ASSESSMENT OF LIPID PROFILE AND GLYCEMIC CONTROL AMONG DIABETIC PATIENTS AT WACHEMO UNIVERSITY NIGIST ELLENI MOHAMMAD MEMORIAL REFERRAL AND TEACHING HOSPITAL, HOSSANA, SOUTHERN ETHIOPIA

BY:-AGEZE ABOSE (MSc candidate)

A Research Thesis Submitted to School of Medical Laboratory Science, Faculty of Health Sciences, Institute of Health, Jimma University; In Partial Fulfillment of the Requirements for Master of Science Degree in Clinical Laboratory Science Specialty in Clinical Chemistry

JIMMA UNIVERSITY

INSTITUTE OF HEALTH

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SCHOOL OF MEDICAL LABORATORY SCIENCE

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BY:-AGEZE ABOSE (MSc candidate)

ADVISORS: MR. SHIFERAW BEKELE (MSc, Ass.Prof)

MR. AKLILU GETACHEW (BSc, MSc)

MR. FANTA OBSA (BSc, MSc)

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JIMMA ETHIOPIA

Abstract

Background: Diabetes mellitus is common hormonal disorder that shares the phenotype of hyperglycemia which results in dyslipidemia and diabetic complication. The fundamental method for glycemic control is hemoglobin A_1c rather than fasting blood glucose, so current study used hemoglobin A_1c.

Objective: To assess lipid profile and glycemic control among diabetic patients at Wachemo University Nigist Elleni Mohammad Memorial Referral and Teaching Hospital, Hossana, Southern Ethiopia from May1 to June 30; 2020.

Method: A hospital based cross sectional study was conducted; involving 307 diabetic patients at follow up and selected by systematic random sampling technique. Interviewer administered questionnaire was used to collect sociodemographic, clinical and behavioral characteristics. Five milliliter of venous blood sample was collected from each study participant after overnight fasting. Level of lipid profile, fasting blood glucose and hemoglobin A_1c were measured by Cobas 4000 series cobas c311Basel, Switzerland. The data were entered into Epi data version 3.1 and analyzed by SPSS version 20. Data were summarized by tables, graph and descriptive statistics. Mann-whiteny U test, spearman's correlation, bivariable and multivariable logistic regression were used. P-value <0.05 was considered as statistically significant.

Result: Overall, 253(82.4%) of the patients had hemoglobin $A_1c \geq 7\%$ and dyslipidemia was 266(86.6%). Having history of diabetic complication (AOR: 7.093, 95% CI 1.725-29.163), duration of diabetes ≥7 years (AOR: 4.096, 95%CI 1.388-12.089), lack of home glucometer usage AOR: 8.276, 95%CI 1.613-42.461), lack of practice of regular physical exercise (AOR: 5.503, 95%CI 1.601-18.9) and dyslipidemia (AOR: 2.743, 95%CI 1.129-6.662) were significantly associated with poor glycemic control. Waist circumference and lack of home glucometer usage were also significantly associated with dyslipidemia.

Conclusion: The prevalence of poor glycemic control and dyslipidemia among diabetic patients were high, therefore strategic and timely intervention is needed on associated factors to delay diabetic complication and improve health of diabetic patients.

Keywords: Diabetes mellitus, Dyslipidemia, glycemic control, Hossana, Ethiopia

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Table of content **Page**

List of Tables

List of Figures

Acronyms and Abbreviations

A_1c	Hemoglobin A_1c
AGE	Advanced glycation end products
AOR	Adjusted odd ratio
ATP	Adult treatment panel
BMI	Body mass index
CI	Confidence interval
COR	Crude odd ratio
DM	Diabetes mellitus
eAG	Estimated average glucose
FFA_S	Free fatty acid
GLUT	Glucose transporter
HC	Hip circumference
HDL-C	High density lipoprotein
IGT	Impaired glucose tolerance
IR	Insulin resistant
LDL-C	Low density lipoprotein
NCEP	National cholesterol education program
PI3-K	Phosphatidylinositol-3'-kinase
PKC	Protein kinase C
SNNPR	Southern Nations Nationalities and People's Region
SOPs	Standard operating procedures
SPSS	Statistical Package for Social Science
T1DM	Type1diabetes mellitus
T2DM	Type2 diabetes mellitus
TC	Total cholesterol
TG	Triglyceride
VLDL-C	Very low density lipoprotein
WC	Waist circumference
WHO	World Health Organization
WHR	Waist to hip ratio
WUNERTH	Wachemo University Nigist Eleni Referral and Teaching Hospital

Operational Definition

Adherence to physical exercise: When the study participants perform any type of physical exercise (aerobic, resistance and flexibility) at least 30 minutes for 3or more days per week without work related exercise.

Alcohol drinking: The study participants who drink alcohol currently.

Anthropometric indicators*:* BMI<18.5 underweight, BMI 18.5-24.9 normal, BMI 25-29.9 over weight and BMI > 30 obesity. WC for female≥ 80 cm is abnormal and WC ≥94 cm for male is abnormal

Co morbidity: The presence of hypertension was studied as co morbidity in the current study.

Dietary adherence: The study participants who follow for 3 or more days per week the recommended diet by their physicians like restriction from sugar, fat and salt.

Dyslipidemia: Is an abnormality in any one of lipid profile when HDL-C < 40 mg/dl, LDL-C >130 mg/dl, cholesterol> 200mg/dl and TG>150mg/dl.

Good glycemic control: The mean of fasting blood glucose measurement is less than 154mg/dl or HbA1c < 7% (1).

Good medication adherence: When the participants score all eight-items morisky scale questions.

Mean fasting blood glucose: Is the average of three consecutive months. The first and the second blood glucose level were taken from participant's card but the third was measured during study period.

Moderate medication adherence: When the study participants missed 1-2 question from eight item morisky scale question.

Poor glycemic control: Is mean of fasting blood glucose measurements greater than or equal to 154mg/dl or HbA1c ≥ 7% (1).

Poor medication adherence: When the study participants missed 3-8 questions from eight items moriskyquestions.

Chapter One: Introduction

1.1 Background

Diabetes mellitus (DM) is a group of common metabolic disorders that share the phenotype of hyperglycemia ,which caused by demise of insulin secretion , impairment of insulin action and resistance of the target organs(1–3). It can be classified into different clinical classes from which 5-10% of diabetic cases are type 1. It is mostly diagnosed during childhood ages and caused by β-cell damage resulting in diminished ability of the pancreas to produce insulin(4). Type 2 diabetes mellitus (T2DM) constitutes over 90-95% of all DM cases. It is mostly diagnosed after the age of 40 (2),but currently diagnosis of T2DM is dramatically increasing in children, adolescents and younger adults due to rising levels of obesity and physical inactivity (4,5).

Persistent chronic hyperglycemia causes macro vascular and micro vascular complication among diabetic patients. Macro vascular diseases include coronary artery disease, cerebral vascular disease and peripheral vascular disease depending upon the occurrence of the atherosclerotic lesions. Micro vascular complication includes nephropathy, retinopathy and neuropathy(6,7).Vascular complications in diabetes arise from one of the underlying features of hyperglycemia such as excessive non enzymatic glycation of proteins, production of oxidative stress , secretion of various cytokines and growth factors which enhance expression of adhesion factor and inflammatory response (6,8).

Glycemic control is fundamental to diabetes management, as diabetes control and complications trial demonstrated that improvement of glycemic control reduces non proliferative and proliferative retinopathy by 47%, micro-albuminuria by 39%, nephropathy by 54%, and neuropathy by 60%) (3).Glycemic management is primarily assessed with the hemoglobin A_1c test. It is one of the glycated hemoglobins and formed in two steps by the non enzymatic reaction of glucose with the N-terminal amino group of the beta-chain of normal adult HbA. The first step is reversible and yields labile HbA1c.It is rearranged to form stable HbA1c in a second reaction which reflects average glycemia of approximately 3 months(1,9).

American diabetic association and the American association for clinical chemistry determined estimated average glucose (eAG) is another way to talk to patients about diabetes management to reduce confusion of patients with the hemoglobinA1c result which is expressed in percent. The correlation between A_1c levels and mean glucose levels based on the international A_1c-Derived Average Glucose study was r= 0.92 which is strong enough to elaborate reporting both the A1C result and the estimated average glucose (1).

The plasma lipoproteins are complex of lipids and proteins. The lipoprotein particles include chylomicrons (CM), very-low-density lipo proteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Dyslipidemia is a group of disorders of lipoprotein metabolism(10). It is presence of one or more abnormal serum lipid concentration according to United State National Cholesterol Education Program Adult Treatment Panel (NCEP-ATP) III guideline**.** Lipid abnormalities are prevalent in type 2 diabetic patients because of IR(insulin resistance) which affects key enzymes and pathways in lipid metabolism, for instance apoprotein production, regulation of lipoprotein lipase, action of cholesterol ester transfer proteins and actions of insulin in hepatic and peripheral tissue (5,11).

Dyslipidemia is aggravated by poor glycaemic control. Its abnormalities play a crucial role for development of diabetic atherosclerosis. The causes for increased cardiovascular risk in type 2 diabetes are multifactorial, elevated triglycerides and low levels of high-density lipoprotein cholesterol are few major modifiable factors (2,12).

Obesity plays an important role in the pathogenesis of insulin resistance, which is commonly seen in type 2 DM patients. It enhance releasing of free fatty acids (FFAs), inflammatory mediators (such as, tumour necrosis factor α, interleukin 6), reactive oxygen species production, and systemic inflammation(13). Insulin resistance is related to abnormal function of the glucose transporter type 4 (GLUT-4) .It is insulin-mediated glucose transporter mainly found in adipose cells and muscle cells. When FFAs bind to Toll-like receptor (TLR) and PI3-kinase (PI3K) their activities are down regulated, which reduces expression of GLUT-4 leading to decreased response to insulin binding (14).

The lipid abnormalities in diabetes include quantitative changes which occur due to an increase of glucose for VLDL synthesis. Decrease in lipoprotein lipase activity lead to decrease of VLDL from peripheral circulation. Increase in LDL-C levels and decrease in HDL-C levels due to increase in hepatic activity and decrease in VLDL clearance. Qualitative changes consists of non-enzymatic glycation of LDL and HDL (15). The anti-atherosclerotic properties of HDL are reverse cholesterol transport, inhibit oxidation of LDL, decrease inflammation and improve endothelial cell functions so all this function will decrease as HDL-C decrease (16). The dyslipidemia and poor glycemic control are two deleterious events for diabetic patients, which enhance vascular complication and reduce quality of life therefore assessment of lipid profile and glycemic control status is very important to prevent vascular complication.

