PREVALENCE OF METABOLIC SYNDROME AMONG INMATES ON HAART AT EMBU PRISON DISPENSARY, COMPREHENSIVE CARE CLINIC.

LEWIS MUENDO MUTINDA

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DECLARATION

I, Lewis Muendo Mutinda, declares that this research proposal is my original work and has not been obtained from any other source other than the sources sighted.

Signature .....Date 25/01/2021

This research proposal is submitted with the approval from my supervisors:

Mr. F W Maina, Bsc, Msc

Lecturer, Thematic Unit Clinical Chemistry
Department of Human Pathology,
School of Medicine, College of Health Sciences,
University of Nairobi.

Signature .....Maina..... Date 20/02/21

Dr. G O Wandolo, M.B Ch.B,Msc
Lecturer; Thematic Unit Clinical Chemistry
Department of Human Pathology,
School of Medicine, College of Health Sciences,
University of Nairobi.

Signature..... Date 20/02/21

DEFINITION

ART	Antiretroviral Therapy
ARV	Antiretroviral
ATP III	Adult Treatment Panel III
BMI	Body mass index
CCC	Comprehensive Care Clinic
CVD	Cardiovascular disease
DM	Diabetes mellitus
E.R.C	Ethical review committee
HAART	Highly Active Antiretroviral Therapy
$HbA1_C$	Glycated hemoglobin
HDL	High density lipoprotein
HIV	Human Immunodeficiency Virus
IDF	International Diabetes Federation
KNH	Kenyatta National Hospital
LDL - c	Low density lipoprotein
Lp(a)	Lipoprotein (a)
LRP	Lipoprotein receptor – related protein
MS	Metabolic Syndrome
NCEP/ATP II	National Cholesterol Education Program
ATP	Adult Treatment Panel
NRTIs	Nucleoside/nucleotide reverse transcriptase inhibitors

PI	Protease inhibitor
TC	Total cholesterol
TGs	Triglycerides
UoN	University of Nairobi
VLDL - c	Very low density lipoprotein – cholesterol
W.H.O	World Health Organization

Drug regiments: AF2B – (TDF/3TC/EFV)

TDF – Tenofovir Disoproxil Fumarate

3TC - Lamivudine

EFV - Efavirenz

AF2E – (TDF/3TC/DTG)

DTG – Dolutegravir

FBG – Fasting plasma glucose

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ABSTRACT

I. Introduction

Metabolic syndrome (MS) is a combination of interrelated risk factors of metabolic origin; the components include increased insulin resistance, obesity, dyslipidemia and hypertension. WHO defines HAART (highly active antiretroviral therapy) as a form of antiretroviral therapy for the treatment of persons infected with HIV. The use of HAART has decreased morbidity and mortality. The use of protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (NTRIs) have been associated to development of lipodystrophy and metabolic abnormalities. In spite of the success of HAART, the prevalence of dyslipidemia, insulin resistance and fat redistribution have increased after its global step up. The prevalence of MS among HIV – infected patients globally ranges from 17.0% to 45.4%, with most reports produced in developed nations. The diagnosis of MS using the International Diabetes Federation is a rise in triglycerides greater than 1.7mMol/l, elevated fasting blood sugar greater than 5.6mMol/l and reduced HDL cholesterol less than 1.03mMol/l. There exists no data on the prevalence of MS among the prison population in Kenya.

II. Study objectives

The broad objective is to determine the prevalence of metabolic syndrome in inmates on HAART at Embu GK prison dispensary, comprehensive care clinic (CCC).

The specific objectives were:

i. To determine the serum concentration of fasting blood sugar, TC, LDL-C, HDL-C and triglyceride,
ii. To determine the BMI and blood pressure among patients on HAART
iii. To compare the different types of regimens to metabolic syndrome
iv. To determine the prevalence of MS in prisoners on HAART

III. Study design

This was a cross-sectional study aimed at establishing the prevalence of MS in patients on HAART at Embu GK prison Dispensary.

IV. Study area.

The study was conducted at Embu Prison Dispensary while the sample analysis was done at the Embu County Referral and Teaching Hospital, clinical chemistry laboratory.

V. Study population

The study population was the inmates at the Embu Prison who are on HAART for at least six months and attending the Prison Dispensary CCC clinic. From this population a sample size of 217 was obtained.

VI. Research methodology

This was a hospital based descriptive cross-sectional study aimed at establishing the prevalence of metabolic syndrome among patients on HAART for at least six months and attending Embu prison Dispensary. The study employed consecutive sampling method on the inmates attending the CCC clinic until the sample size was reached. Blood samples was collected and analyzed at the Embu teaching and referral hospital laboratory, biochemistry department. Research results were disseminated to department of human pathology UoN, KNH to be published in a reputable peer reviewed journal.

VII. Ethical consideration

Ethical clearance was sought from UoN/KNH ethical review committee. Research results were disseminated to department of human pathology UoN, to be published in a reputable peer reviewed journal.

VIII. Data analysis.

Data was collected and stored in file maker software and later analyzed using statistical tools which include SPSS version 20

CHAPTER ONE

INTRODUCTION

Metabolic syndrome (MS) is an accumulation of metabolic abnormalities and central obesity that present an increased risk of cardiovascular disease and type 2 diabetes mellitus, (Jericó et al., 2005). It consists of a group of metabolic risk factors which include but not limited to high blood pressure, impaired carbohydrate metabolism, dyslipidemia and obesity (Osei, 2010). HIV infected patients are put on highly active antiretroviral therapy (HAART)/ antiretroviral therapy (ART)so as extend their life and also reduce the viral load thereby increasing their health status. There is increased reports of high levels of lipodystrophy, obesity and hypercholesterolemia among patients on ART (Kiama et al., 2018).

Despite the success of HAART, the prevalence of diabetes mellitus (DM), fat redistribution, blood pressure and mainly dyslipidemia have considerably increased after its global scaling up (Barbaro G, 2006). The inclusion of protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (NTRIs) has been linked to development of lipodystrophy and metabolic abnormalities. The increased rates of MS in the HIV infected population has placed it in a high risk cardiovascular disease (CVD) category turning the MS into an important public health concern (Paula et al, 2013). HIV patients who have MS are more likely to develop subclinical carotid artery atherosclerosis, a surrogate marker of cardiovascular disease than those without MS (Mangili et al 2007). Nucleoside reverse transcriptase inhibitors (NRTIs), are the most cited antiretroviral drugs associated with MS and HIV related fat accumulation (Matienez et al 2004). There is also a link between patients infected with HIV and elevated lipid levels even in those who have not yet been put in ART (Rhew et al 2003). In patients with AIDS, very low density lipoprotein cholesterol (VLDL-C) and triglycerides (TGs) increase, (Pao et al 2008) Cardiovascular disease(CVD) may become an important clinical problem for HIV infected patients mainly because the cluster of risk factors defining the metabolic syndrome increases cardiovascular risk more than each single component, (Isomaa et al 2001). The prevalence of MS among HIV –infected patients globally ranges from 17.0% to 45.4%, with most reports produced in developed nations, (Worm *et al.*, 2010). A recent study by (Kiama et al., 2018) showed a prevalence of 19.2%, higher in female at 20.7% than in males at 16.0% in an urban population of adults living with HIV in Kenya.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Introduction

The introduction of highly active antiretroviral therapy (HAART), has greatly improved the quality of health and life for those infected with HIV/AIDS. HAART has transformed a previously deadly diseases that spelt doom to those infected to a chronic manageable infectious disease that requires one to be on medication throughout their life (Alberti *et al.*, 2005)thereby decreasing morbidity and mortality. The inclusion of protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (tNRTIs) has closely been linked to the development of metabolic syndrome abnormalities and lipodystrophy (Nzuza *et al*., 2017). Metabolic syndrome is defined as a cluster of conditions that incorporates , elevated blood sugar levels, excess body fat around the waist, increased blood pressure and abnormal cholesterol or triglyceride levels which together, increase the risk of heart disease, diabetes and stroke(Alberti *et al.*, 2006).The clinical significance of these abnormalities is demonstrated by the increased prevalence of cardiovascular disease and diabetes among HIV patients (Domingos et al., 2009).

2.1.1 Prevalence of MS

The global prevalence of MS among HIV infected patients is between 17.0% and 45.5% with most reported cases from the developing nations,(Jericó et al., 2005).A study done by NguyenI *et al.*, in 2015 found the prevalence in Africa to be between 16.7% and 31.3% which almost corresponds to the study done by Jericó *et al*..in 2005.

A study done in Uganda by Muyanja *et al*, in 2016, established the prevalence of metabolic syndrome to be 17% to 58% among patients on ART. In Kenya a study done by Kaduka et al.in 2012 had a prevalence of MS among the urban population at 34.6%; 40.2% in women and 29% in men. Another study done on the prevalence and factors associated with MS among adult population living with HIV in Kenya by Kiama et al.,in 2018, had a prevalence of 19.2%; 20.7% in women and 16.0% in men.

2.2 Definition and classification of MS

Metabolic syndrome is defined as a cluster of conditions that encompasses , elevated blood sugar levels , increased blood pressure, excess body fat around the waist and elevated cholesterol or triglyceride levels which together, elevate the risk of heart disease, diabetes and stroke.(Alberti *et al.*, 2006)

There are several classifications used for defining MS, they include:

A, International Diabetes Federation (IDF) 2006

The IDF consensus worldwide definition of metabolic syndrome (2006) is, central obesity and any of the following:

i. Raised triglycerides > 1.7mMol/L
ii. Reduced HDL cholesterol < 1.03mMol/L in male and < 1.29mMol/L in women
iii. Raised blood pressure: systolic greater than 130mm Hg, diastolic greater than >85mm Hg or treatment of previously diagnosed hypertension
iv. Elevated fasting plasma glucose > 5.6mMol/L or previously diagnosed type 2 diabetes

If body mass index (BMI) is greater than 30kg/m^2, central obesity can be presumed and waist circumference does not need to be measured. (K.G.M.M. Alberti et al., 2009)

B, World Health Organization (WHO) 1998

The WHO 1998 criteria(Alberti *et al.*, 1998) require the presence of any one of the diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose or insulin.

i. Blood pressure greater than 140/90mm Hg
ii. Dyslipidemia: Triglycerides (TG) > 1.695mMol/L and High Density Lipoprotein cholesterol (HDL-C) < 0.9mMol/L in male and < 1.0mMol/L in female
iii. Central obesity: waist/hip ratio > 0.90 male, >0.85 female or a BMI >30kg/m^2

C, Modified National Cholesterol Education Program Adult Treatment Panel (NCEP/APT III) 2005

At least three of the following:(Grundy et al., 2006)

i. Abdominal obesity: waist circumference >90 cm for men, >80 cm for women
ii. High triglycerides > 1.69mMol/L
iii. Low High-Density Lipoprotein (HDL-C) <1.03mMol/L men, <1.29mMol/L women
iv. Hypertension, BP > 130/85 mmHg
v. Impaired glucose metabolism, fasting glucose level >5.6mMol/L

2.3 Pathogenesis of metabolic syndrome

There is yet a clear definition on the pathogenesis of metabolic syndrome but it is thought to be as a result of interaction between the environment and genetic factors(Grundy et al., 2006). The first event is suspected to be the development of abdominal obesity which results in insulin resistance. The adipose tissue is generally known to be metabolically active which produces various types of cytokines- adipocytokines. These include; free fatty acids, glycerol, proinflammatory mediators i.e. Tumor Necrosis Factor Alpha, Interleukin-6 and C-Reactive Protein(Lau *et al.,* 2005). With the escalating production of free fatty acids, inflammatory cytokines, adipokines and mitochondrial dysfunction contribute to impaired insulin signaling. This in turn results to decreased skeletal muscle glucose uptake, increased hepatic gluconeogenesis and *B*- cell dysfunction , resulting to hyperglycemia (Einhorn *et al*., 2003).

