhigaa fi safaraallee keessan akka kennitaanu hin dirqaamtaanu. Itti dabalaatanis hirmaachuu dhissu kessaniin kan ka'e yaalama argaachuu irraatis ta'e faayyidaa addaa argaachuu qabdanuu irraatti rakkinni ykn dhiibbaan isiini irra gahu tokkoollee hin jiru. Akkasummas yeroo kamiyuu hordoofi dhiisuu ni dandeessuu.

**Qaama wajjiin walqunnamtti tasiftaan(arguu dandeessanu)**

Hojii kamiyuu ilaachisee ammas ta'e fuula durratti gaafi qabdan narsii ykn doktora isin ilaaletti dhiyyessu ni dandeessu. Booda gaaffii yoo qabaattan Abdurahiman Isheete (0912056717) qunnamuu nidandeessu.

**Kutaa B- billeettaa waligalaa**

Qorannoo hongaa 'DM' ykn dhibee sukkarafi dhiga fi kannen armaan olitti caqasaman akkan kennuu waamichii nagodhamera. Kutaa tokkoffaa keessatti dhimmi (barreefamni) dhiyatee naaf dubifameera. Gaaffis akkan gaafadhu carraan naaf latamee gaaffii koo hundaafis deebii quubsaa argadheera.Fedhiikootiin walii galee jedhee maqaa kootiin ni malateessa.

Maqaa qoratamaa_____Mallattoo_____ Guyyaa_____

Maqaa qorataa_____Mallatoo_____ Guyyaa_____

**Yoo kan barreessuu hindandeenye ta'e**

Maqaa wabii_____ Mallatoo_____ Guyyaa_____

## ANNEX V -QUESTIONNAIRE

Jimma University

College of public health & medical sciences

Department of medical laboratory science & pathology

The questionnaire is designed to collect socio-demographic and some clinical information about diabetes mellitus and associated factors among HIV infected individuals at JUSH, comprehensive chronic care and training center.

| Ser. no | Question to be asked | Proposed response | Coded response |
|---|---|---|---|
| 1. | Age | | |
| 2. | Sex | 1. Male<br>2. Female | |
| 3. | Present address | 1. Urban<br>2. Rural | |
| 4. | Occupation | 1. Farmer<br>2. Merchant<br>3. Employed<br>4. Unemployed<br>5. Others | |
| 5. | Educational Status | 1. Literate<br>2. Illiterate | |
| 6. | If literate, what educational level | 1. Read and write only<br>2. 1-4 grade<br>3. 5-8 grade<br>4. 9-12 grade<br>5. Above 12 | |
| 7. | Ethnicity | 1. Oromo<br>2. Amhara<br>3. Tigrie<br>4. Gurage<br>5. Dawuro<br>6. Yem<br>7. Others | |
| 8. | Religion | 1. Orthodox<br>2. Muslim<br>3. Catholic<br>4. Protestant<br>5. Others | |

| | | | |
|---|---|---|---|
| 9. | Marital status | 1. Married<br>2. Single<br>3. Widowed<br>4. Divorced | |
| 10. | Sign and symptoms | 1. Thirst<br>2. Polyphagia<br>3. Polyuria | |
| 11. | Family history of DM | 1. Yes<br>2. No | |
| 12. | Smoking | 1. Never smoked<br>2. Former smoker<br>3. Current smoker | |
| 13. | Weight in kg | | |
| 14. | Height in cm | | |
| 15. | Waist in cm | | |
| 16. | Hip in cm | | |
| 17. | Waist to hip ratio | | |
| 18. | BMI | | |
| 19. | Type of HAART | | |
| 20. | Duration of HAART in years | | |
| 21. | Systolic BP (mmHg) | | |
| 22. | Diastolic BP (mmHg) | | |
| 23. | Hypertension | 1. Yes<br>2. No | |
| 24. | HIV duration (in years) | | |
| 25. | Do you know your DM status before you know your HIV status | 1. Yes<br>2. No | |
| 26. | WHO clinical staging of HIV/AIDS | 1. Clinical stage 1<br>2. Clinical stage 2<br>3. Clinical stage 3<br>4. Clinical stage 4 | |
| **Laboratory analysis result** | | | |
| 27. | Glucose test | mg/dl | |
| 28. | Total cholesterol test | mg/dl | |
| 29. | HDL-cholesterol test | mg/dl | |
| 30. | LDL-cholesterol test | mg/dl | |
| 31. | Triglycerides test | mg/dl | |
| 32. | HCV antibody strip test | 1. Positive<br>2. Negative | |

## ANNEX VI: DECLARATION

I, the undersigned , declare that this research paper is my original work and has not been presented for degree in this or any other university and all source of materials used for this thesis has been fully acknowledge.

Name of the student: **Abdurehman Eshete Mohammed**

Signature _____

Name of the institution: Jimma University

Date _____

**APPROVAL OF THE FIRST ADVISOR**

This paper has been submitted with my approval as university advisor.

Name of the first advisor: **Mr. Waqtola Cheneke**

Name of institution: Jimma University

Date_____ Signature _____

**APPROVAL OF THE SECOND ADVISOR**

This paper has been submitted with my approval as university advisor.

Name of the second advisor: **Dr.Tilahun Yemane**

Name of institution: Jimma University

Date_____ Signature _____

This paper has been submitted with my approval as internal examiner

**Name of the internal examiner: Mr. Wondimagegn Addisu**

Name of institution: Jimma University

Date_____ Signature _____