1.2 Statement of Problem

Diabetes mellitus is a hormonal disorder that share phenotype of hyperglycemia and it is a major global health problem which affects 463 million people worldwide (3,17). According to the International Diabetes Federation (IDF) 2000 report DM prevalence was 151 million but at 2019 it was 463 million and it will extended to 700.2 million at 2045. About 9.3% of adults with in age of 20-79 years had diabetes and 79.4% live in low and middle income countries like Ethiopia. Around 4.2 million people aged between 20 -79 years die in 2019(17).

In Africa 19 million people were affected by diabetes mellitus from 20-79 year age group. Some of Africa's most populous countries have the highest numbers of people with diabetes which includes South Africa 4.5 million, Nigeria 2.7 million, Democratic Republic of Congo 1.8 million and Ethiopia 1.7 million. About 45.1% of all adults aged 20-79 years with diabetes in the region live in these four countries. Around 366,200 deaths in Africa region are attributed to diabetes with the highest percentage of all-cause mortality due to diabetes in age group 30-39. In addition to this 73.1 % of all deaths attributable to diabetes occurred in people under 60 years and the highest prevalence in the world (17).

Currently, Ethiopia has been challenged by the growing magnitude of non-communicable diseases such as diabetes. Ethiopia is among the top four countries with the highest adult diabetic populations in sub-Saharan Africa. Patient attendance rates and medical admissions related to diabetes in major hospitals have been rising. The prevalence of diabetes mellitus is reaching epidemic proportions because of obesity and sedentary life style in adults. The incidence and prevalence of diabetes mellitus in general Ethiopian populations are unknown, but IDF of 2019 estimates that the prevalence of diabetes in age 20-70 years is 3.2% (17–19).

Poor glycemic control is major problem which causes blindness, kidney failure, lower limb amputation and several other long-term consequences that impact significantly on quality of life. Diabetes and its complications bring about substantial economic loss to people with diabetes and their families due to health systems and national economies through direct medical costs, loss of work and wages. According to World Health Organization statistics, each diabetic patient spends about three times more money on his or her health than a person without diabetes. The long-term

complications that result from poor glycemic control contribute substantially to the morbidity, mortality, and economic burden of diabetes (18,19). Based on WHO global estimates, dyslipidemia cause one third of ischemic heart disease and one fifth of global cerebrovascular disease and nearly 2.6 million deaths every year worldwide (20).

Around 72%-85% of dyslipidemia cases occur in type 2 diabetic patients (21). It is a major risk factor for macro vascular complications in type 2 diabetic patients. In patients with diabetes alteration in the distribution of lipids increases the risk of atherosclerosis. Its association with diabetes mellitus is the major cause of morbidity and mortality due to the high rate of severe cardiovascular diseases (22,23).

As I reviewed literatures from Ethiopia and abroad on lipid profile and glycemic control among diabetes mellitus patients showed that prevalence of poor glycemic control and dyslipidemia almost more than 50%. This indicates that poor glycemic control and dyslipidemia are highly increasing problem around world. Eventhougth this study conducted in some part of Ethiopia, lipid profile and glycemic control status varies due to several factors like behavior of patients, life style, genetic factors, environmental factors and awareness of diabetic patients. In addition to this most of the studies used fasting blood glucose to determine poor glycemic control which underestimates prevalence of poor glycemic control. Furthermore, no study conducted in South Nation Nationalities People Region in generally and particularly in study area Hossana, therefore the present study was designed to assess the prevalence of dyslipidemia , glycemic control and their associated factors among diabetic patients in Nigist Ellen Mohammed Memorial Referral and Teaching Hospital, Hossana ,Southern Ethiopia .

1.3 Significance of the Study

The importance of present study is to delay diabetic patients from developing complication and for early management of patients with dyslipidemia. It serves as a base data for health policy makers to formulate and implement policies to intervene poor glycemic control and dyslipidemia. In addition to this the present study may provide valuable information for different health organization workers such as physicians, nurses, pharmacist and laboratory professionals to provide good medical care for diabetic patients. The finding may serve as supportive data for the future longitudinal researches on this area.

Chapter Two: Literature Review

Diabetes is a complex chronic illness that requires continuous medical care with multifactorial risk reduction strategies like glycemic control and prevention of dyslipidemia (24). There are various factors associated to poor glycemic control and dyslipidemia like obesity, physical inactivity, alcohol consumption, type of medication, poor adherence to drug and dietary plan ,but there were inconsistence of results on factors associated with glycemic control (25,26).

Across-sectional study conducted across Europe among 5817 type 2 diabetic patients revealed that the prevalence of poor glycemic control was 37.4 %. The younger age, duration of diabetes , poor adherence to medication and poor adherence to lifestyle were significantly associated with poor glycemic control, but gender, income , body mass index, and complication type not significantly associated in poor glycemic control (27).

A retrospective cross-sectional study performed in India among a total of 55 639 eligible records of type 2 diabetic patients showed that 76.6% had poor glycemic control. Obesity, hypertension and diabetes duration ≥2 years were significantly associated to poor glycemic control, but age, gender, BMI and type of micro vascular complication were not significantly associated with poor glycemic control(28). Other cross-sectional studies conducted in India among220 diabetic patients revealed that the prevalence of poor glycemic control was 91.8% and among194 type 2 showed that dyslipidemia and lack of self glucose monitoring were significantly associated to poor glycemic control(29,30).

A cross-sectional study conducted at a diabetic center in Tabuk, Saudi Arabia among 423 type 2 diabetic patients showed that 74.9% of the patients had poor glycemic control. Family histories of diabetes, longer duration (5–10 years and >10 years), insufficient physical exercise showed that statistically significant association with poor glycemic control but residence, self-monitoring blood glucose and dietary plan were not significantly associated (31).

A cross-sectional study conducted in Bangladesh among 1253 type 2 diabetic patients revealed that the prevalence of poor glycemic control was 82%. Low education level, being female, rural residence, unhealthy eating habits, insulin use and history of coronary artery diseases were

significantly associated with poor glycemic control, but income ,physical activity and family history were not significantly associated(25).

A cross-sectional study conducted in Venezuela health centers among 4075 diabetic patients from which 349 with type1 diabetes and 3726 with type2 diabetes showed that the magnitude of poor glycemic control was 76%. It was more common in T1D patients (87%) than in those with T2D (75%)(32).

A cross-sectional study conducted in Tanzania among 469 type 2 diabetic patients indicated that the prevalence of poor glycemic control was 69.7%. Age between 40-50 years, duration of diabetes between 10-19 year, BMI between 25-29.9, low medication adherence and combination therapy of oral hypoglycemic agents were significantly associated with poor glycemic control but, marital status, sex, employment, healthy eating plan, physical activity, and self-monitoring of blood glucose, education level of the patients were not significantly associated with glycemic control(33).

A cross-sectional study was conducted in university clinics at Gadarif, eastern Sudan among 339 type 2 diabetic patients indicated that the prevalence of poor glycemic control status was 71.9%. Duration of diabetes, type medications, and the triglycerides were not significantly associated with poor glycemic control, but being single significantly associated with poor glycemic control status(34).

Institution based cross-sectional study conducted in Asmara, Eritrea among 309 patients with diabetes mellitus revealed that the magnitude of poor glycemic control was 76.7% .Abnormal WHR and patients without hypertension were significantly associated with poor glycemic control(35).

A cross-sectional study conducted at the University of Gondar Referral Hospital among 391 diabetic patients revealed that the prevalence of poor glycemic control was 64.7%. Poor glycemic control was higher among Type 1 diabetic patients (82.9%) as compared with Type 2 patients (57.5%). Insulin only treatment and poor medication adherence were significantly associated with poor glycemic control among type 2 DM and increased waist circumference had negative association with poor glycemic control among type1 DM, but duration of diabetes

,physical exercise, hypertension as co morbidity and dyslipidemia were not significantly associated .Another study carried out at Gondar after four years showed slight improvement on prevalence of poor glycemic control status which was 60.5% and agreed with pervious study poor glycemic control was higher among type1 than type2 and support the previous study which revealed that increased waist circumference had significant negative association with poor glycemic control(36,37).

A institution based case-control study done in Jimma among 410 type 2 diabetic patients revealed that co morbidities, lack of self-monitoring of blood glucose, total cholesterol level ≥200 mg/dl , duration of DM ≥7 years, physical activity, type of medication were significantly associated with poor glycemic control. Other cross-sectional studies were carried out at Nekemte Referral Hospital, Jimma University Specialized hospital and shanan gibe hospital had prevalence of poor glycemic control 73%. 70.9 %, 59.5% and 59.2 % respectively. Duration of DM and physical activity had significant association, but type of treatment; healthful eating plan and alcohol drinking were not significant in study conducted at Nekemte. Type of medication, medical adherence and being a farmer were significantly associated in study done in Jimma University Specialized hospital. Level of education and duration of diabetes were significantly associated to poor glycemic control in study conducted at shanan gibe hospital (38–42).

A comparative cross-sectional study done at Mekelle-Ethiopia among 336 diabetic patients indicated that the prevalence of poor glycemic control was 61.9%. High-triglyceride, high low-density lipoprotein and non glucometer user were significantly associated with poor glycemic control ,but type of medication, hypertension and alcohol intake were not significantly associated with poor glycemic control. Other cross-sectional studies conducted Suhul Hospital, Tigray and Dessie, Ethiopia showed the prevalence of poor glycemic control were 63.5% and 70.8% respectively. Factors such as longer duration of diabetes, low educational level and type of medication were significantly associated with poor glycemic control in study done Dessie. Physical exercise and medical adherence were significant ,but home glucometer usage was not significant in study conducted at Suhul hospital (43–45).

A hospital based cross- sectional study conducted in Tikur Anbessa Specialized Hospital among 412 type 2 diabetic patients which revealed that the prevalence of poor glycemic control was

80%. Another study conducted in the same area after two years stated that the prevalence of poor glycemic control was 68.3%. Body mass index and medical adherence were significantly associated, but physical exercise, dyslipidemia and antidiabetic medication were not significantly associated with poor glycemic control(46,47).