Though environmental and genetic factors have been linked to development of obesity, chronic low grade inflammation of multiple etiologies of both infectious and non-infectious type have been linked to the development of obesity in susceptible individuals which is further compounded by the use of disease specific medication(Grundy et al., 2006). It has been observed that chronic infections like HIV and Hepatitis C and B are associated with MS (Roed et al., 2014). The infectious disease that has been conclusively linked with MS is HIV/AIDS infection even if the patient has responded well to antiretroviral therapy. The high prevalence of MS in HIV infection has opened up new dimensions in the pathogenesis of MS(Paula *et al.,* 2013)

2.3.1 Pathogenesis of MS among HIV patients on HAART

Metabolic syndrome was first described after the initiation of protease inhibitor (PI) mainly ritonavir plus saquinavir combined antiretroviral therapy by Carr A in 1998 in a study on syndrome of peripheral lipodystrophy, insulin resistance and hyperlipidaemia among patients receiving HIV protease inhibitors, Carr A *et al,* 1998. The irrefutable success of ART led to the global availability of the drug which resulted in un-anticipated aspect of drug therapy of HIV(Paula *et al.,* 2013). PIs are aimed to target the catalytic region of HIV-1 protease. This region is homologous with regions of two human proteins which regulate lipid metabolism, they are cytoplasmic retinoic acid binding protein 1 (CRABP-1) and low density lipoprotein receptor-related protein (LRP)(Assmann *et al.,* 1996). PIs interfere with the proteins which may be the source of metabolic and somatic changes that develop in PIs-treated patients, i.e. dyslipidemia, increased C-peptide levels, insulin resistance and lipodystrophy (Murata *et al.,* 2000).

The theory is that PIs inhibit CRABP-1 and cytochrome P450-3A mediated synthesis of *cis*-9-retinoic acid and peroxisome proliferator-activated receptor type-ɣ (PPAR-ɣ) heterodimer. The inhibition heightens the rate of apoptosis of adipocytes and decreases the rate in which pre-adipocytes differentiate into adipocytes, which has a resulting effect of decreasing triglyceride storage and increasing lipid release. PIs-binding to LRP impair hepatic chylomicron uptake and endothelial triglyceride clearance, which result in hyperlipidemia and insulin resistance(Murata *et al.*, 2000). There is data that indicate dyslipidemia may be, in part, caused by either PIs-mediated inhibition of proteasome activity and accumulation of active portion of sterol regulatory element-binding protein-1c in liver cells and adipocytes(Mooser *et al.*, 2001) or Apo CII polymorphisms in HIV infected patients (Fauvel et al., 2001)

There is evidence that PIs directly inhibit the uptake of glucose in insulin-sensitive tissues such as skeletal muscle and fat tissue by inhibiting glucose transporter Glut4 (Murata *et al.*, 2000). The relationship between levels of insulin resistance and soluble type 2 tumor necrosis factor α receptors indicate an inflammatory stimulus which contribute to development of HIV associated lipodystrophy (Mynarcik *etal.,* 2000). Multiple antiretroviral drugs, stavudine, lamivudine, efavirenz, zidovudine and most of the PIs have been found to have an influence on glucose metabolism through different mechanisms with some not yet fully understood. There is

documented evidence that endothelial dysfunction has been recently described in PIs recipients, supporting the increased risk of cardiovascular disease(Stein et al., 2001)

Metabolic changes among the HIV infected start early and this is due to the systematic targeting of the immune system by the virus followed by its deterioration. The deterioration causes interruption of the cytokine network starting at the first stages of infection. There is an increase of cytokines like IL-6, tumor necrosis factor, hs-CRP, urokinase plasminogen activator receptor (suPAR) in the early stages which not only contribute to viral replication but also are the first changes that lead to the development of metabolic syndrome in future(Bastard et al., 2006). Even after the initiation of HAART and HIV viral load below detectable levels, cytokine levels in blood remain elevated. In the Isotonic Solutions and Major Adverse Renal Events Trial (SMART trial), individuals with less than 400 copies/mL of HIV RNA had high levels of hs-CRP and IL-6, 38% and 60% respectively compared to normal individuals from cohorts for cardiovascular outcomes(Neuhaus et al., 2010)

2.4 HIV – related risk factors

Inflammation is seen as a major determinant in the pathogenesis of atherosclerosis and DM, however in patients on HAART the key inflammatory molecules involved are poorly understood(De Lorenzo et al., 2008). Thoracic and epicardial fat deposition have been linked to high levels of hs-CRP, sub clinical atherosclerosis and insulin resistance in patients on HAART have been related to MS(Longenecker et al., 2013), A study done in Cameroon in 2013, showed that the prevalence of DM, fasting TG levels were higher in HIV patients than in controls(Ngatchou et al., 2013). HIV infection is an independent risk factor responsible for elevated prevalence of MS and arterial function impairment(Maloberti et al., 2013).

Changes in lipid levels, increased thrombosis and cholesterol metabolism occur as a result of increased inflammatory response and immune dysfunction due to HIV infection and are responsible for MS and cardiovascular risk in general population(Gibellini et al., 2012). Pathogenesis of dylipididemia among HIV infected individuals is associated with increased hepatic synthesis of VLDL-C, increased apolipo protein levels, decreased TG clearance (Iroezindu *et al.*, 2013), and also the effects of the viral infection which include increased circulating cytokines, IL-6, and acute phase proteins(Maloberti et al., 2013).

The HIV-1 infection can elicit alterations in the adipose tissues critical to development of lipodystrophy through adipose tissue gene expression alterations. Subsequently these adipose tissues have reduced mRNA levels of cytochrome c oxidase subunit II compared to HIV negative individuals. The concentrations decrease more in association to patients on HAART (Giralt et al., 2006)

2.5 Antiretroviral-related risk factors

Despite the success of HAART in HIV management, there is a lot of evidence that point to the relationship between HAART use and metabolic disorders, hyperlipidemia, insulin resistance and lipodystrophy(Wand et al., 2007). HIV suppression by HAART acts like as a double edged sword, in that it reduces HIV replication and also increases HIV-related cardiovascular risk through toxicity(Palella et al., 2006). Toxicity levels are dependent on the type of antiretroviral drug used and may include adverse lipoprotein changes, inflammation, insulin resistance, platelet dysfunction and vascular injury. *In-vitro* studies have demonstrated that HAART regiments containing zidovudine, efavirenz and indinavir induce toxicity through induction of cardiomyocyte and endothelial cell apoptosis resulting to endothelial dysfunction and vascular damage(Fiala et al., 2004). Imbalance in glucose metabolism depend on the antiretroviral drug used. Use of stavudine, didanosine(Adelzon *et al.*, 2013), lamivudine, zidovudine,(Blümer et al., 2008), indinavir or lopinavir/ritonavir (van Vonderen et al., 2010)and efavirenz (Dave et al., 2011) have been implicated in insulin resistance, DM and glucose metabolism changes. PI has been implicated to induce insulin resistance by acutely blocking transport of glucose by the insulin-sensitive glucose transporter GLUT4, a mechanism not found in non HIV patients with DM (Murata *et al.*, 2000)

Among the HIV population, dyslipidemia can occur as a result of uncontrolled HIV disease and clinical restoration after HAART initiation. Besides the general side effects of antiretroviral drugs, individual and genetic makeup greatly contribute to the type and degree of dyslipidemia.(Moog *et al.*, 2012). The use of stavudine, zidovudine/lamivudine, efavirenz, lopinavir/ritonavir and didanosine/stavudine have been reported to cause dyslipidemia by the following mechanisms:(Adelzon *et al.*, 2013)

i. Increased LDL-C levels
ii. Increased TG levels

iii. Reduced HDL-C levels

Another adverse side effect of some of the HAART drugs is CVD, independent of metabolic disorders(Souza, Luzia, Santos, & Rondó, 2013).

2.6 Relationship between MS and sedentary life style

Physical inactivity and sedentary behavior as habits are considered major causes of MS (Cheung, 2006), therefore increasing physical activity is important in preventing MS. A study by Ekelund *et al.*,2005, on physical activity and MS reported that the prevalence was lower in active groups than in inactive groups (Ekelund et al., 2005)

Prisoners also called inmates or offenders share environmental, social and health characteristics associated with the development of obesity, mostly due to their sedentary life style. Some studies have suggested that the prison lifestyle contribute to exacerbate chronic disease, obesity and are an impediment to the improvement of their health. The design of the prisons, which are designed in a way to reduce the movement of prisoners due to security reasons also contribute to weight gain due to restricted movement (Ginn, 2012).

The general health of prisoners has an impact in the society when they are integrated into the general population, which occurs on a daily basis. Inmates should be considered as a vulnerable population because they have limited access to healthcare resulting in many of them having poor health (Baillargeon et al., 2010).

A study done in India by (Pemminati *et al.*, 2009) on the prevalence of metabolic syndrome among inmates found it to be 57%, Triglycerides were increased in 71.9%, low HDL-C present in 86.0%, raised FPG present in 66.7% and hypertension in 45.6%.

2.7 Laboratory's role in the Diagnosis of the Metabolic Syndrome

The laboratory has an important role to play in the diagnosis of metabolic syndrome because of the 5 features; 3 are laboratory-based, including triglyceride levels of 1.7 mmol/L or more; HDL-C levels less than 1.0 mmol/L and 1.3 mmol/L in men and women, respectively; and fasting plasma glucose levels of 5.6 mmol/L. The importance of the metabolic syndrome is that it confers at least a 2-fold risk of cardiovascular disease and at least a 5-fold increased risk for subsequent diabetes. Jialal 2009

With regard to atherogenic dyslipidemia, because the laboratory reports the results for triglycerides, HDL-C, and LDL cholesterol (LDL-C) in a standardized manner, it is also incumbent on the clinical laboratory to report the non–HDL-C value, which embraces all of the atherogenic apolipoprotein B (ApoB)-carrying particles. It is simply obtained by subtracting the HDL-C value from the total cholesterol value. The non–HDL-C value is especially important in patients with triglyceride levels of 2.3 mmol/L or more, and the goal for non–HDL-C is the LDL-C goal plus 0.78 mmol/L. Thus, in a patient with diabetes and metabolic syndrome, the LDL-C goal would be less than 2.6 mmol/L and the non–HDL-C goal would be less than 3.4 mmol/L.

In addition to offering measurement of triglycerides, LDL-C, and HDL-C to help with the diagnosis of the metabolic syndrome and its management, it is incumbent on the laboratory to report the non–HDL-C value, which involves a simple calculation. Furthermore, the laboratory should gear up in the future to also report ApoB levels, which could easily emerge as a target for treatment. While much is needed with regard to standardization of an adiponectin assay, there are also other circulating biomarkers such as interleukin 6, monocyte chemotactic protein-1, leptin, plasminogen activator inhibitor-1, and retinol binding protein-4 that might be relevant to the pathogenesis of the metabolic syndrome but presently belong in the research arena.

2.8 Problem statement

Metabolic syndrome is associated with an increased risk of both diabetes and cardiovascular disease. It is a manageable condition when detected early but if not, it may lead to development of Diabetes and CVD. Metabolic syndrome is a major global healthcare challenge affecting 7% - 47% of the global population, among the patients on HAART it affects 17.0% to 45.4%, with most reports produced in developed nations. Kenya has a prevalence of 19.2%, higher in women at 20.7% while men at 16% but no data on the prevalence of inmates on HAART. The health of inmates' it is important because when they are released, they are integrated into the general population.

2.9 Justification

Metabolic syndrome is associated with increased fasting blood sugar, increased triglycerides, reduced HDL and increased blood pressure.In 2005, the major chronic, noncommunicable diseases accounted for 60% of all deaths and 47% of the global burden of disease. 80% of

chronic disease deaths are already occurring in low and middle income countries,(WHO 2005).The prevalence of MS among HIV –infected patients globally ranges from 17.0% to 45.4%, with most reports produced in developed nations, (Worm *et al.*, 2010). A study by Madison et al on the impact of incarceration on obesity, found that due to the sedentary lifestyle of inmates they were at a higher risk developing obesity, these individuals may develop hypertension and type 2 DM. The fact that they are incarcerated and in HAART necessitated the need to establish the prevalence of MS among the prison population in Kenya. The health of inmates has an impact on public health when they are integrated into the general population; they ultimately affect public health resources and communities they return to.

2.10 Study question

What is the prevalence of metabolic syndrome among inmates on HAART in Kenya?

2.11 Hypotheses

Null hypotheses: There is no relationship between the use of HAART and the Metabolic syndrome.