A cross-sectional study done in Nepal among 497 type2 diabetic patients showed that prevalence of dyslipidemia was (88.1%). Another cross-sectional studies conducted in India showed that the prevalence dyslipidemia were 90% and 97.2%(48–50).Also a nationally representative cross-sectional study conducted in china among diabetic patients showed that waist circumference was significantly associated with dyslipidemia(51).

A cross-sectional studies conducted in South Africa, Nigeria and Kenya among diabetic patients showed that prevalence of dyslipidemia were 93.5% , 90.7% and 86.1% respectively (49,52,53). Another cross-sectional study done in Tanzania among 119 type2 diabetic patients showed that waist circumference was not significantly associated, but BMI was significantly associated with dyslipidemia(15).

A facility based cross-sectional study conducted at Durame, South Ethiopia among 224 diabetic patients showed that prevalence of dyslipidaemia was 65.6%(54).

After reviewing all the above articles, all most all were used fasting blood glucose to assess the glycemic control status which underestimate the poor glycemic control. In addition to these there were variations on associated factors on some studies. The present study was tried to assess the poor glycemic control status based on hemoglobin A_1c, which recommended by American diabetic association to manage the glycemic control status and some of the studies use result of lipid profile from medical records which couldn't represent the present status of the study participants, but the present study used active lipid profile measurement.

2.2 Conceptual frame work

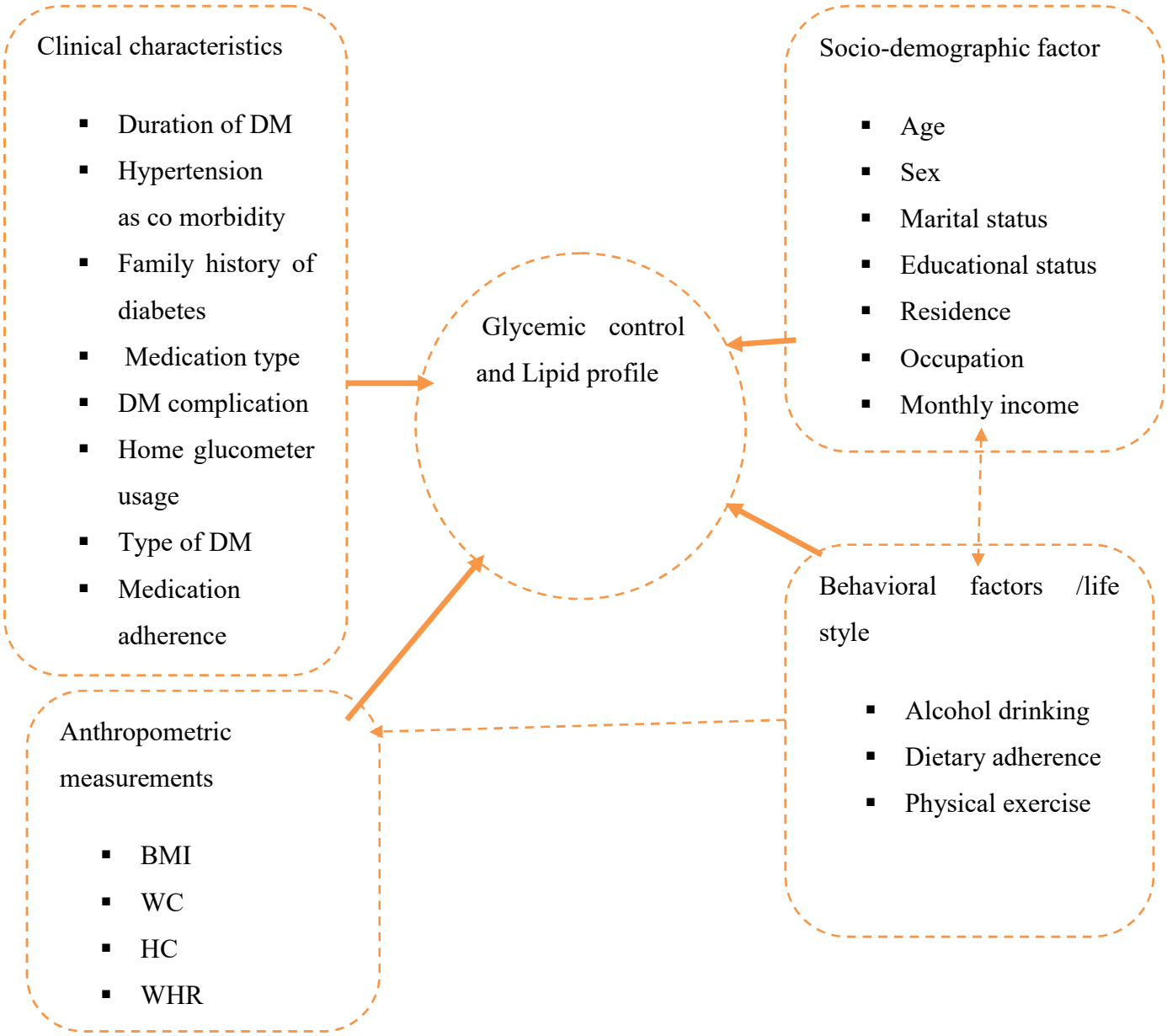

Figure 1: Conceptual frame work which was adapted from different articles for the assessment lipid profile and glycaemic control status among diabetic patients in Wachemo University Nigist Ellen Mohammad Memorial Referral and teaching Hospital, Hossana, Southern Ethiopia from May 1 to June 30, 2020.

→ The relation between independent and dependent variable and it is the aim of this study.

- - - → The relation of independent variables and it is not aim of this study.

Chapter Three: Objectives

3.1 General Objective

- To assess lipid profile and glycemic control among diabetes mellitus patients at Wachemo University Nigist Elleni Mohammad Memorial Referral and Teaching Hospital from May 1 to June 30, 2020.

3.2 Specific Objectives

- To determine prevalence of poor glycemic control among diabetic patients.
- To determine prevalence of dyslipidemia among diabetic patients.
- To identify factors associated to poor glycemic control among diabetic patients.
- To identify factors associated to dyslipidemia among diabetic patients.

Chapter Four: Methods and Materials

4.1. Study Area and Setting

The study was conducted at Wachemo University Nigist Elleni Mohammed Memorial Referral Hospital (WUNEMMRH) which is located in Hossana town, Haddiya Zone, southern, Ethiopia. The town is 232 km far from the capital-city Addis Ababa due north and 157km from regional city Hawassa due south east(55). The hospital catchment area population estimated around 3,200,000. There were a total of 1346 DM patients. The hospital provides service on different departments such as Surgical, Gyne& obstetric, Internal medicine and Pediatrics. The study was conducted at chronic care clinic. The laboratory investigation performed in central laboratory which provides parasitological, hematologic, microbiological, clinical chemistry and serological service.

4.2. Study Design and Period

A hospital based cross-sectional study design was carried out from May 1 to June30, 2020

4.3. Population

4.3.1 Source Population

All adult diabetic patients attending WUNEMMRTH chronic care clinic.

4.3.2 Study Population

All selected adult diabetic individuals attending WUNEMMRTH chronic care clinic during the study period.

4.4. Sample Size Determination and Sampling Technique

The sample size was calculated by taking 59.2% proportion of poor glycemic control from study conducted on glycemic control and associated factors among type 2 diabetic patients at Shanan Gibe Hospital, Southwest Ethiopia (41).

n= $\frac{(Z)^2 xp(q)}{d^2}$

n= $(1.96)^2$ *0.592(1-0.592)/$(0.05)^2$

n=3.84 *0.592(0.408)/$(0.05)^2$

n=371

Where n=Sample size

Z= Confidence level is 95%

P= prevalence of poor glycemic control= 59.2%

q= 1-p. 1-0.592 =0.408 d= Margin of error 5%

After finite population correction the minimum sample size was 291

By using 10 % non response rate the final sample size was 320

Sampling Technique

Systematic random sampling technique was used to select participants from a total of diabetic patients by k^{th} interval that fulfilled the inclusion criteria based on appointment frame on medical registration book at the study period. The K^{th} interval = 1346/320= 4. Every k^{th} value was selected until determined sample size obtained.

4.5 Variables

4.5.1. Dependent Variables

- ❖ Lipid profile
- ❖ Glycemic control

4.5.2. Independent Variables

- ➢ Socio-demographic factors: Age, sex, residence, marital status, educational status, occupational status and monthly income.
- ➢ Anthropometric: BMI, WC, HC and WHR.
- ➢ Clinical characteristics: Duration of diabetes from time of diagnosis , type of medication, home glucometer usage, hypertension as co morbidity, DM complication, types of diabetes, medication adherence and family history of diabetes.
- ➢ Behavioral characteristics/lifestyle: Alcohol drinking, physical exercise and dietary plan adherence.

4.6. Inclusion and Exclusion Criteria

4.6.1 Inclusion Criteria

Diabetic patients those attending chronic care follow up clinic and had regular follow-up in the diabetes clinic, at least one year follow up and on treatment with age ≥18 years were included.

4.6.2 Exclusion Criteria

The patients those were pregnant, newly diagnosed, on Statin or other lipid lowering drugs, known chronic liver disease and confirmed hypothyroidism were excluded by review of history, face to face interview, and by asking the follower physician. The pregnant women were excluded by due to anatomical and physiological changes occur during pregnancy as the result they may be anemic due to reduction of life span of red blood cells and increase of plasma volume which falsely decreaseA_1c(56). Patients with overt chronic liver disease were excluded due to anemia of chronic disease that can shorten life span of red blood cells as the result glycation of

hemoglobin A_1c will be reduced, but blood glucose level increased(57). Patients with hypothyroidism were excluded due to decrease of glucose metabolism and utilization as the result glycation of hemoglobin increase. In addition to this glucose induced insulin secretion decrease in hypothyroidism which aggravates blood glucose concentration(58). Patients on lipid lowering drug were excluded due to decrease of lipid profile.

4.7. Data Collection Techniques and Instrument

4.7.1. Socio-Demographic, Behavioral and Clinical Data

Data were collected by trained data collectors under supervision of principal investigator via structured questionnaire. The first part of questionnaire involves socio-demographic characteristics of patient's age, sex, occupation, educational status, marital status, residence and monthly income. The second part includes about life style of patients alcohol consumption, physical exercise, dietary plan adherence and home glucometer usage. The third part involves clinical characteristics of patients, type of DM, type of medication, history of diabetic complication, hypertension as co morbidity were collected by review of medical card and duration of diabetes from time of diagnosis and family history were collected by face to face interview. Medication adherence was assessed by modified eight-item Morisky medication adherence scale.