2.12 STUDY OBJECTIVES

i. Broad Objective

Determine the prevalence of metabolic syndrome among inmates on HAART at Embu GK prison

ii. Specific Objectives:

i. To determine the serum concentration of fasting blood sugar,TC, LDL-C, HDL-C and triglyceride
ii. To determine the BMI and blood pressure among patients on HAART
iii. To determine compare different regimens to Ms
iv. To correlate the presence of MS with ART used

CHAPTER THREE

3.0MATERIALS AND METHODS

3.1 Methodology

3.1.1 Study Design

This was a hospital based cross-sectional study of patients on HAART for at least six months

3.1.2 Study area

The study was conducted in Embu G.K Prison CCC clinic and Biochemistry Laboratory of Embu Teaching and Referral Hospital. There is one clinic day in a week, Wednesday, where by the inmates are monitored and given their monthly dose of ARTs. In the clinic there are two qualified staff, a nurse and a clinical officer. The nurse takes their weight, height and blood pressure before they see the clinical officer. For each clinic day a total of 20 clients are attended to. The reports are manually entered into files and cards then stored in lockable cabinets.

Study subjects will be recruited from Embu G.K Prison CCC clinic. The biochemical analysis will be done at the Biochemistry Laboratory of Embu Teaching and Referral Hospital. The lab is divided into sections with each section having its own separate room for analysis. Samples were received at the reception and given a lab number. The patient information was entered into a hospital management system that generates a barcode that is then attached to the sample containers. The samples were then separated and taken to the relevant test rooms for analysis.

3.1.3 Study population

The inmates attending Embu GK Prison Dispensary CCC clinic and on HAART for at least six months

3.1.4 Inclusion criteria

i. All inmates on HAART for at least six months.
ii. Only those inmates who give informed consent will be included in the study
iii. All inmates incarcerated for 6 months or more and on HAART

3.1.5 Exclusion criteria

i. All inmates on HAART for less than 6 months.
ii. Inmates in remand but on HAART
iii. Those that do not give consent.
iv. Inmates on HAART but jailed for less than 6 months

3.2 Sample size determination

The Cochran equation (1977) below will be used to estimate the sample size:

$$n_o = \frac{Z^2 pq}{e^2}$$

Where n_0 is the sample size, Z^2 is the abscissa of the normal curve that cuts off an area α at the tails; (1 – α) equals the desired confidence level, e.g., 95%); *e* is the desired level of precision, *p* is the estimated proportion of an attribute that is present in the population, and *q* is 1-p.The value for Z is found in statistical tables which contain the area under the normal curve. E.g. Z = 1.96 for 95 % level of confidence

Therefore:

$n=1.96^2*0.17*0.83/0.05^2$, n=216.819, n= 217

p value obtained from a study done by Worm *et al.*, 2010,where p is the prevalence of Ms in the study.

3.3 Sampling method

The study recruited participants using consecutive sampling of inmates attending Embu GK prison dispensary CCC clinic who have been on HAART for more than six months until the required sample size was achieved.

Those who met the inclusion criteria were asked to fill out a consent form that explains all that was done during the course of the study.

3.4 Laboratory methods

3.4.1 Specimen Collection for laboratory analysis

Blood samples

The clinical officer obtained a signed consent from the patient then explained the procedure, assured and placed the patient in a comfortable position for sample collection. 4 ml of venous blood was collected using an aseptic technique and transferred into a plain tube and labeled appropriately. A questionnaire was filled with the help of the clinical officer.

A 12-14 hours fasting blood sample was obtained through a needle prick in the morning and the levels of Fasting Blood Sugar estimated. This was done by the research assistant, nurse. The fasting blood sample was collected just before they had their breakfast.

The blood samples were left to clot for 30 minutes then centrifuged at 2000rpm for 3 minutes. The serum was then be aspirated and transferred to serum vials, labeled appropriately and stored at 4°C awaiting transportation.

Non laboratory methods

Physical Examination

With the help of a research assistant, nurse, the height (meters) and weight (kilogram) of the study subjects was measured with the shoes off and in light clothing. The body mass index (BMI) of the participants was calculated from weight (kg)/height^2 (m^2). An automated digital manometer was used to measure blood pressure (BP) with the participants in a sitting position. two blood pressure measurements were taken while in a rested position for each study subject and then the average was calculated. Hypertension was defined as a blood pressure that is greater than or equal to 140/90 mmHg. Obesity was defined as a BMI of greater than or equal to 30.0 kg/m^2

3.5 Transporort

The blood samples were left to clot then centrifuged at 2000rpm for 3 minutes. The serum was then be aspirated and transferred to serum vials, labeled appropriately and stored at 4°C, in a fridge, awaiting transportation.

Samples were transported to Embu Teaching and Referral Hospital in a cool box for analysis.

3.6 Laboratory analysis

After recruitment of the patients, they were required to fast overnight. A fasting sample was collected for the estimation of blood glucose and lipid profile.

Fasting blood glucose was estimated using a point of care machine, OnCall Plus glucometer.

Lipid profile includes the estimation of Low-density lipoprotein (LDL), High-density lipoprotein (HDL), Triglycerides (TGs) and Total cholesterol (TC). Only the results of TGs and HDL-C will be used to aid in the classification of Ms**.** Analysis will be done using Chemray 120 Analyzer that employs turbidimetric technique.

The criteria for diagnosis of Metabolic Syndrome was the use of NCEP/APT III

3.7 Quality assurance

The study was conducted by qualified practicing personnel in the hospital. Standard operating procedures (SOPs) were followed for sample collection, processing and recording of results.

Pre-analytical phase: proper identification of patients using at least two identifiers (name/CCC number) were followed, proper labeling of samples was done.

Analytical phase: Internal Quality Control (IQC) samples were run before study samples were analyzed. Westgard rules for assessing acceptability of IQC were applied. Study samples were only run if the IQC was acceptable. The IQC material was obtained from reconstituted purchased material.

Post-Analytical phase: Machine print outs were verified by a second technical staff. The print outs were stored as required for laboratory records.

3.8 Ethical considerations

Ethical clearance was sought from UON/KNH Ethics and research committee upon proposal approval by the department.

Since prisoners are classified as a vulnerable group, permission was sought from the Prison Commissioner's office through the Embu Prison Officer in charge

Permission to analyze samples at Embu County referral clinical chemistry laboratory was sought from the laboratory management in accordance with their research activity policy.

The inmates were not enticed to participate in the study or any form of token given during or after the study.

3.9 Data management

Data was collected and stored in file maker software. Quantitative data was summarized as a mean±standard deviation. Chi-square was used to test for statistical significance

The following criteria was used for identifying MS using NCEP/APT III,

1. Raised triglycerides >1.69 mMol/l
2. Reduced HDL <1.03mmol/l (males), <1.29 mMol/l (female)
3. Raised blood pressure: systolic >130 mm Hg or diastolic >85 mm Hg
4. Raised fasting blood glucose >5.6 mMol/l
5. Body mass index > 30 kg/m^2

The prevalence of MS will be calculated using the following formula:

Prevalence = Total number with MS/Total number studied *100

Pearson correlation coefficient was used to get the r that ranges from +1 to -1 where a positive r shall show positive association of Ms and ARV used and its statistical significance.

3.10 Study limitations

Since there was no specific person assigned to that clinic and different clinicians were reviewing the patients at different times during the study period, there was likely that variation in precision of measurement could occur.

CHAPTER FOUR

RESEARCH FINDINGS, ANALYSIS AND PRESENTATION

4.1 Introduction

This section presents the analysis, results, and presentation of data.

The purpose of the study was to determine the prevalence of metabolic syndrome among inmates on HAART at Embu GK prison. The following research objectives guided the study;

i. Determining the serum concentration of fasting blood sugar,TC, LDL-C, HDL-C and triglyceride
ii. Determining the BMI and blood pressure among patients on HAART
iii. To compare different regimens to Ms
iv. To correlate the presence of MS with ART used

4.2 Data Analysis and Results

4.2.1 Response Rate

This cross-sectional study evaluated the prevalence of the metabolic syndrome in a cohort comprising of two hundred and seventeen HIV-infected patients at the Embu GK prison. The research comprised data from Jan 2020 to Jun 2020. Of the 217 samples, 64.5% (*n=140*) were male and 35.5% (*n=77*) were female. The age ranged from 23 to 83 years with mean age of 39.8, the weight ranged from 40-105 kg with the mean of 61.9 Kg and height ranged from 1.4 – 1.9 m with a mean of 1.7 m. Out of the total study subjects, 28.1% (n=61) were under AF2B regimens with 71.9% (n=156) under AF2E regimens. *See Appendix A.*