4.7.2 Anthropometrical Measurements

Weight and height of study participants were measured by using calibrated FAZZINI scale S7200HR, Vidmodrone (MI), Italy with height rod as the patients wore light clothe and no shoes. BMI was calculated from the body weight (kg) and height (m) by dividing weight to height square. WC was measured over light clothing at the level halfway between the iliac crest and the costal margin in the mid-axillary line after exhaling at standing position with the body weight evenly distributed across the feet. HC was measured over light clothing at widest portion of buttocks as the participants in standing position and both feet together. Two consecutive recordings were made using a non-stretchable fiber tape on a horizontal plane without compression of skin. The averages of two measurements were used. Waist-to-hip ratio (WHR) was calculated by dividing WC to HC. While the cut-off point considered for WC ≥80 cm for

females and ≥94 cm for males to define overweight, the cut-off value taken for WHR was >0.8 for females and >0.9 for males as per the criterion of the WHO (59,60).

4.7.3 Laboratory Analysis and Biochemical Test

The study participants were asked for informed consent to be interviewed and to give blood sample. About 5 mL blood was withdrawn from each study participant who was fasting overnight. Three ml of blood was collected in gel separator tube to determine the levels of TC, HDL-C, LDL-C, TG, fasting blood glucose in milligram per deciliter and 2ml of blood was collected in EDTA tube to determine hemoglobin A_1c in percent by qualified laboratory professionals. Cobas 4000 series Cobas c311 (Basel, Switzerland) analyzer were used. The abnormal results on lipid profile, fasting blood glucose and hemoglobin A_1C results were reported immediately to the physician.

Test principles of the laboratory analytes

Determination of Fasting Blood Glucose Level

Cobas 4000 series Cobas c311(Basel, Switzerland) chemistry analyzer used to determine the second and third blood glucose by hexokinase method ,but the first fasting blood glucose determined by mindray BS-200E(Shenzhen, China Mindray Bio-Medical Electronics Co., Ltd.) chemistry analyzer by glucose oxidase method because Cobas 4000 series Cobas c311 was not available.

Principle: Oxidation of glucose by glucose oxidase generates D-gluconolactone and hydrogen peroxide. The hydrogen peroxide reacts with phenol and 4- aminoantipyrine in the presence of the enzyme, peroxidase, to form a colored quinoid dye product. The absorbance of colored product was proportional to the glucose concentration in the sample and measured at 546 nm. Hexokinase catalyzes the phosphorylation of glucose to glucose-6-phosphate by ATP. Glucose-6-phosphate dehydrogenase oxidizes glucose-6-phosphate in the presence of NADP to gluconate-6-phosphate. No other carbohydrate is oxidized. The rate of NADPH formation during the reaction is directly proportional to the glucose concentration.

Lipid-profile: Basic lipids that are measured in the laboratory include total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol. They were measured by Cobas 4000 series Cobas c311 clinical chemistry analyzer. It is an automated system which follows principle of electrometric (ion-selective electrode) and photometric to perform variety of tests.

Principle of total cholesterol: An enzymatic colorimetric test method used to determine cholesterol in human serum. Cholesterol esters were break down by cholesterol esterase to release free cholesterol and fatty acids then cholesterol oxidized by cholesterol oxidase to cholest-4-en-3-one and hydrogen peroxide. In the presence of peroxidase, hydrogen peroxide coupled with phenol and 4-aminophenazone to form a red quinoneimine dye. The color intensity of the dye formed is directly proportional to the cholesterol concentration. It is determined by measuring increase in absorbance.

Principle of triglyceride: Triglycerides are hydrolyzed by lipoprotein lipase to glycerol and fatty acids. Glycerol is phosphorylated to glycerol-3-phosphate in ATP requiring reaction by glycerol kinase. Glycerol-3-phosphate is oxidized by glycerol phosphate oxidase to form dihydroxyacetone phosphate and hydrogen peroxide. In the presence of peroxidase, hydrogen peroxide affects the oxidative coupling of 4-chlorophenol and 4-aminophenazone to form a red-colored dye. The increase in absorbance is directly proportional to the concentration of triglycerides in the sample.

Principle of HDL-C: Homogeneous enzymatic colorimetric method is used. In the presence of magnesium ions, dextran sulfate selectively forms water-soluble complexes with LDL, VLDL, and chylomicrons which are resistant to PEG-modified enzymes. The cholesterol concentration of HDL-cholesterol is determined enzymatically by cholesterol esterase and cholesterol oxidase coupled with PEG to the amino groups. Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by cholesterol esterase. In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to delta 4-cholestenone and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-amino-antipyrine and HSDA (Sodium N-(2-hydroxy-3-sulfopropyl)-3, 5-dimethoxyaniline) to form a purple-blue dye. The color intensity of the blue dye formed is directly proportional to the HDL-cholesterol concentration. It is determined by measuring the increase in absorbance photo metrically.

Principle of LDL-C: Homogeneous enzymatic colorimetric assay used for determination of LDL-cholesterol. LDL-cholesterol selectively solubilized by a nonionic detergent. In the presence of Mg++ a sugar compound markedly reduces the enzymatic reaction of the cholesterol measurement in VLDL and chylomicrons. The combination of a sugar compound with detergent enables the selective determination of LDL-cholesterol in serum. In the presence of cholesterol esterase, cholesterol esters quantitatively break down into free cholesterol and fatty acids. In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to delta 4-cholestenone and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-aminoantipyrine and HSDA to form a purple-blue dye. The color intensity of this dye is directly proportional to the cholesterol concentration and is measured photo metrically.

Hemoglobin A_1c test measurement

Principle: The HbA1c determination is based on the turbidimetric inhibition immunoassay (TINIA). This method uses TTAB (Tetradecyltrimethylammonium bromide) as the detergent in the hemolyzing reagent to eliminate interference from leukocytes. Sample pretreatment to remove labile HbA1c is not necessary. All hemoglobin variants which are glycated at the β-chain N-terminus and which have antibody-recognizable regions identical to that of HbA1c are determined by this assay.

4.8. Data Quality Assurance

Data quality of questionnaire, laboratory investigation and anthropometric measurements were assured by following mechanism, checking consistence of questionnaire meaning as English version translated to Amharic then to Hadiysa and back translation to English by language expert. Training was given for data collectors.

To maintain quality of laboratory data daily maintenance of clinical chemistry analyzer was done, standard operating procedures were followed during specimen collection and processing of sample, daily quality controls were done for lipid profile, fasting blood glucose and hemoglobin A_1c tests before running patient sample. Expiration date for all reagents were checked, calibration was done for new reagent lot number and all the laboratory process was supervised by principal investigators. To keep quality of anthropometric measurements calibrated weight

and height measuring instrument was used and non stretchable fiber tape was used to measure hip circumference and waist circumference. Further more data were entered in epidata version 3.1, which improve data quality.

4.9. Statistical Analysis

After checking the data for completeness, missing values and coding of the questionnaires. The data were entered into Epi data version 3.1(Epi-Data, Odense, Denmark) and analyzed by using Statistical Package for Social Science (SPSS) version 20 (IBM, Chicago, IL, U.S.A). Continuous and categorical variables were summarized by tables, graph and descriptive statistics. Logistic regression was used to determine factors associated with poor glycemic control and dyslipidemia. Bivariate analysis was performed for independent variables to select candidate variable for multivariable logistic regression. The variables with p-value <0.25 were taken to multivariable logistic regression. The multivariable logistic regression was done to control effect of confounding variables and to identify associated risk factors with poor glycemic control and dyslipidemia.

Normality of data was checked by histogram and Shapiro-wilk test. Mann-whitney U test was used to compare lipid profile median and interquartile range between good glycemic and poor glycemic control status. Spearman's rank correlation was done for lipid profile and average fasting blood glucose with hemoglobin A_1c. 95% CI and P-value <0.05 was considered as statistically significant. Adjusted odd ratio was used to determine strength of association of predictors. Age and duration of diabetes from time of diagnosis were categorized based on previous study (37).Monthly income of the participants categorized based on previous study(46).

4.10. Ethical Consideration

Ethical clearance was obtained from the Institutional review Board of Health Sciences faculty, Jimma University. Letter of cooperation was written from Jimma university research coordination office to WUNEMMRTH. The purpose, benefit and method of the study were clearly explained to participant with the language they can understand. All of participants were informed that, their response would be kept confidential. The written informed consent was taken from the participants and those who have willingness to participate in the study was

included (Annex IV). Participation in the study was voluntary and refusal was permitted. To ensure confidentiality of data, study participants were identified using codes and unauthorized persons were not able to access the collected data. In addition, the clinical specimen collected during the study period was used for the stated objectives only. The study participants' result was reported to the physician for proper management as necessary**.**

4.11. Plans for Dissemination and Utilization of Results

The findings will be presented to Jimma University, Faculty of Health Science, School of Medical Laboratory Science and WUNEMMRTH. It will also be disseminated through publication on peer reviewed scientific journals and presented on scientific conferences. The study finding and recommendation will be given to Nigist Ellen Mohammed Memorial and Teaching hospital. The copy of the result will be submitted to Jimma University Faculty of Health Science.

Chapter Five: Results

5.1 Socio-Demographic Characteristics of Study Participants

A total of 320 diabetic patients were recruited to participate in this study of which 307(96%) provided complete information. From 307 diabetic patients 280(91.2%) were type 2 and 27(8.8%) patients were type1. The majority of the study participants were male 201(65.5%). The mean age of the study participants were 47.44±13.3 years. Most of the type 2 diabetic patients were in age range of 45-64(48.5%). Majority of the study participants were married 252(82.1%), urban residents 213(69.4%) and had monthly income 268(87.3%) >1000ETB. Sociodemographic factors are summarized in (Table 1).