Figure 1. Gender distribution

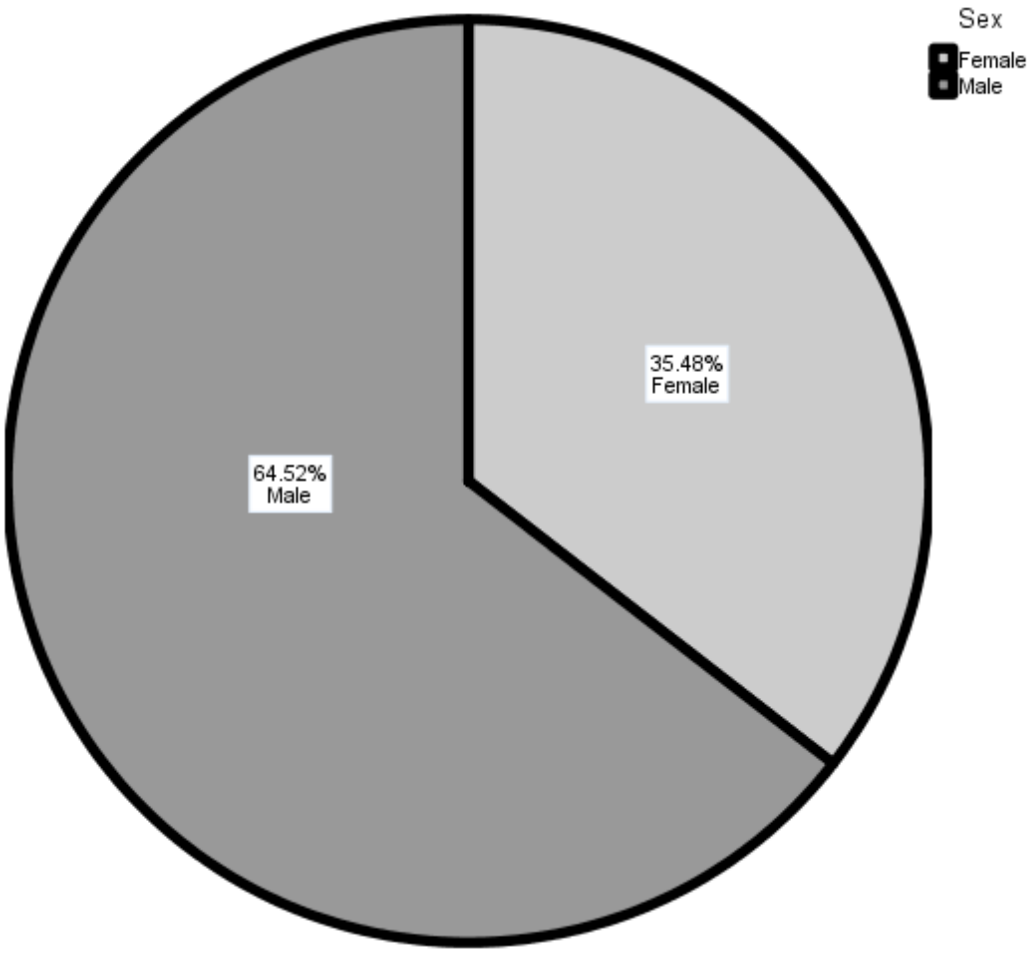


Figure 1 shows males were more at 65% than females at 35%

Figure 2. Drug combination regimes

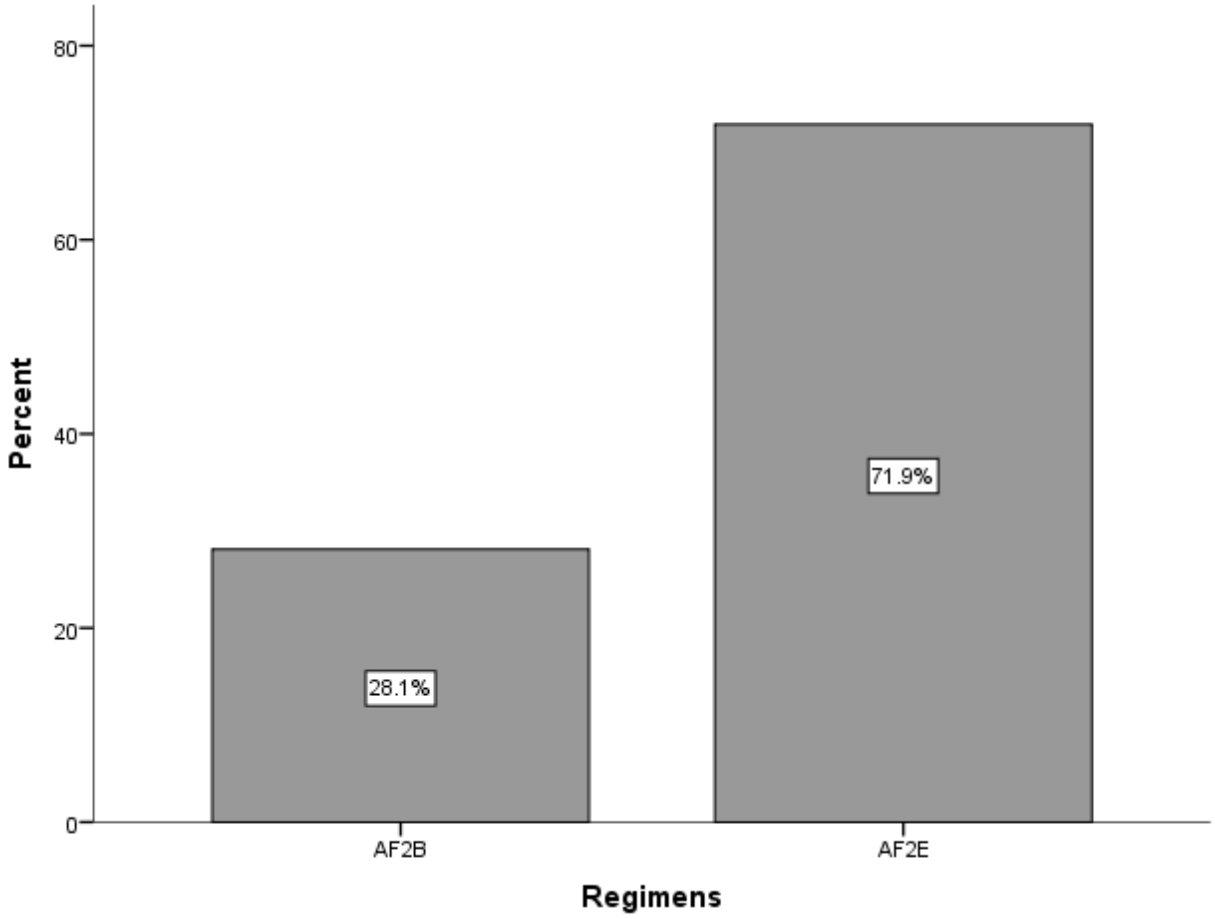


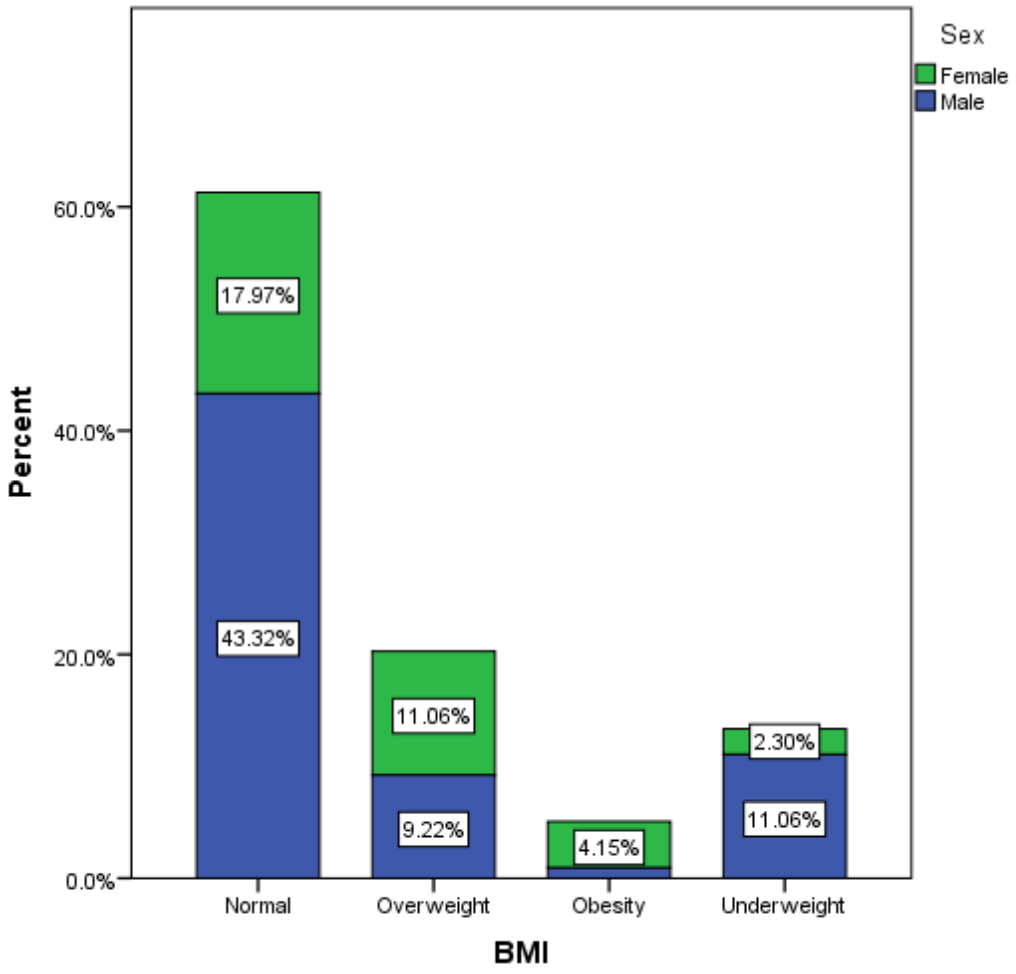
Figure 2 shows most of the inmates (71%) were on AF2E compared to AF2B (28%)

4.2.2 The serum concentration of fasting blood sugar, TC, LDL-C, HDL-C and triglyceride

Among the sampled study, 13.4% (*n=29*) showed hypertension, 10.6% (*n*=23) had low HDL, 17.1% (*n*=37) had hypertriglyceridemia, 22.1% (*n*=48) showed high fasting blood sugar, and 25.4% (*n*=55) indicated central obesity. The most frequent component observed was central obesity (25.4%) and the lowest was hypertension (10.6%).

The study went further to investigate the distribution of the risk factors against the gender. It was established that of those indicated with low HDL 15 (65.2%) were female and 8 (34.8%) were male. Those who showed hypertriglyceridemia 17 (45.9%) were female and 20 (54.1%) were male. Those showed high fasting blood sugar were female 19 (39.6%) while 29 (60.4%) were male. Of those showed hypertension 7 (24.1%) were female while 22 (75.9%) were male, and of those indicated central obesity 33 (60.0%) were female while 22 (40.0%) were male.

Figure 3: Distribution of the risking factors against gender



While crosschecking the distribution of the risk factors against age, the study established that among the patients who showed hypertension 13.8% were aged between 20-29 years, 10.3% between 30-39 years, 34.4% between 40-49 years, 27.6% between 50-59 years. The remaining 13.8% were aged 60 years and above. Of those patients who indicated low HDL 17.4% were aged 20-29 years, 26.1% were aged 30-39 years similarly to the one aged 40-49 years, 13.0% aged 40-49 years, and 17.4% aged 60 years and above. While examining those that showed hypertriglyceridemia against age, it was established that age groups 20-29, 30-39, and 40-49 each had 21.6% of the cases, age group 50-59 years had the highest cases covering 24.3%, while

60 years and older were 10.8%. Of the 55 patients who indicated central obesity 10.9% were aged 20-29 years, 25.5% were aged 30-39 years, 32.7% were aged 40-49 years, 21.8% were aged 50-59 years, and 9.1% were aged 60 years and above. Of those who showed high fasting blood sugar 20.8% were aged 20-29 years, 33.3% were aged between 30-39 years, 16.7% aged 40-49 and 50-59 years, while 60 years and above were 12.5% of the patients. *See Appendix B*

4.2.3 Determining the BMI and blood pressure among patients on HAART

Figure 4: The BMI of patients on HAART at Embu GK prison

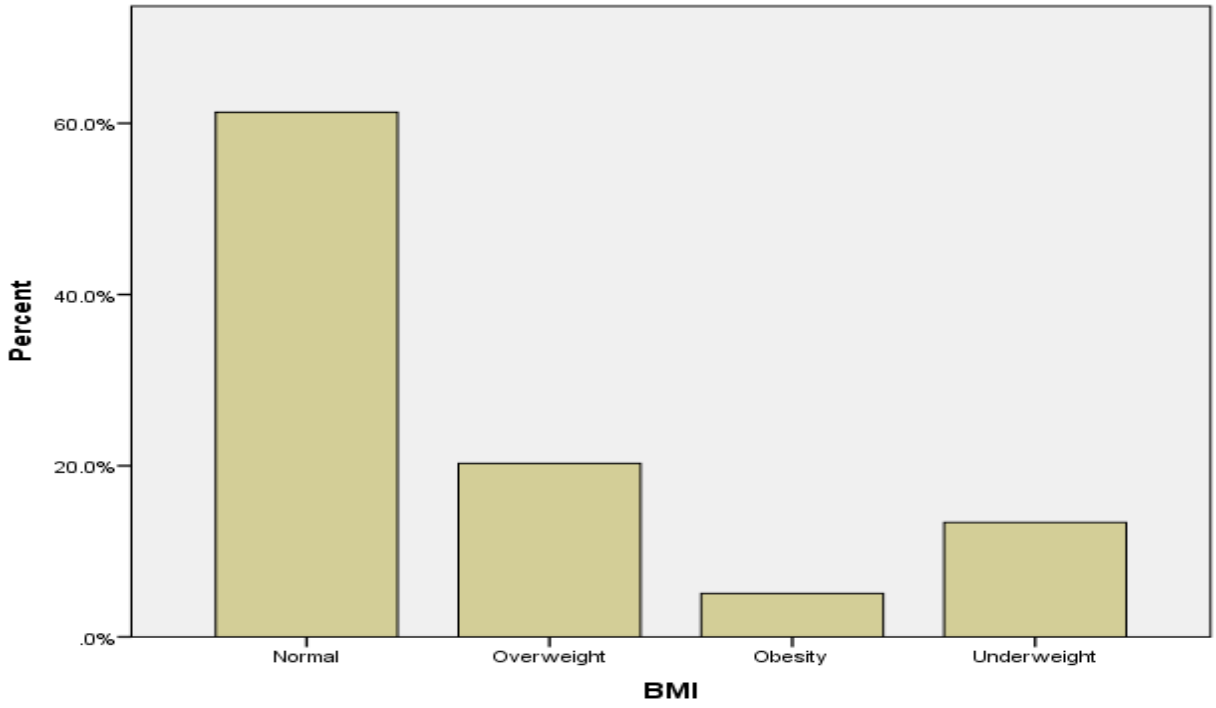


Figure 4 above shows the BMI among patients seeking HIV care at the Embu GK prison with 61.3% having normal BMI (18.5-24.9 kg/m^2), 13.4% underweight (below 18.5 kg/m^2), 20.3% overweight (25.0-29.9 kg/m^2), and 5.1% being obese (above 30 kg/m^2).

Figure 5: The blood pressure of patients on HAART at Embu GK prison

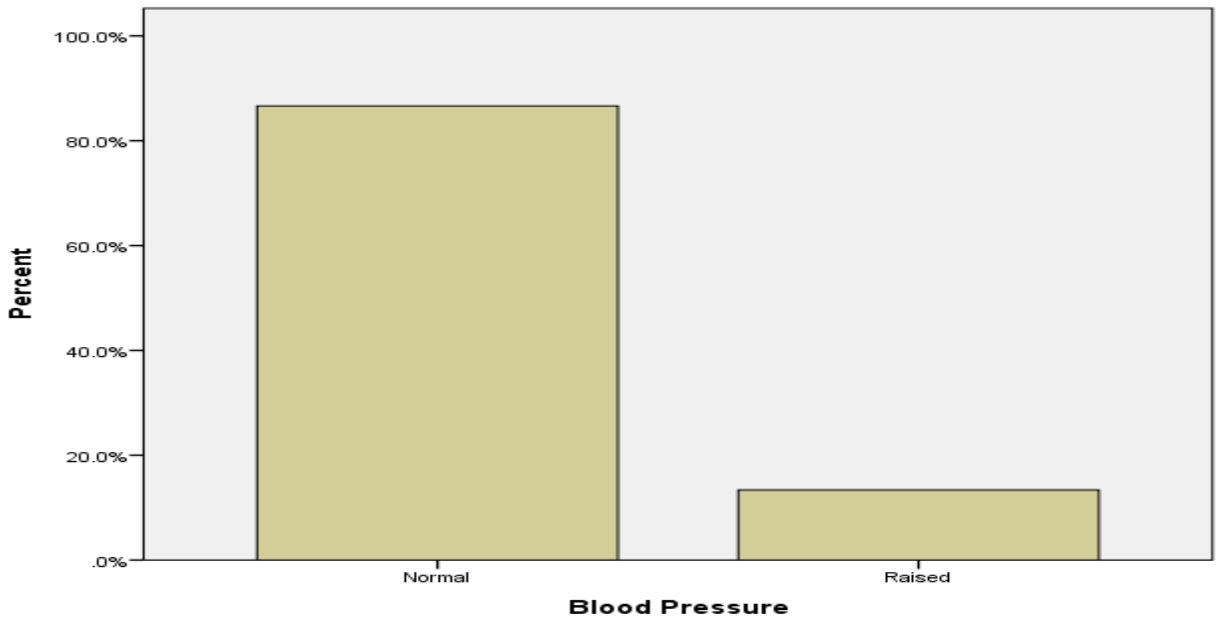


Figure 5 shows the blood pressure among patients on HAART at Embu government prison with 86.6% of the patients indicating normal blood pressure and 13.4% showing hypertension.

4.2.4 Determining the presence of metabolic syndrome in patients on HAART

Metabolic syndrome prevalence was tested using International Diabetes Federation (IDF) criteria. HIV patients with three or more of the following risk factors were defined as having metabolic syndrome: 1) hypertension (systolic greater than 130mmHg and diastolic greater than 85mmHg), 2) low HDL (< 1.03mmol/L in male and < 1.29mmol/L in women), 3) hypertriglyceridemia (>1.7mmol/L), 4) high fasting glucose (>5.6 mmol/L), and 5) central obesity (> 25.0 kg/m^2). Among the sampled HIV patients, one single risk factor presented in 57 (26.3%) patients, two in 9 (4.1%), three in 13 (6.0%), four in 12 (5.5%), and five in 6 (2.8%). Of all the subjects 120 (55.3%) showed no risk factors. The study established that 31 (14.3%) HIV patients had metabolic syndrome (they had at least three risk factors). *See Appendix C*.

Figure 6. Number of risk factors

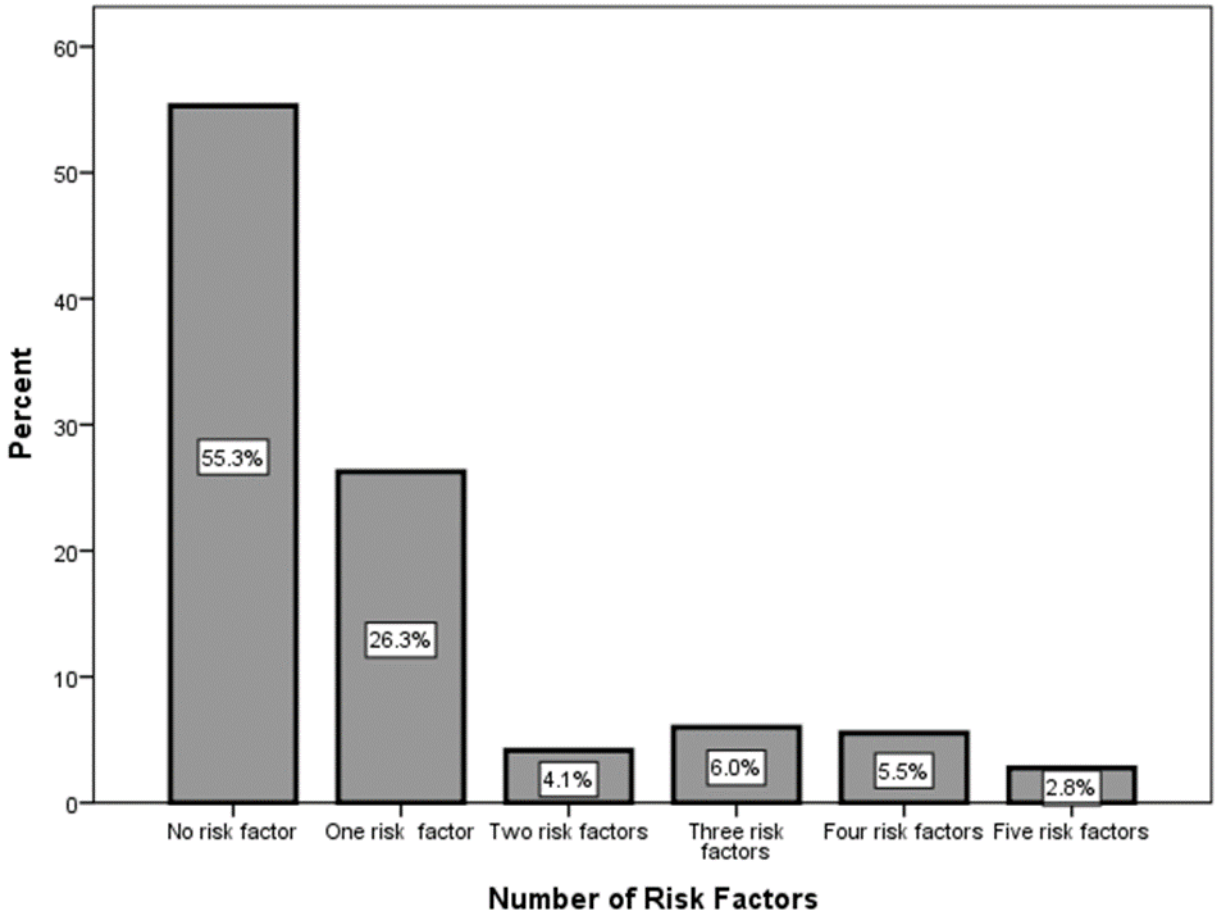


Figure 6 shows the number of risk factors.

4.2.5 Examining the relationship between presences of metabolic syndrome with ART used and against sex

The research examined the metabolic syndrome against sex and it was established that out of 31 patients who showed metabolic syndrome 17 (54.8%) were female and 14 (45.2%) were male. Using ANOVA, the study tested whether there was relationship between **p**resence of metabolic syndrome between male and female. The output indicated that there was a significant different

between sex on presence of metabolic syndrome in patients on HAART with F (1,215) = 198.887, p< 0.001.

Upon examining whether there was significant different between presences of metabolic syndrome with ART used, the data showed that there was no significant different between type of ART used and presence of metabolic syndrome among patients on HAART with F (1, 215) = 0.208, p = 0.649. The study went further to investigate the correlation between the variables "Presences of metabolic syndrome and ART type used"**,** it was established that there was a positive weak correlation between the two variables with R=0.073 and p =0.649**.** *See Appendix D.*

4.2.6 Examining the relationship between Metabolic syndrome, Regimen used and Sex

Frequency Table 1: Relationship between regimens and Metabolic syndrome

Regimens * Metabolic syndrome Crosstabulation

Count

		Metabolic syndrome		Total
		No metabolic syndrome	Presence of metabolic syndrome	
Regimens	AF2B	48	13	61
	AF2E	138	18	156
Total		186	31	217

From crosstabulation table above, out of 31 patients who indicated metabolic syndromes 13 (41.9%) were under AF2B ART while 18 (58.1%) were using AF2E ART.

While examining the relationship between specific regimens and metabolic syndromes it was established that 13 (21.3%) patients out of the total 61 HAART patients under AF2B ART showed metabolic syndrome. On the other hand, 18 (11.5%) patients out of the total 156 HAART patients under AF2E ART indicated presence of metabolic syndrome.

Frequency Table 2: Relationship between regimens against sex in patients with Metabolic syndrome

Regimens * Sex Crosstabulation

Count

		Sex		Total
		Male	Female	
Regimens	AF2B	0	13	13
	AF2E	14	4	18
Total		14	17	31

The crosstabulatin above shows that out of the 31 patients with metabolic syndromes no male under AF2B showed presence of metabolic syndrome implying that all patients under AF2B ART who indicated presence of metabolic syndrome were female. Those under AF2E ART and showed presence of metabolic syndrome, 14 (77.8%) were male while 4 (22.2%) were female.

Frequency Table 3: Distribution of regimens between sexes in the total study population

Regimens * Sex Crosstabulation

Count

		Sex		Total
		Male	Female	
Regimens	AF2B	7	54	61
	AF2E	133	23	156
Total		140	77	217

Appendix I: Descriptive Output
Appendix A
Frequency Table 4: Gender distribution

Sex

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	77	35.5	35.5	35.5
	Male	140	64.5	64.5	100.0
	Total	217	100.0	100.0	

Frequency Table 5: Statistical distribution

Statistics

		Sex	Weight	Height	Age
N	Valid	217	217	217	217
	Missing	0	0	0	0
Mean			61.92	1.6600	39.78
Minimum			40	1.44	23
Maximum			105	1.91	83

Frequency Table 6: Age distribution

Age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	20-29	32	14.7	14.7	14.7
	30-39	88	40.6	40.6	55.3
	40-49	61	28.1	28.1	83.4
	50-59	26	12.0	12.0	95.4
	60 and above	10	4.6	4.6	100.0
	Total	217	100.0	100.0	

Frequency Table 7: Distribution of regiments used

Regimens

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	AF2B	61	28.1	28.1	28.1
	AF2E	156	71.9	71.9	100.0
	Total	217	100.0	100.0	

Appendix B

Frequency Table 8: Fasting blood sugar levels

FBS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	169	77.9	77.9	77.9
	Raised	48	22.1	22.1	100.0
	Total	217	100.0	100.0	

Frequency Table 9:BMI levels

BMI

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	133	61.3	61.3	61.3
	Overweight	44	20.3	20.3	81.6
	Obesity	11	5.1	5.1	86.6
	Underweight	29	13.4	13.4	100.0
	Total	217	100.0	100.0	

Frequency Table 10: Systolic blood pressure

BP Systolic

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	188	86.6	86.6	86.6
	Raised	29	13.4	13.4	100.0
	Total	217	100.0	100.0	

Frequency Table 11: Distribution of HDL levels

HDL

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	193	88.9	89.4	89.4
	Reduced	23	10.6	10.6	100.0
	Total	216	99.5	100.0	
Missing	System	1	.5		
Total		217	100.0		

Frequency Table 12: Distribution of TG levels

TG

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	180	82.9	82.9	82.9
	Raised	37	17.1	17.1	100.0
	Total	217	100.0	100.0	

Distribution of the risking factors against age:

Frequency Table 13: Distribution of TG levels against Age

Age * TG Crosstabulation

Count

		TG		Total
		Normal	Raised	
Age	20-29	24	8	32
	30-39	80	8	88
	40-49	53	8	61
	50-59	17	9	26
	60 and above	6	4	10
Total		180	37	217

Frequency Table 14: Distribution of BP levels against Age

Age * BP Systolic Crosstabulation

Count

		BP Systolic		Total
		Normal	Raised	
Age	20-29	28	4	32
	30-39	85	3	88
	40-49	51	10	61
	50-59	18	8	26
	60 and above	6	4	10
Total		188	29	217