5.2 Clinical and Behavioral Characteristics

From the study participants 87(28.3%) had history of diabetic complication. Majority of the complication was retinopathy 54 (17.6%) followed by nephropathy 22(7.2%). Majority of the study participants did not follow dietary plan 227 (73.9%), didn't perform regular physical exercise 289(94.1%) and not perform self glucose monitoring 294(95.8%). Duration of diabetes from the time of diagnosis less than seven years was 213(69.4%) and greater than or equal to seven years were 94(30.6%). Family history of diabetes observed on 54(17.6%) and hypertension as co morbidity reported by 58(18.9%) study participants. Most of the patients 193(62.9%) were treated by oral glucose lowering agents only. Ninety six (31.3%) patients were treated by insulin only and 18(5.9%) treated by combination of oral glucose lowering agents with insulin. Medication adherence of patients were good for 164(53.4%), moderate 90(29.3%) and poor 53(17.3%). Majority of the patients had abnormal lipid profile on triglyceride and high density lipoprotein 239(78.9%), 129(42%) respectively (Table2, 3).

Table 1:Sociodemographic characteristics, glycemic control and dyslipidemia of DM patients attending at Wachemo University Nigist Elleni Mohamed memorial referral and teaching hospital, Hossana, Southern Ethiopia, May 1 to June 30, 2020.

Variables		N (%)	Glycemic control		Dyslipidemia	
			Good (%)	Poor (%)	Yes (%)	No (%)
Age in years	<25	20 (6.5)	5(25)	15(75)	15(75)	5(25)
	25-44	102(33.2)	21(20.6)	81(79.4)	89(87.3)	13(12.7)
	45-64	149(48.5)	22(14.8)	127(85.2)	131(87.9)	18(12.1)
	≥65	36(11.7)	6(9.5)	30(80.5)	31(86.1)	5(13.9)
Sex	Male	201(65.5)	36(17.9)	165(82.1)	174(86.6)	27(13.4)
	Female	106(34.5)	18(17)	88(83)	92(86.8)	14(13.2)
Educational status	No formal education	66(21.5)	7(10.6)	59(89.4)	54(81.8)	12(18.2)
	Primary(1-8)	79(25.7)	14(17.7)	65(82.3)	67(84.8)	12(15.2)
	Secondary(9-12)	62(20.2)	6(9.7)	56(90.3)	56(90.3)	6(9.7)
	Diploma and above	100(32.6)	27(27)	73(73)	89(89)	11(11)
Marital status	Single	34(11.1)	9(26.5)	25(73.5)	26(76.5)	8(23.5)
	Married	252(82.1)	41(16.3)	211(83.7)	224(88.9)	28(11.1)
	Divorced	14(4.6)	2(14.3)	12(85.7)	12(85.7)	2(14.3)
	Widowed	7(2.3)	2(28.6)	5(71.4)	4(57.1)	3(42.9)
Residence	Urban	213(69.4)	40(18.8)	173(81.2)	188(88.3)	25(11.7)
	Rural	94(30.6)	14(14.9)	80(85.1)	78(82.9)	16(11.1)
Occupation	Government employed	95(30.9)	25(26.3)	70(73.7)	83(87.4)	12(12.6)
	Housewife	65(21.2)	7(10.8)	58(89.2)	58(89.2)	7(10.8)
	Merchant	69(22.5)	13(18.8)	56(82.2)	58(84)	11(16)
	Farmer	53(17.3)	4(7.5)	49(92.5)	48(90.5)	5(9.5)
	Other	25(8.1)	5(20)	20(80)	19(76)	6(24)
Monthly income	≤1000ETB	30(9.8)	5(16.7)	25(83.3)	24(80)	6(20)
	>1000ETB	268(87.3)	47(17.5)	221(82.5)	235(87.7)	33(12.3)
Type of DM	Type1	27(8.8)	4(14.8)	23(85.2)	21(77.8)	6(22.2)
	Type2	280(91.2)	50(17.9)	230(82.1)	245(87.5)	35(12.5)

Note: *ETB: Ethiopian Birr, DM: diabetes mellitus, other (NGO Worker, student and private worker).*

Table 2:Clinical and Behavioral Characteristics of DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern Ethiopia, May 1 to June 30, 2020.

Variables		N (%)	Glycemic control		Dyslipidemia	
			Good (%)	Poor (%)	Yes (%)	No (%)
History of complication	Yes	87(28.3)	3(3.4)	84(96.6)	75(86.2)	12(13.8)
	No	220(71.7)	51(23.2)	169(76.8)	191(86.8)	29(13.2)
Dietary plan adherence	Yes	80(26.1)	22(27.5)	58(82.5)	71(88.7)	9(11.3)
	No	227(73.9)	32(14)	195(86)	195(85.9)	32(14.1)
Physical Exercise	Yes	18(5.9)	11(61.1)	7(38.9)	12(66.7)	6(33.7)
	No	289(94.1)	43(14.9)	246(85.1)	251(86.9)	38(13.1)
Glucometry usage	Yes	13(4.2)	9(69.2)	4(30.8)	8(61.5)	5(38.5)
	No	294(95.8)	45(15.3)	249(84.7)	258(87.8)	36(12.2)
Alcohol drinking	Yes	8(2.6)	1(12.5)	7(87.5)	8(100)	0(0)
	No	299(97.4)	53(17.7)	246(81.3)	258(86.2)	41(13.8)
Duration of DM	<7 Years	213(69.4)	47(22)	166(78)	185(86.9)	28(13.1)
	≥7 Years	94(30.6)	7(7.4)	87(92.6)	81(86.2)	13(13.8)
Micro-complication	Retinopathy	54(17.6)	2(3.7)	52(96.3)	47(87)	7(13)
	Neuropathy	4(1.3)	1(25)	3(75)	4(100)	0(0)
	Nephropathy	22(7.2)	0(0)	22(100)	18(81.8)	4(18.2)
Macro-complication	Coronary arterydisease	4(1.3)	0(0)	4(100)	3(75)	1(25)
	Peripheral arterydisease	3(0.97)	0(0)	3(100)	3(100)	0(0)
Family history of DM	Yes	54(17.6)	10(18.5)	44(81.5)	45(83.3)	9(16.7)
	No	253(82.4)	44(17.4)	209(82.6)	221(87.4)	32(12.6)
Hypertension as co morbidity	Yes	58(18.9)	6(10.3)	52(89.7)	51(87.9)	7(12.1)
	No	249(81.1)	48(19.3)	201(81.7)	215(86.3)	34(13.7)
Type of medication	Insulin only	96(31.3)	13(13.5)	83(86.5)	81(84.4)	15(15.6)
	OHA	193(62.9)	30(15.5)	163(84.5)	170(88)	23(12)
	Insulin and OHA	18(5.9)	11(61.1)	7(39.9)	15(83.3)	3(16.7)
Medication Adherence	Good	164(53.4)	36(22)	128(78)	139(84.8)	25(15.2)
	Moderate	90(29.3)	12(13.3)	78(86.7)	80(89.9)	10(11.1)
	Poor	53(17.3)	6(11.3)	47(88.7)	47(88.7)	6(11.3)

Table 3:Lipid profile and Anthropometric measurement of DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern Ethiopia, May 1 to June 30, 2020.

Variables		N (%)	Good glycemic control N (%)	Poor glycemic control N (%)
Cholesterol	≤200 mg/dl	223(72.6)	47(21)	176(79)
	>200 mg/dl	84(27.4)	7(8.3)	77(91.7)
TG	≤150 mg/dl	68(22.1)	19(27.9)	49(72.1)
	>150mg/dl	239(78.9)	35(14.6)	204(85.4)
HDL-C	≥40 mg/dl	178(58)	35(19.7)	143(80.3)
	<40 mg/dl	129(42)	19(14.7)	110(85.3)
LDL-C	≤130mg/dl	211(68.7)	44(20.9)	167(79.1)
	>130 mg/dl	96(31.3)	10(10.4)	86(89.6)
BMI	18.5-24.9	159(51.8)	28(17.6)	131(82.3)
	25-29.99	118(34.4)	22(18.6)	96(81.4)
	≥30	30(9.8)	4(13.3)	26(86.7)
WHR	≥0.90 for male	116(37.8)	21(18.1)	95(81.9)
	≥0.8 for female	56(18.2)	5(8.9)	51(91.9)

5.3 Prevalence of Poor Glycemic Control among study participants

The mean ± SD value of hemoglobin A_1c and average fasting blood glucose level were 8.77±2.32% and 193±59 mg/dl respectively. The overall prevalence of poor glycemic control based on hemoglobin A_1c and average fasting blood glucose were 253(82.4%) and 212(69.1%) respectively. From study participants 17.6% had good glycemic control, 30.9% had satisfactory glycemic control and 51.5% had unsatisfactory glycemic control based on hemoglobin A_1c. Poor glycemic control was predominant among age group 45-64,127(85.2%), married 211(83.7%), rural residence 80 (85.1%), participants who had history of diabetic complication 84(96.6%),

duration ≥7 years 87(92.6%), poor medication adherence 47(88.7%), cholesterol >200mg/dl 77(91.7%) and LDL-C >130mg/dl 86(89.6%) (Table 1, 2, 3) and figure 2.