Frequency Table 15: Distribution of MBI levels against Age

Age * BMI Crosstabulation

Count

		BMI				Total
		Normal	Overweight	Obesity	Underweight	
Age	20-29	24	6	0	2	32
	30-39	56	7	7	18	88
	40-49	36	18	0	7	61
	50-59	14	10	2	0	26
	60 and above	3	3	2	2	10
Total		133	44	11	29	217

Frequency Table 16: Distribution of FBS levels against Age

Age * FBS Crosstabulation

Count

		FBS		Total
		Normal	Raised	
Age	20-29	22	10	32
	30-39	72	16	88
	40-49	53	8	61
	50-59	18	8	26
	60 and above	4	6	10
Total		169	48	217

Appendix C

Frequency Table 17: Distribution of the risk factors

Number of Risk Factors

Number of Risk Factors

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No risk factor	120	55.3	55.3	55.3
	One risk factor	57	26.3	26.3	81.6
	Two risk factors	9	4.1	4.1	85.7
	Three risk factors	13	6.0	6.0	91.7
	Four risk factors	12	5.5	5.5	97.2
	Five risk factors	6	2.8	2.8	100.0
	Total	217	100.0	100.0	

Appendix D

Relationship between presence of metabolic syndrome with ART used and against sex

Frequency Table 18: Distribution of risk factors against sex

Number of Risk Factors * Sex Crosstabulation

Count

		Sex		Total
		Male	Female	
No of Risk Factors	No risk factor	83	37	120
	One risk factor	35	22	57
	Two risk factors	8	1	9
	Three risk factors	6	7	13
	Four risk factors	8	4	12
	Five risk factors	0	6	6
Total		140	77	217

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
No of Risk Factors	Between Groups	.370	1	.370	.208	.649
	Within Groups	381.750	215	1.776		
	Total	382.120	216			
Sex	Between Groups	23.872	1	23.872	198.887	.000
	Within Groups	25.806	215	.120		
	Total	49.677	216			

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	.031	.073	.456	.649[c]
Ordinal by Ordinal	Spearman Correlation	-.041	.071	-.600	.549[c]
N of Valid Cases		217			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

CHAPTER FIVE

5.1 Discussion

This study was conducted at Embu GK prison dispensary, comprehensive care clinic to determine the prevalence of metabolic syndrome among patients who were on HAART.

The study had a sample size of 217 participants of whom 64.5% were males while 35.5% females. From this population it was estimated that the prevalence of metabolic syndrome was 14.3% which compares to the global prevalence that ranges from 17.0% to 45.4% with most reported cases in developing nations, Worm *et al.*,2010. The prevalence of MS was higher in female at 54.8% while that of males was 45.2% which is in line with a study done in Kenya by Kiama *et al*.,2019 that showed females had a higher prevalence that males. It also corresponds to a study done by Okafor *et al.*,2012 that MS was more common in females than males in Africa. From the study ANOVA was used and it estimated that there was significant difference between sexes on the prevalence of metabolic syndrome in patients on HAART.

It was established that the distribution of MS by age among participants who were aged 20-29, 30 – 39, 40 – 49 had a prevalence of 21.6%. Of the cases, ages 50 – 59 had the highest prevalence at 24.3% which suggests an increase in the prevalence of MS with age. This is in line with studies conducted by Okafor *et al.*,2012, Ogbera *et al.*,2010 and Fezeu *et al.*,2007. Available data from the study shows that despite the increasing trend in prevalence of MS with increase in age, adults classified as middle aged 40 – 59 years are predominantly affected. In the prison setup this can be attributed to the sedentary life style due to reduced activities and movement.

Out of the total study subjects 28.1% were under AF2B regimens with 71.9% were under AF2E regimens from the study it was established there was no significant difference between the type of ART used and the prevalence of metabolic syndrome among patients on HAART with $F(1,215) = 0.208$, $p= 0.649$. Out of the 31 patients with metabolic syndromes no male under AF2B showed presence of metabolic syndrome implying that all patients under AF2B ART who indicated presence of metabolic syndrome were female. Those under AF2E ART and showed presence of metabolic syndrome, 14 (77.8%) were male while 4 (22.2%) were female. The study further investigated correlation between the variables, TC, FBS, BMI, LDL-C and HDL-C, presence of metabolic syndrome and ART used, it was established that there was a weak positive correlation between the variables with $R= 0.073$ and $p= 0.649$.

The study established that among those with metabolic syndrome, females had the highest percentage of low HDL-C at 65.2% and central obesity a 60.0% which corresponds to studies done recently by Kiama *et al*., 2018 and Munyua *et al*.,2016. Males had a higher prevalence of hypertriglyceridemia at 54.1%, elevated fasting blood sugar at 60.4% and hypertension at 75.9%.in terms of age, the middle aged had a higher prevalence of hypertension that is 40-49 years 43.4%while those aged 50 – 59 at 27.6%. A study by Madison *et al.*,2015 On the impact of incarceration on obesity found that due to the sedentary life of inmates, they were at a higher risk of developing obesity, DM and hypertension. The elevated levels correspond to a study done on prisoners in India by Pemminati *et al*., 2009. The prevalence of Dm, hypertension and obesity was higher among inmates than in the general population when we compare this study to that done by Kiama *et al.*,2018 among patients on HAART which can be attributed to their sedentary lifestyle. A study by Ekulund *et al*., 2005 on physical activity and MS, reported that the prevalence was lower in active groups than in inactive groups.

For those who were found to be having metabolic syndrome, the study established that they had a number of risk factors that led to the diagnosis. Those that had 3 risk factors were 13 representing 6.0% of the total population: those with 4 risk factors were 12 representing 5.5% of the entire population while those with 5 risk factors were 6 representing 2.8% of the population. From these its evident that majority of those with MS had only 3 risk factors. The distribution of the risk factors by gender revealed that the number of males and females who had 3 risk factors was almost equal with males having 6 while females had 7. For those who had 4 risk factors the number of males was higher at 8 compared to females at 4. There were no males who had 5 risk factors yet there were 6 females who had 5risk factors. The finding of this study are in agreement with previous studies by Kiama *et al*., Munyunja *et al.,* and Pemminati *et al*., these findings are encouraging and prompts further studies to evaluate the prevalence of MS among inmates in the country.

5.2 Conclusion

- This study estimated the prevalence of metabolic syndrome among inmates on HAART was 14.3%
- The data showed that there was no significant difference between ART used and presence of metabolic syndrome among patients on HAART.
- The study established a weak positive correlation between presence of metabolic syndrome and ART used.
- Using ANOVA it indicated that there was significant difference between sex on the presence of metabolic syndrome in patients on HAART.

REFERENCES:

1. Alberti, K. G. M. M., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., … International Association for the Study of Obesity. (2009). Harmonizing the Metabolic Syndrome. *Circulation*, *120*(16), 1640–1645. https://doi.org/10.1161/CIRCULATIONAHA.109.192644
2. Alberti, K. G. M. M., P. Zimmet, & J. Shaw. (2006). Metabolic syndrome—a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabetic Medicine*, *23*(5), 469–480. https://doi.org/10.1111/j.1464-5491.2006.01858.x
3. Alberti, K. G. M. M., Zimmet, P., & Shaw, J. (2005). The metabolic syndrome - A new worldwide definition. *Lancet*. https://doi.org/10.1016/S0140-6736(05)67402-8
4. Alberti, K. G. M. M., & Zimmet, P. Z. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO Consultation. *Diabetic Medicine*, *15*(7), 539–553. https://doi.org/10.1002/(SICI)1096-9136(199807)15:7<539::AID-DIA668>3.0.CO;2-S
5. Assmann, G., Schulte, H., & von Eckardstein, A. (1996). Hypertriglyceridemia and elevated lipoprotein(a) are risk factors for major coronary events in middle-aged men. *The American Journal of Cardiology*, *77*(14), 1179–1184. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8651092
6. Baillargeon, J., Giordano, T. P., Harzke, A. J., Spaulding, A. C., Wu, Z. H., Grady, J. J., … Paar, D. P. (2010). Predictors of Reincarceration and Disease Progression Among Released HIV-Infected Inmates. *AIDS Patient Care and STDs*, *24*(6), 389–394. https://doi.org/10.1089/apc.2009.0303
7. Barbaro G. Metabolic and cardiovascular complications of highly active antiretroviral therapy for HIV infection. (2006).
8. Bastard, J.-P., Maachi, M., Lagathu, C., Kim, M. J., Caron, M., Vidal, H., … Feve, B. (2006). Recent advances in the relationship between obesity, inflammation, and insulin resistance. *European Cytokine Network*, *17*(1), 4–12. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16613757
9. Blümer, R. M., van Vonderen, M. G., Sutinen, J., Hassink, E., Ackermans, M., van Agtmael, M. A., … Sauerwein, H. P. (2008). Zidovudine/lamivudine contributes to

insulin resistance within 3 months of starting combination antiretroviral therapy. *AIDS*, *22*(2), 227–236. https://doi.org/10.1097/QAD.0b013e3282f33557

10. Carr A1, Samaras K, Burton S, Law M, Freund J, Chisholm DJ, C. D. (n.d.). A syndrome of peripheral lipodystrophy, hyperlipidaemia and insulin resistance in patients receiving HIV protease inhibitors. *US National Library of Medicine National Institutes of Health*, *12*(7), 5–8. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9619798
11. Cheung, B. M. Y. (2006). The metabolic syndrome in China. *British Journal of Clinical Pharmacology*, *62*(3), 260–263. https://doi.org/10.1111/j.1365-2125.2006.02663.x
12. Dave, J. A., Lambert, E. V, Badri, M., West, S., Maartens, G., & Levitt, N. S. (2011). Effect of Nonnucleoside Reverse Transcriptase Inhibitor–Based Antiretroviral Therapy on Dysglycemia and Insulin Sensitivity in South African HIV-Infected Patients. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, *57*(4), 284–289. https://doi.org/10.1097/QAI.0b013e318221863f
13. De Lorenzo, F., Collot-Teixeira, S., Boffito, M., Feher, M., Gazzard, B., & McGregor, J. L. (2008). Metabolic-inflammatory changes, and accelerated atherosclerosis in HIV patients: rationale for preventative measures. *Current Medicinal Chemistry*, *15*(28), 2991–2999. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19075647
14. Domingos, H., Cunha, R. V. da, Paniago, A. M. M., Martins, D. M., Elkhoury, E. B., & Souza, A. S. de. (2009). Metabolic effects associated to the highly active antiretroviral therapy (HAART) in AIDS patients. *The Brazilian Journal of Infectious Diseases : An Official Publication of the Brazilian Society of Infectious Diseases*, *13*(2), 130–136. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/20140358
15. Einhorn, D., Reaven, G. M., Cobin, R. H., Ford, E., Ganda, O. P., Handelsman, Y., … Wilson, P. W. F. (2003). American College of Endocrinology position statement on the insulin resistance syndrome. *Endocrine Practice : Official Journal of the American College of Endocrinology and the American Association of Clinical Endocrinologists*, *9*(3), 237–252. https://doi.org/10.4158/EP.9.S2.5
16. Ekelund, U., Brage, S., Franks, P. W., Hennings, S., Emms, S., & Wareham, N. J. (2005). Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians: the Medical Research Council Ely Study. *Diabetes Care*, *28*(5), 1195–1200. Retrieved from

http://www.ncbi.nlm.nih.gov/pubmed/15855588

17. Fauvel, J., Bonnet, E., Ruidavets, J. B., Ferrières, J., Toffoletti, A., Massip, P., … Perret, B. (2001). An interaction between apo C-III variants and protease inhibitors contributes to high triglyceride/low HDL levels in treated HIV patients. *AIDS (London, England)*, *15*(18), 2397–2406. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11740190
18. Fiala, M., Murphy, T., MacDougall, J., Yang, W., Luque, A., Iruela-Arispe, L., … Arthos, J. (2004). HAART drugs induce mitochondrial damage and intercellular gaps and gp120 causes apoptosis. *Cardiovascular Toxicology*, *4*(4), 327–337. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15531776
19. Gibellini, D., Miserocchi, A., Tazzari, P. L., Ricci, F., Clò, A., Morini, S., … Re, M. C. (2012). Analysis of the effects of HIV-1 Tat on the survival and differentiation of vessel wall-derived mesenchymal stem cells. *Journal of Cellular Biochemistry*, *113*(4), 1132–1141. https://doi.org/10.1002/jcb.23446
20. Ginn, S. (2012). Prison environment and health. *BMJ*, *345*, e5921. https://doi.org/10.1136/BMJ.E5921
21. Giralt, M., Domingo, P., Guallar, J. P., Rodriguez de la Concepción, M. L., Alegre, M., Domingo, J. C., & Villarroya, F. (2006). HIV-1 infection alters gene expression in adipose tissue, which contributes to HIV- 1/HAART-associated lipodystrophy. *Antiviral Therapy*, *11*(6), 729–740. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17310817
22. Grundy, S. M., Cleeman, J. I., Daniels, S. R., Donato, K. A., Eckel, R. H., Franklin, B. A., … Costa, F. (2006). Diagnosis and management of the metabolic syndrome An American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Current Opinion in Cardiology*. https://doi.org/10.1113/jphysiol.2006.113472
23. Iroezindu, M., & Daniyam, C. (2013). Lipid Profile of Anti-Retroviral Treatment-Naove HIV-Infected Patients in Jos, Nigeria. *Annals of Medical and Health Sciences Research*, *3*(1), 26. https://doi.org/10.4103/2141-9248.109468
24. Jericó, C., Knobel, H., Montero, M., Ordoñez-Llanos, J., Guelar, A., Gimeno, J. L., … Pedro-Botet, J. (2005). Metabolic syndrome among HIV-infected patients: prevalence, characteristics, and related factors. *Diabetes Care*, *28*(1), 132–137. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15616246

25. Kaduka, L. U., Kombe, Y., Kenya, E., Kuria, E., Bore, J. K., Bukania, Z. N., & Mwangi, M. (2012). Prevalence of Metabolic Syndrome among an Urban Population in Kenya. *Diabetes Care*, *35*(4), 887–893. https://doi.org/10.2337/dc11-0537
26. Kiama, C. N., Wamicwe, J. N., Oyugi, E. O., Obonyo, M. O., Mungai, J. G., Roka, Z. G., & Mwangi, A. (2018). Prevalence and factors associated with metabolic syndrome in an urban population of adults living with HIV in nairobi, Kenya. *Pan African Medical Journal*. https://doi.org/10.11604/pamj.2018.29.90.13328
27. Lau, D. C. W., Dhillon, B., Yan, H., Szmitko, P. E., & Verma, S. (2005). Adipokines: molecular links between obesity and atheroslcerosis. *American Journal of Physiology-Heart and Circulatory Physiology*, *288*(5), H2031–H2041. https://doi.org/10.1152/ajpheart.01058.2004
28. Longenecker, C. T., Jiang, Y., Yun, C.-H., Debanne, S., Funderburg, N. T., Lederman, M. M., … McComsey, G. A. (2013). Perivascular fat, inflammation, and cardiovascular risk in HIV-infected patients on antiretroviral therapy. *International Journal of Cardiology*, *168*(4), 4039–4045. https://doi.org/10.1016/j.ijcard.2013.06.059
29. Maloberti, A., Giannattasio, C., Dozio, D., Betelli, M., Villa, P., Nava, S., … Mancia, G. (2013). Metabolic Syndrome in Human Immunodeficiency Virus–Positive Subjects: Prevalence, Phenotype, and Related Alterations in Arterial Structure and Function. *Metabolic Syndrome and Related Disorders*, *11*(6), 403–411. https://doi.org/10.1089/met.2013.0008
30. Moog, A.-M., & Becker, W. (2012). [Management of dyslipidemia in HIV-infected patients]. *MMW Fortschritte Der Medizin*, *154 Suppl 1*, 34–36. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22916597
31. Mooser, V., & Carr, A. (2001). Antiretroviral therapy-associated hyperlipidaemia in HIV disease. *Current Opinion in Lipidology*, *12*(3), 313–319. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11353335
32. Murata, H., Hruz, P. W., & Mueckler, M. (2000). The Mechanism of Insulin Resistance Caused by HIV Protease Inhibitor Therapy. *Journal of Biological Chemistry*, *275*(27), 20251–20254. https://doi.org/10.1074/jbc.C000228200
33. Muyanja, D., Muzoora, C., & Muyingo, A. (2016). High Prevalence of Metabolic Syndrome and Cardiovascular Disease Risk Among People, *30*(1), 4–10.

https://doi.org/10.1089/apc.2015.0213

34. Mynarcik, D. C., McNurlan, M. A., Steigbigel, R. T., Fuhrer, J., & Gelato, M. C. (2000). Association of severe insulin resistance with both loss of limb fat and elevated serum tumor necrosis factor receptor levels in HIV lipodystrophy. *Journal of Acquired Immune Deficiency Syndromes (1999)*, *25*(4), 312–321. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11114831
35. Neuhaus, J., Jacobs, Jr, D. R., Baker, J. V., Calmy, A., Duprez, D., La Rosa, A., … Neaton, J. D. (2010). Markers of Inflammation, Coagulation, and Renal Function Are Elevated in Adults with HIV Infection. *The Journal of Infectious Diseases*, *201*(12), 1788–1795. https://doi.org/10.1086/652749
36. Ngatchou, W., Lemogoum, D., Nga, E., Yiagnigni, E., Tiogou, E., Kouanfack, E., … Hermans, M. P. (2013). Increased burden and severity of metabolic syndrome and arterial stiffness in treatment naïve HIV+ patients from Cameroon. *Vascular Health and Risk Management*, *9*, 509. https://doi.org/10.2147/VHRM.S42350
37. Nguyen, K. A., Peer, N., Mills, E. J., Kengne, A. P., & Menéndez-arias, L. (2015). A Meta-Analysis of the Metabolic Syndrome Prevalence in the Global HIV-Infe...: EBSCOhost, (January 1998). Retrieved from http://web.a.ebscohost.com/ehost/pdfviewer/pdfviewer?vid=0&sid=c4dafe8d-84d3-4bf2-abbf-01274f6c75b1%40sessionmgr4010
38. Nzuza, S., Zondi, S., Hurchund, R., & Owira, P. M. (2017). Highly Active Antiretroviral Therapy-Associated Metabolic Syndrome and Lipodystrophy: Pathophysiology and Current Therapeutic Interventions. *Journal of Endocrinology and Metabolism*, *7*(4), 103–116. https://doi.org/10.14740/JEM.V7I4.364
39. Osei, K. (2010). Metabolic syndrome in blacks: Are the criteria right? *Current Diabetes Reports*. https://doi.org/10.1007/s11892-010-0116-4
40. Palella, F. J., Baker, R. K., Moorman, A. C., Chmiel, J. S., Wood, K. C., Brooks, J. T., … HIV Outpatient Study Investigators. (2006). Mortality in the Highly Active Antiretroviral Therapy Era. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, *43*(1), 27–34. https://doi.org/10.1097/01.qai.0000233310.90484.16
41. Paula, A. A., Falcão, M. C. N., & Pacheco, A. G. (2013a). Metabolic syndrome in HIV-infected individuals: Underlying mechanisms and epidemiological aspects. *AIDS*

Research and Therapy, *10*(1). https://doi.org/10.1186/1742-6405-10-32

42. Paula, A. A., Falcão, M. C., & Pacheco, A. G. (2013b). Metabolic syndrome in HIV-infected individuals: underlying mechanisms and epidemiological aspects. *AIDS Research and Therapy*, *10*(1), 32. https://doi.org/10.1186/1742-6405-10-32
43. Pemminati, S., Adhikari, P., Pai, M. R. S. M., & Pathak, R. (2009). Metabolic syndrome among inmates of a "home for aged" using IDF 2005 criteria. *Nepal Medical College Journal : NMCJ*, *11*(1), 31–33. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19769234
44. Roed, T., Kristoffersen, U. S., Knudsen, A., Wiinberg, N., Lebech, A.-M., Almdal, T., … Weis, N. (2014). Increased prevalence of coronary artery disease risk markers in patients with chronic hepatitis C--a cross-sectional study. *Vascular Health and Risk Management*, *10*, 55–62. https://doi.org/10.2147/VHRM.S53557
45. Souza, S. J., Luzia, L. A., Santos, S. S., & Rondó, P. H. C. (2013). Lipid profile of HIV-infected patients in relation to antiretroviral therapy: a review. *Revista Da Associação Médica Brasileira*, *59*(2), 186–198. https://doi.org/10.1016/j.ramb.2012.11.003
46. Stein, J. H., Klein, M. A., Bellehumeur, J. L., McBride, P. E., Wiebe, D. A., Otvos, J. D., & Sosman, J. M. (2001). Use of human immunodeficiency virus-1 protease inhibitors is associated with atherogenic lipoprotein changes and endothelial dysfunction. *Circulation*, *104*(3), 257–262. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11457741
47. van Vonderen, M. G. A., Blümer, R. M. E., Hassink, E. A. M., Sutinen, J., Ackermans, M. T., van Agtmael, M. A., … Reiss, P. (2010). Insulin Sensitivity in Multiple Pathways Is Differently Affected During Zidovudine/Lamivudine-Containing Compared With NRTI-Sparing Combination Antiretroviral Therapy. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, *53*(2), 186–193. https://doi.org/10.1097/QAI.0b013e3181c190f4
48. Wand, H., Calmy, A., Carey, D. L., Samaras, K., Carr, A., Law, M. G., … INITIO Trial International Coordinating Committee. (2007). Metabolic syndrome, cardiovascular disease and type 2 diabetes mellitus after initiation of antiretroviral therapy in HIV infection. *AIDS*, *21*(18), 2445–2453. https://doi.org/10.1097/QAD.0b013e3282efad32
49. Worm, S., Friis-Møller, N., Bruyand, M., Aids, A. M.-, & 2010, undefined. (n.d.). High prevalence of the metabolic syndrome in HIV-infected patients: impact of different definitions of the metabolic syndrome. *Journals.Lww.Com*. Retrieved from

https://journals.lww.com/aidsonline/Fulltext/2010/01280/Cardiovascular_disease_risk_factors_in_HIV.15.aspx

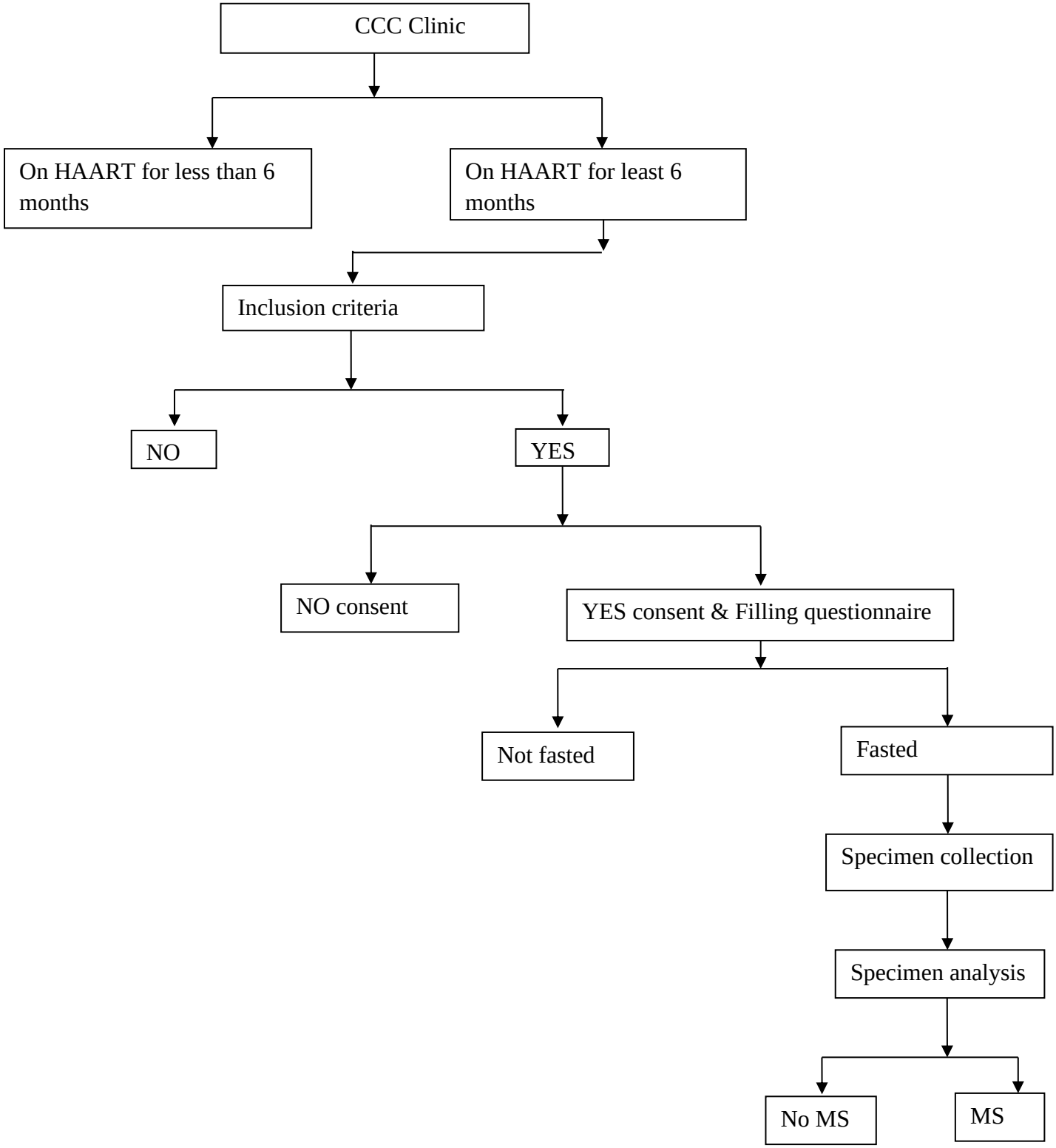
50. Yerramasu, A., Dey, D., Venuraju, S., Anand, D. V., Atwal, S., Corder, R., … Lahiri, A. (2012). Increased volume of epicardial fat is an independent risk factor for accelerated progression of sub-clinical coronary atherosclerosis. *Atherosclerosis*, *220*(1), 223–230. https://doi.org/10.1016/j.atherosclerosis.2011.09.041