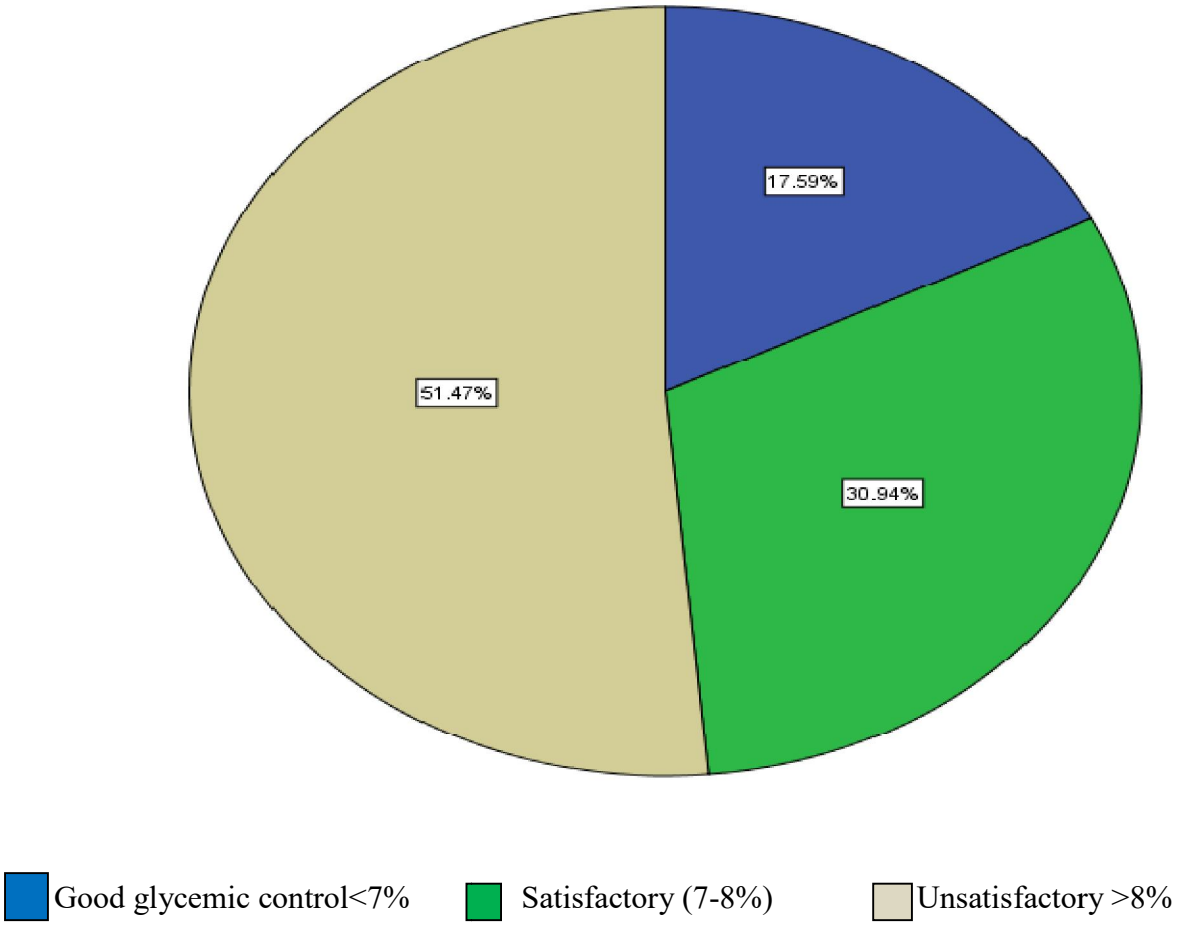

Figure 2: Degree of glycemic control status among study participants, HbA_1c < 7% is good glycemic control and HbA1c ≥7% is poor glycemic control which includes satisfactory and unsatisfactory glycemic control at Wachemo University Nigist Elleni Mohammed Memorial Teaching and Referral Hospital from may 1 to June 30, 2020.

5.4 Prevalence of dyslipidemia among study participants

The overall prevalence of dyslipidemia among study participants was 266(86.6%). Dyslipidemia was higher in type2 diabetic patients, 245(87.5%) than type1 diabetic patients, 21(77.8%). Majority of study participants had high triglyceride 239(78.9%) and low high density lipoprotein cholesterol, 129(42%) (Table1, 2).

5.5 Factors associated with glycemic control status.

In bivariate analysis ,educational status, occupation, history of diabetic complication ,dietary adherence, physical exercise , home glucometer usage , hypertension as co morbidity, type of medication, medication adherence, waist to hip ratio and dyslipidemia were identified as the candidate variables for multivariable logistic regression with p-value <0.25 (Table 4,5).

Table 4:- Bivariate logistic regression analysis of sociodemographic factors associated to poor glycemic control among DM patients attending at Wachemo University Nigist Elleni Mohammed memorial Referral and Teaching Hospital, Hossana, Southern, Ethiopia

Variables		N	Glycemic control status		COR(95% CI)	P-Value
			Good (%)	Poor (%)		
Age in years	<25	20	5(25)	15(75)	Ref	0.531
	25-44	102	21(20.6)	81(79.4)	1.286(0.419-3.941)	0.660
	45-64	149	22(14.8)	127(85.2)	1.924(0.635-5.831)	0.247
	≥65	36	6(9.5)	30(80.5)	1.667(0.437-6.358)	.0455
Sex	Male	201	36(17.9)	165(82.1)	Ref	
	Female	106	18(17)	88(83)	1.067(0.573-1.987)	0.839
Educational status	No formal education	66	7(10.6)	59(89.4)	3.117(1.268-7.662)	0.013
	Primary(1-8)	79	14(17.7)	65(82.3)	1.717(0.83-3.552)	0.145
	Secondary(9-12)	62	6(9.7)	56(90.3)	3.452(1.334-8.932)	0.011
	Diploma and above	100	27(27)	73(73)	Ref	0.015
Marital status	Single	34	9(26.5)	25(73.5)	Ref	0.429
	Married	252	41(16.3)	211(83.7)	1.853(0.806-4.258)	0.146
	Divorced	14	2(14.3)	12(85.7)	2.16(0.403-11.586)	0.369
	Widowed	7	2(28.6)	5(71.4)	0.9(0.148-5.489)	0.909
Residence	Urban	213	40(18.8)	173(81.2)	Ref	
	Rural	94	14(14.9)	80(85.1)	1.321(0.680-2.566)	0.411
Occupation	Government Employed	95	25(26.3)	70(73.7)	Ref	0.038
	Housewife	65	7(10.8)	58(89.2)	2.959(1.194-7.333)	0.019
	Merchant	69	13(18.8)	56(82.2)	1.538(0.722-3.279)	0.265
	Farmer	53	4(7.5)	49(92.5)	4.375(1.432-13.36)	0.010
	Other	25	5(20)	20(80)	1.429(0.485-4.212)	0.518
Monthly income	≤1000ETB	30	5(16.7)	25(83.3)	Ref	
	>1000ETB	268	47(17.5)	221(82.5)	0.94(0.342-2.583	0.905
Type of DM	Type1	27	4(14.8)	23(85.2)	Ref	
	Type2	280	50(17.9)	230(82.1)	0.8(0.265-2.415)	0.692

Note: *Ref: reference, ETB: Ethiopian Birr*

Table 5:- Bivariate logistic regression analysis of clinical, behavioral and anthropometric factors associated to poor glycemic control among DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern, Ethiopia, May 1 to June 30, 2020.

Variables		N	Glycemic control status		COR(95% CI)	P-Value
			Good (%)	Poor (%)		
History of complication	Yes	87	3(3.4)	84(96.6)	8.45(2.562-27.87)	0.001
	No	220	51(23.2)	169(76.8)	Ref	
Dietary plan adherence	Yes	80	22(27.5)	58(82.5)	Ref	
	No	227	32(14)	195(86)	2.311 (1.247- 4.284)	0.008
Physical Exercise	Yes	18	11(61.1)	7(38.9)	Ref	
	No	289	43(14.9)	246(85.)	8.99 (3.302-24.474)	0.001
Glucometry usage	Yes	13	9(69.2)	4(30.8)	Ref	
	No	294	45(15.3)	249(84.)	12.45(3.676-42.16)	0.001
Alcohol drinking	Yes	8	1(12.5)	7(87.5)	1.508 (0.182-12.517)	0.704
	No	299	53(17.7)	246(81.3)	Ref	
Duration of DM	<7 Years	213	47(22)	166(78)	Ref	
	≥7 Years	94	7(7.4)	87(92.6)	3.519(1.526-8.113)	0.003
Family history of DM	Yes	54	10(18.5)	44(81.5)	Ref	
	No	253	44(17.4)	209(82.6)	1.08 (0.505-2.308)	0.843
Hypertension as co morbidity	Yes	58	6(10.3)	52(89.7)	2.07 (0.84-5.1)	0.114
	No	249	48(19.3)	201(81.7)	Ref	
Type of medication	Insulin only	96	13(13.5)	83(86.5)	10.033 (3.295-30.55)	0.001
	OHA	193	30(15.5)	163(84.5)	8.538 (3.065-23.785)	0.001
	Insulin and OHA	18	11(61.1)	7(39.9)	Ref	0.001
Medication Adherence	Good	164	36(22)	128(78)	Ref	0.117
	Moderate	90	12(13.3)	78(86.7)	1.649 (0.839-3.24)	0.147
	Poor	53	6(11.3)	47(88.7)	2.611 (0.874-7.798)	0.086
BMI	18.5-24.9	159	28(17.6)	131(82.3)	Ref	0.794
	25-29.99	118	22(18.6)	96(81.4)	0.933 (0.503-1.729)	0.825
	≥30	30	4(13.3)	26(86.7)	1.389 (0.449-4.297)	0.568
WHR					9.326 (0.508-171.16)	0.133
WC					1.013 (0.991-1.035)	0.255
Dyslipidemia	Yes	266	41(15.4)	225(84.6)	2.548(1.219-5.325)	0.013
	No	41	13(31.7)	28(68.3)	Ref	

5.6 Multivariable Logistic Regression Analysis of Predictors of Poor Glycemic Control.

Candidate variables for multivariable logistic regression model were educational status, occupation, history of complication ,dietary adherence, physical exercise , duration of DM from time of diagnosis, home glucometer usage , hypertension as co morbidity, type of medication, medication adherence, waist to hip ratio and dyslipidemia by considering p-value <0.25(Table 4 and 5). In multivariable logestic regression model secondary school educational status, history of diabetic complication, home glucometer usage, physical exercise, duration of DM from time of diagnosis, type of medication and dyslipidemia were significantly associated with glycemic control status (Table6).

The patients who had history of diabetic complication were 7 times more likely have poor glycemic control as compared to the patients without history of complication(AOR: 7.093,95%CI 1.725-29.163). The patients who lack home glucometer usage were 8.3 times more likely have poor hemoglobin A_1c when compared to the patents who had self monitoring of their blood glucose at home (AOR: 8.276,95%CI 1.613-42.461).The patients who had no habit of performing regular exercise for 30 minutes ≥three days per week were 5.5 times more likely have poor glycemic control as compared to the patients who had the habit of performing regular exercise for 30 minutes ≥three days per week(AOR: 5.503, 95%CI 1.601-18.908).

Patients who had duration of DM ≥7 years were 4 times more likely have poor glycemic control as compared to the patients who had <7 years(AOR: 4.096,95%CI 1.388-12.089).The patients on oral hypoglycemic agents only were 1.2 times more likely have poor glycemic control as compared to the patients on insulin only(AOR: 1.160, 95% CI 0.534-2.520). Patients on both insulin and oral hypoglycemic agents revealed that the odds of developing poor glycemic control were lower as compared to the patients on insulin only(AOR: 0.106, 95% CI 0.025-0.444). Patients who had dyslipidemia were 2.7 times more likely have poor glycemic control as compared to patients who had no dyslipidemia (AOR: 2.743,95%CI 1.129-6.662). Eventhougth medication adherence was not significantly associated in this study. Patients who had poor medication adherence were 2 times more likely have poor glycemic control as compared to patients who had good medication adherence(AOR: 2.131 95%CI 0.546-8.318).