BUDGET

ITEM	COST PER ITEM	NUMBER OF ITEMS	TOTAL
Triglyceride kit	200	240	48,000
HDL-C kit	200	240	48,000
LDL-C kit	200	240	48,000
Glucometer (oncall plus)	5,000	1	5,000
Glucostrips	2,000	15	30,000
Gloves	300	4	1,200
Needles and syringes	1,200 per pkt	5 pkts	6,000
Vacutainer (red top)	2,500 per pkt	3 pkts	7,500
Research assistant	10,000	2	20,000
Statistician			25,000
Stationary and printing			10,000
		TOTAL	**237,200**

APPENDICES:

APPENDIX 1: WORK FLOW DIAGRAM



APPENDIX 2: PATIENT INFORMATION AND CONSENT FORM

This informed consent form has two parts:

i. Information sheet, containing information on the study
ii. Certificate of consent

PART 1 INFORMATION SHEET

My name is Lewis Mutinda, am a postgraduate student in the Department of Human Pathology at the University of Nairobi, am conducting this study to determine the prevalence of metabolic syndrome among inmates on HIV drugs

You are invited to take part in the study. In case you do not understand any of the words used in this information sheet or have any questions please ask me to stop and explain.

Purpose of the study:

This study involves inmates attending this CCC clinic. The purpose of this study is to check the fasting blood sugar level, body fat serum levels. The BMI (if someone is over weight)will be calculated after obtaining the Height and weight. The blood pressure will be obtained before the blood sample is collected. Elevation of these parameters is indicative of metabolic syndrome

Participant selection:

The target is adult inmates attending the CCC clinic at Embu Prison Dispensary who have been on HIV drugs for 6 months or more

Voluntary participation:

Your participation in this research study is entirely voluntary and it is your choice to take part in it or not. There is no incentive or token of whichever form that shall be given during the duration of the study. All the medical care and tests will be done with no charge to the participant

Procedure and protocol:

Eligible participants will be interviewed and asked to join the study. All those who agree to take part in the study will be requested to sign a consent form. You will then be asked some questions that will be recorded in a questionnaire. The weight and height will be measured and the BMI calculated. The blood pressure will then be measured when the participant is seated and rested. Thereafter 5ml of blood from the vein in the forearm will be collected in a red vacutainer bottle. You may feel a little prick when the blood is been drawn which will be over within a few minutes. Glucose will be estimated using a point of care glucometer. Clean sterile needs will be used to draw the blood the disposed in the sharps container immediately after use. The blood will let to clot then centrifuged at 2000rpm for 3 minutes, the serum will then be aspirated and transferred to serum vials, labeled appropriately and stored at 4°C, in a fridge, awaiting transportation. Samples will be transported to Embu Teaching and Referral Hospital in a cool box for analysis in the clinical chemistry laboratory where the TG, HDL-C will be estimated. Samples will be disposed a week after analysis.

Side effects:

There are no side effects that can occur for taking part in this study.

Risks:

There are no risks expected during the study, but in rare times a hematoma may form at the site of the needle prick but that should subside within a few days. There is no risk of infection as we will be using sterile needles and syringes.

Benefits:

We will be able to diagnose metabolic syndrome and refer you to a physician for further care and management and the tests will be done for free.

Reimbursement:

You will not be given any money, gifts or any form of token for taking part in the study.

Confidentiality:

All the participants will be identified using a number, names will not be used. All information shared by you during this study will be viewed by the researchers only.

Sharing the results:

The results obtained during this study will be forwarded to the doctor. We will publish the results in order that other interested people may learn from it, however your identity will never be revealed.

Request to participate in the study:

You have been explained to about the study and if you are willing to join the study I kindly request you to fill the consent form provided.

Right to refuse:

Should you decline to participate in this study, this will not affect your treatment in any way. You will still have all the benefits that you would have otherwise.

Who to contact:

If you have any questions regarding this study at anytime you may contact the principal investigator, Lewis Mutinda, 0711387870 or any of my supervisors:

i. Dr. G wandolo, P.O Box 19676-00202, Nairobi. Tel: 0721563947
ii. Mr. Maina, P.O Box 19676-00202, Nairobi. Tel: 0720713580

You can also contact the Ethics and Research committee at Kenyatta National Hospital (KNH/UON – ERC) P.O Box 20723-00202, Nairobi, Tel:+254 020 726300-9 Ext 44102

PART II: CERTIFICATE OF CONSENT:

I have read the foregoing information, or it has been read to me. I have the opportunity to raise any questions about my participation in the study and any questions I asked have been answered to my satisfaction. My rights have been explained to me and I consent voluntarily to participate in this study.

Name of participant:……..

Signature of participant:……..

If illiterate:

A literate witness must sign; if possible this person should be selected by the participant and should have no connection to the research team. Participants who are illiterate should include their thumbprint as well.

I have witnessed the accurate reading of the consent form to the potential participant and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely

Name of Witness: ……..

Signature of witness: ……

Thumb print of the Participant

FOMU YA IDHINI KICHWA CHA UTAFITI:

Hii fomu ya idhini ina sehemu mbili:

i. Habari kuhusu utafiti huu
ii. Cheti cha ithinisho

i. HABARI KUHUSU UTAFITI HUU:

Jina langu ni Lewis M Mutinda, mwanafunzi wa chuo kikuu cha Nairobi idara ya Pathologia ya wanadamu. Nigepeda kufanya utafiti ambao nitawaelezea. Tafthadhali soma ujumbe ufuatao kwa makini. Ujumbe huu utaelezwa kwa njia ya kiigereza na Kiswahili. Una uhuru wa kuchahua lugha ambayo utaelewa vyema.

Maelezo kwa ufupi

Kipimo cha damu kuchunguza ugonjwa wa dalili za metaboli kwa wagonjwa wanaotumia dawa za kupunguza makali ya virusi vya ukimwi katika hospitali ya Embu prison. Tutachukua damu yako kutoka kwa mkono na kupeleka katika hospitali kuu ya Embu. Huko nitapima vipimo kathaa ambavyo vithaonyesha iwapo una dalili za metaboli. Urefu na kilo zako zitapimwa pia shinikizola damu pia kupimwa.

Faida na tatizo za utafiti huu

Damu yako itapimwa kuchunguza ugonjwa wa dalili metaboli. Utanufaika kwa kupatamatokeo ya damu kuhusu ugonjwa wa dalili metaboli. Isipokuwa uchungu kidogo wa kudungwa, hakuna madhara ama tatizo linguine.

Taratibu ya kushiriki

Watakao shiriki katika uchunguzi huu itakuwa kwa njia ya hiari bila kushurutishwa. Ukiamua kutoshiriki, hautapoteza kwa njia yeyote haki yako ya kuhudumiwa inavyostahili.

Majibu ya uchunguzi huu utapewa daktari wako wa kufuata kliniki yako ya kawaida

Idhini ya mshiriki

Watakao shiriki katika utafiti huu itakuwa kwa hiari bila kisurutishwa. Una uhuru wa kutoshiriki au kutojibu swali lolote kwenye dodoso au kukatiza kipindi cha maswali iwapo hautaridhika na jambo lolote.

Pia waweza kutamatisha ushiriki wako kwenye utafiti huu bila kupoteza haki yako ya kutibiwa katika hospitali hii.

Anwani

Mchunguzi Lewis Mutinda, chuo kikuu cha Nairobi, SLP 19676-00202 Nairobi.

Nambari ya simu 0717387870

Pia unaweza kutafuta wasimamizi wafuatao:

i. Dr. G wandolo, SLP 19676-00202, Nairobi. Nambari ya simu 0721563947
ii. Mr. Maina, SLP 19676-00202, Nairobi. Nambari ya simu 0720713580
iii. Ethics and Research committee at Kenyatta National Hospital (KNH/UON – ERC) SLP, 20723-00202, Nairobi, Nambari ya simu: +254 020 726300-9 Ext 44102

ii. IDHINI YA MSHIRIKI:

Kama utashiriki tafathali tia sahii kwenye pengo lililoachwa hapo chini,

Mimi ……..nimesoma n anime elewa nia ya uchunguzi huu, utaratibu utakaotumika kuchukua kipimo, faida na madhara yanayo ambatana na uchunguzi huu. Nimekubali kushiriki kwa hiari bila kushurutishwa.

Sahihi ya Mshiriki …… Tarehe……

Sahihi ya Shahidi…… Tarehe……

Alama ya Kidole

Statement of the researcher/ person taking consent

I have accurately read out the information sheet to the potential participant and to the best of my ability made sure that the participant understands what the research is all about.

I confirm that the participant was given an opportunity to ask questions about the study and all the questions asked by the participant have been answered correctly and to the best of my ability.

I confirm that the individual has not been forced into giving consent and the consent has been given freely and voluntarily.

A copy of this document has been provided to the participant.

Name of Researcher/ person taking the consent:……...

Signature of Researcher/ person taking the consent:…….

Date:……..

APPENDIX 3 :DATA COLLECTION FORM
PREVALENCE OF METABOLIC SYNDROME AMONG INMATES ON HAART ATTENDING CCC CLINIC AT EMBU PRISON DISPENSARY

A. PATIENT DETAILS

STUDY NUMBER	

i. AGE

ii. GENDER (please tick)

MALE	
FEMALE	

iii. WEIGHT IN KGS

iv. HEIGHT IN METERS

v. CALCULATED BMI (Weight,kg,/Height,M^2)

vi. BLOOD PRESSURE

Diastolic	
Systolic	

B. i. TYPE/ COMBINATION OF ART

1ST LINE		
	TDF/3TC/DTG	
	TDF/3TC/EFV	
2ND LINE		
	ABC/3TC/LPVR	
	AZT/3TC/ATVR	
	TDF/3TC/ATVR	

ii. Duration of treatment (in months)

Laboratory tests results and interpolations

Triglyceride levels	Results	Reference range	Normal	abnormal
1				
2				
3				
4				
5…				

HDL-C levels	Results	Reference range	Normal	abnormal
1				
2				
3				
4				
5…				

BP levels	Results	Reference range	Normal	abnormal
1				
2				
3				
4				
5…				

FBS levels	Results	Reference range	Normal	abnormal
1				
2				
3				
4				
5…				

BMI levels	Results	Reference range	Normal	abnormal
1				
2				
3				
4				
5…				

Summary for each participant for the diagnosis of MS

Triglyceride levels	Normal	Elevated
HDL-C levels		
FBS levels		
TG levels		
BP levels		
BMI		

If any 3 are elevated its diagnostic for MS