Table6:Multivariable logistic regression analysis of factors associated to poor glycemic control status among DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern, Ethiopia, May 1 to June 30, 2020.

Variables		N	Glycemic control status		AOR(95% CI)	P-Value
			Good (%)	Poor (%)		
Educational status	No formal education	66	7(10.6)	59(89.4)	2.645 (0.913-7.66)	0.073
	Primary(1-8)	79	14(17.7)	65(82.3)	1.503 (0.601-3.76)	0.384
	Secondary(9-12)	62	6(9.7)	56(90.3)	3.14(1.003-9.84)	0.049#
	Diploma and above	100	27(27)	73(73)	Ref	0.138
Occupation	Government Employed	95	25(26.3)	70(73.7)	Ref	0.909
	Housewife	65	7(10.8)	58(89.2)	0.646 (0.115-3.62)	0.620
	Merchant	69	13(18.8)	56(82.2)	0.511 (0.125-2.08)	0.349
	Farmer	53	4(7.5)	49(92.5)	0.703 (0.11-4.49)	0.709
	Other	25	5(20)	20(80)	0.617 (0.124-3.07)	0.556
History of complication	Yes	87	3(3.4)	84(96.6)	7.093 (1.725-29.16)	0.007#
	No	220	51(23.2)	169(76.8)	Ref	
Dietary plan adherence	Yes	80	22(27.5)	58(82.5)	Ref	
	No	227	32(14)	195(86)	1.627 (0.783-3.38)	0.192
Physical Exercise	Yes	18	11(61.1)	7(38.9)	Ref	
	No	289	43(14.9)	246(85.)	5.503 (1.601-18.9)	0.007#
Glucometry user	Yes	13	9(69.2)	4(30.8)	Ref	
	No	294	45(15.3)	249(84.)	8.276 (1.613-42.5)	0.011#
Duration of DM	<7 Years	213	47(22)	166(78)	Ref	
	≥7 Years	94	7(7.4)	87(92.6)	4.096 (1.388-12.1)	0.011#
Hypertension as co morbidity	Yes	58	6(10.3)	52(89.7)	1.379 (0.448-4.24)	0.576
	No	249	48(19.3)	201(81.7)	Ref	
	Insulin only	96	13(13.5)	83(86.5)	Ref	0.003#
Type of medication	OHA	193	30(15.5)	163(84.5)	1.16 (0.534-2.52)	0.709
	Insulin and OHA	18	11(61.1)	7(39.9)	0.106 (0.025-0.444)	0.002
Medication Adherence	Good	164	36(22)	128(78)	Ref	0.542
	Moderate	90	12(13.3)	78(86.7)	1.241 (0.559-2.76)	0.595
	Poor	53	6(11.3)	47(88.7)	2.131(0.546-8.32)	0.276
WHR					2.76(0.027-278.86)	0.666
Dyslipidemia	Yes	266	41(15.4)	225(84.6)	2.743(1.129-6.662)	0.026#
	No	41	13(31.7)	28(68.3)	Ref	

Note: *#: significant p-value in multivariable model, Ref: reference, OHA: oral hypoglycemic agents, WHR: waist to hip ratio and continuous data, AOR: adjusted odd ratio, CI: confidence interval.*

Table7:Multivariable logistic regression analysis of factor associated to dyslipidemia among DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern, Ethiopia, May 1 to June 30, 2020.

Variables		N	Dyslipidemia		COR(95%CI)	AOR(95%CI)	P-Value
			Yes (%)	No (%)			
Marital status	Single	34	26(76.5)	8(23.5)	Ref	Ref	0.157
	Married	252	224(88.9)	28(11.1)	2.462(1.016-5.96)	2.057(0.73-5.83)	0.174
	Divorced	14	12(85.7)	2(14.3)	1.846(0.339-10)	2.363(0.32-17.5)	0.399
	Widowed	7	4(57.1)	3(42.9)	0.41(0.075-2.232)	0.408(0.063-2.6)	0.347
Residence	Urban	213	188(88.3)	25(11.7)	Ref	Ref	
	Rural	94	78(82.9)	16(11.1)	0.648(0.328-1.281)	0.965(0.412-2.26)	0.934
Monthly income	≤1000ETB	30	24(80)	6(20)	Ref	Ref	
	>1000ETB	268	235(87.7)	33(12.3)	1.78(0.678-4.677)	0.954(0.318-2.86)	0.933
Type of DM	Type1	27	21(77.8)	6(22.2)	Ref	Ref	
	Type2	280	245(87.5)	35(12.5)	2(0.755-5.297)	1.67(0.134-3.1)	0.163
Glucometer usage	Yes	13	8(61.5)	5(38.5)	Ref	Ref	
	No	294	258(87.8)	36(12.2)	4.45(1.389-14.44)	4.36(1.285-14.77)	0.018#
BMI	<18.5-24.9	159	28(17.6)	131(82.3)	Ref	Ref	0.528
	25-29.99	118	22(18.6)	96(81.4)	3.33(1.47-7.545)	1.75(0.661-4.62)	0.260
	≥30	30	4(13.3)	26(86.7)	3.391(0.766-15.0)	1.49(0.206-10.8)	0.692
HC					1.037(1.010-1.066)	1.02(0.981-1.061)	0.321
WC					1.053(1.025-1.082)	1.064(1.031-1.09)	0.001#
Glycemic control	Good	54	41(76)	13(24)	Ref		
	Poor	253	225(89)	28(11)	2.548(1.219-5.325)	1.71(0.721-4.06)	0.223

Note: *Ref : reference ,#: significant p-value*

Glucometer usage and waist circumference were significantly associated with dyslipidemia. Patients who lack home glucometer usage were 4.5 times more likely had dyslipidemia as compared to home glucometer user. .As WC increase by one unit, developing dyslipidemia increase by factor 0.06

Chapter-Six: Discussion

Glycemic control is the basic for managing diabetic patients. It is essential to prevent and delay diabetes complications. Glucose measurement is major tool to improve glycemic control status(25).The fasting serum glucose and hemoglobin A_1c tests are important to determine glycemic control status. The American diabetic association standard was used to determine the cutoff value for poor glycemic control. For fasting mean blood glucose ≥154mg/dl is poor glycemic control in the same way for hemoglobin A_1c ≥7% is poor glycemic control for non pregnant adult patients(1). According to this standard the overall prevalence of poor glycemic control in this study were 82.4% for hemoglobin A_1c and 69.1% for the mean fasting blood glucose.

The overall prevalence of poor glycemic control in current study was 82.4%. This finding is similar with previous study conducted in Bangladesh (82%), India (76.6%),Asmara, Eritrea (76.7%) and Tikur Anbessa Specialized Hospital , Addis Ababa (80%) (25,29,35,46). Higher than study conducted across Europe (37.4%),Tanzania(69.7%), Tikur Anbessa specialized hospital (68.9%), Nekemte Referral Hospital(64.9%), Jimma university specialized hospital(59.5%) and Gondar (60.5%) (27,37,40,42,47,61). It is lower than studies done in South Africa (86.5%) and India (91.8%)(28,52).This variation might be due to the difference in the method of glucose measurement, sample size, cut point value, socioeconomic status, environmental factors and genetic factor which provoke participants to develop poor glycemic control.

The overall prevalence of dyslipidemia in current study was 86.6%. This finding is comparable with the study conducted in Nepal (88.1%), India (90%), Kenya (86.1%) and Nigeria (90.7%) (48,49,53,62). Lower than the study conducted in India (97.2%),South Africa(93.5%) and higher than the study conducted at Durame General Hospital, Ethiopia (65.6%) (50,52,54). This difference might be due to method of lipid profile measurement like estimation of low density lipoprotein cholesterol by Friedewald equation which excludes the triglyceride amount greater than 400mg/dl, sample size, socio-economic status and lifestyle.

The results of our study revealed that, patients on secondary school educational status, history of complication, home glucometer usage, physical exercise, duration of DM, type of medication and dyslipidemia were significantly associated with glycemic control status, but dietary plan adherence, medication adherence, occupation, hypertension as co morbidity and waist to hip ratio were not significantly associated with glycemic control status (Table 6). Factors like waist circumference and home glucometer usage were significantly associated with dyslipidemia, but BMI, type of DM, residence, marital status; monthly income and glycemic control were not significantly associated with dyslipidemia (Table 7).

The current study result showed that secondary school educational status had significant association with poor glycemic control. This might be due to most of type1 diabetic participants in this study were under 25 years and they were secondary school students as the result poor glycemic control was higher in type1 diabetic (85.2%) when compared to type 2 diabetic (82.1%).This finding is similar with study conducted in Venezuela(T1DM patients (87%) ,T2DM,75%), Gondar (T1DM, 61.4%, T2DM, 59.8%) (32,37).

Participants who had history of diabetic complication more likely had higher odds of having poor glycemic control as compared to who hadn't history of diabetic complication. In contrary to this finding the studies done in India and at Tikur Anbessa Specialized Hospital ,Ethiopia revealed that there were no significant association of diabetic complication and poor glycemic control(29,47). This variation might be due to method of glucose determination and ethnic factors, but major cause for diabetic complication is hyperglycemia which leads to advanced glycation end product formation, oxidative stress and atherosclerosis.

The result of current study revealed that the odds of developing poor glycemic control was higher in study participants who lack home glucometer usage when compared to the study participants who use home glucose monitoring. This finding is similar with study conducted in Jimma, Tigray (39,43). In contrary to this, the studies done in Saudi Arabia , Tanzania and Gondar revealed that there were no significant association on lack of home glucometer usage and poor glycemic control (37,63,64). This variation might be due to inappropriate use of glucometer at home and calibration problem of glucometer. The Canadian Diabetes Association guideline indicates that self-monitoring of blood glucose provides feedback on the results of

lifestyle, pharmacological treatments, and adherence to treatment. It also helps health professional to facilitate longer-term and shorter-term treatment modifications, for instance insulin dosing for people with type 1 or type 2 diabetes(65).

The study participants who didn't perform regular physical exercise at least 30minutes for three or more days in this study showed that the odds of developing poor glycemic control was higher when compared with those perform physical exercise. This finding is similar with the studies done in Saudi Arabia, Nekemte Referral Hospital, Gondar (31,36,40) this might be due to physical exercise increase glucose uptake , consumption of glucose in muscle ,decrease visceral fat, increases insulin sensitivity of receptors and reduce circulation of advanced glycation end product ,unlike this studies, physical exercise was not significantly associated with the study conducted in Tanzania(64) ,but I couldn't get multivariable logistic regression result ,so this variation might be not controlling of confounders.

The respondents with duration of DM ≥ 7 years in this study revealed that the odds of developing poor glycemic control were higher when compared to duration less than seven years. This finding is in agreement with studies conducted in across Europe ,Jordan and India (27,29,66). This might be due to gradual impairment of insulin secretion by beta cell as result of compensatory response of hyperglycemia, increase in insulin resistance and beta cell of pancreas may be damaged. Unlike these findings the study conducted in Sudan revealed that there was no significant association with longer duration and developing poor glycemic control (34). This variation might be due to Sociodemographic and ethnic characteristics.

Types of antidiabetic medication were also predictor for poor glycemic control in current study. Patients on oral hypoglycemic agents only revealed that there was no significant association with poor glycemic control when compared to patients on insulin only, but patients on both oral hypoglycemic agents and insulin showed that the odds of developing poor glycemic control was more likely lower when compared to the patients on insulin only. This finding is similar with the study done in Dessie, Ethiopia ,which revealed that the odds of developing poor glycemic control more likely higher in patients on oral hypoglycemic agents when compared to the patients on both oral hypoglycemic and insulin (45). This might be due to synergistic effect of oral hypoglycemic agents and insulin because some oral hypoglycemic agents increase insulin

secretion and tissue response to glucose. Another study done in Jimma showed an agreement with present study which stated that there was no significant association with patients on insulin only when compared to patients on oral hypoglycemic agents only(38). In contradiction to the latter finding the study done in Bangladesh revealed that there was significant association patients on insulin only and had higher odds of developing poor glycemic control when compared to the patients on oral hypoglycemic agents only(25). In addition to this the study conducted in Saudi Arabia and Mekelle, Ethiopia revealed that there were no significant association between types of medication and poor glycemic control (44,63).This variation might be due to glucose measurement method, not following glycemic control treatment algorithm because most of diabetic patients are not aware of their diabetes at time of diagnosis as the result their hemoglobin A_1c value much higher at the time of diagnosis and many hospital use fasting blood glucose which has higher intera individual variation.

Dyslipidemia in the current study was significantly associated with poor glycemic control Patients with dyslipidemia had higher odds of developing poor glycemic control when compared with non dyslipidemia. This finding is similar with the study performed in China, total cholesterol and high density lipoprotein cholesterol showed significant association with poor glycemic control (67). In line with these findings the study conducted in Eastern Sudan revealed patients with abnormal cholesterol and triglyceride had higher odds of developing poor glycemic control(34). In contradiction to these findings the study conducted in Jimma revealed that there was no significant association between dyslipidemia and developing poor glycemic control(47). This difference might be due to method of lipid profile measurement and using results from records rather than using active lipid measurement.

Eventhougth medication adherence was not significantly associated in the current study; the results suggested that the odds of developing poor glycemic control were higher on patients with moderate medication adherence and poor medication adherence when compared with patients with good medication adherence. The study carried out across Europe revealed that there was significant association in patients with poor medication adherence and poor glycemic control(27). Another study done in Gondar revealed that the odds of developing poor glycemic control more likely higher on patients with poor medication adherence as compared to patients

with good medication adherence(36). The absence of significant association in current study might be due to using self respondent questionnaires which was highly exposed to recall bias.

Waist circumference of the study participant was significantly associated with dyslipidemia in current study which was similar with study done in China(51). In contrary to this, studies done in Tanzania and Asmara, Eritrea revealed that there was no significant association between waist circumference and dyslipidemia(15,35). This variation might be due to sample size and life style.

Home glucometer usage was also predicator in current study for developing dyslipidemia. Patients who lack home glucometer usage had higher odds of developing dyslipidemia as compared to home glucometer user. Self-glucose monitoring helps patient to evaluate their own response to therapy and assess whether glycemic targets are achieved(24). Good glycemic control reduces dyslipidemia by decreasing circulating very-low-density lipoprotein, up regulation of LDL receptors and decrease efflux of fatty acid from adipose tissue(68).

6.2 Strength and Limitation of the Study

Since the glycemic control status was determined by hemoglobin A_1c. It is gold standard test for glycemic control management. In addition to this the instrument used to investigate lipid profile, hemoglobin A_1c and blood glucose was Cobas 4000sreies Cobas c311 which was more accurate and sensitive. The limitation of this study was the nature of cross-sectional study design which does not show causality relationship of predictors and outcome variable. Not checking of conditions affect red blood cell turnover such as hemoglobinopathy and using of self response questionnaire to measure medication and diet adherence which is highly exposed to recall bias.

Chapter Seven: Conclusion and Recommendation

7.1 Conclusion:

We summarized that high proportion of diabetic patients had poor glycemic control and dyslipidemia. Longer duration of DM, lack of home glucometer usage and dyslipidemia were significant predictors for poor glycemic control. Waist circumference and lack of home glucometer usage were also significantly associated with dyslipidemia.

7.2 Recommendation:

The finding of present study revealed that the prevalence of poor glycemic control and dyslipidemia were very high. It is known that poor glycemic control leads the patient to micro vascular complication or macro vascular complication. Based on findings we recommended that hemoglobin A1c test to be performed for all diabetic patients at least twice a year because the fasting blood glucose is underestimating the prevalence of poor glycemic control. The health professionals should advice the patients to have practice of home glucometer usage and to perform regular physical exercise. Lipid profile should be checked for all diabetic patients and physicians who give care for diabetic patients should follow treatment algorithm for poor glycemic control. Further longitudinal studies should be conducted on large population to identify predictors for poor glycemic control after a period of time.

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Annexes

Annex I: Information Sheet

English Version

Title of the project: Lipid profile and glycemic control among diabetes mellitus patients attending Wachemo Unversity Nigist Ellen Mohammad Memorial Referral and teaching Hospital, Hosanna, southern Ethiopia.

Name of Principal Investigator: Ageze Abose

Organization: Jimma University (School of Medical Laboratory Science)

Name of sponsor organization: Wachemo University

This information sheet was prepared for adult diabetic patients who visit chronic care clinic at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital who were involved on the study mentioned above.

Description and Purpose of the study

Diabetes mellitus is a hormonal disorder characterized by hyperglycemia. Hyperglycemia is the major problem that causes vascular complication in diabetic patient. It is global public health problem which affect both developing and developed countries with major consequence on human health and socioeconomic status. Glycemic control and lipid profile are basic in diabetic management. Plenty of factors are responsible for the occurrence of poor glycemic control and dyslipidemia among diabetic patient, therefore the present study was designed to assess lipid profile and glycemic control and associated factors among diabetic patients at Wachemo University Nigist Ellen Mohammad Memorial Referral Hospital from May1 to June30, 2020.

Procedures: In order to undertake the above-mentioned study, some questions related with the topic; socio-demographic characteristics, clinical data, behavioral data and anthropometric measurements were taken. Informed consent was obtained from each study participant and venous blood specimen and related data were taken from study participant for laboratory

investigation. The collected sample was processed in Wachemo University Nigist Ellin Mohammad Memorial Referral and Teaching Hospital Central Laboratory.

Risks associated to study: There was no any possible risk but there is little pain and discomfort during venous blood sample collection. All samples were collected following Standard operational procedures.

Benefits and Compensation from the study: There was no direct financial benefit for participating in this study. Based on the Laboratory result they were referred to the physician for further care and treatment.

Confidentiality: The information obtained during this study was kept confidential. Disclosure of data to third parties other than those allowed in the informed consent was not permitted. Records were remained confidential. To maintain confidentiality, the investigator kept records in safe way and results of the tests were coded to prevent identification of the volunteers. Collected samples were not used for other research purposes and safely disposed of after the completion of the study.

Right to refuse or withdraw: Free to withdraw from the study at any time and were not discriminated in any form of health services due to refusal.

Contact information: If you have any question about this study, you can contact principal investigator and advisors for further information any time by following address:

Mr. Ageze Abose, Phone No +251-920993169, Email: ageabose12@gmail.com

Mr.Shiferew Bekele No+251-917233710, Email bekeleshiferaw@yahoo.com

Mr.Aklilu Getachew phone NO +251911743331 Email :akachew.2@gmail.com

Mr. Fanta Obsa Phone No +251917001033 Email: sinbona2006@gmail.com

Annex II: Information Sheet Hadiyissa Version

Sorophii qoxoi woshi; Wachaami Yunvursitena Nigiste Ellinii Mohammad Matasabeei Hospitalena sukal jabii hikimina qerral amane awonom manane orachi dili qaxoma eesam xiqi woro yoo sukal qaxoma egeramakoda laimma.

Wonii sarayanchi: Agaza Abosse

Haramuki kitaphii mini: Wachaami Yunvursitee/Labratoorii Losanni Mine

Kuhi woshii kitabi gudukoki hanani kuramuki sorrobane bikkokik manina ihukuyyaa lulomanemii kahi sorrobimane hasisokii luwuwwaa kuriminate . kaahi sorobane bikokii mani hundemi ixxi hasenina ihoisa chakinsammo.

Sorophii woni woshii: Sukal jabii hikimina qerral amane awonom manane orachi dili qaxoma eesam xiqi woro yoo sukal qaxoma egeramakoda laimina wachaami unvursitena nigiste ellinii mohammad sawaisanchi hospitalena sorrobima

Sorophii fintouwaa

Sorrobane bikokimina itamuki manisi woroni yokii woshuwaa xigga masinomisa kullamo.

- Fayyommi mazigabaa monomissa
- Oddimi 10 dakikina xamicha xaminomisa
- xigga masina monomisa.

Kuhi massako xiggi oddimii wachaami yunvursitena nigisti ellinii mohammad sawaisanchi hospitalane lamebanchi labratorena baxamo.

Sorophii affoo daffii

Xigga massakamare hofi qaxii xissi machesamoisa kullamo.

Sidesena xanokii erbituwa

Kahi sorobane bikamichine sidamoki luwuwuwi dinatem ihuki muli luwi beisa danamisa kullamo. Kahi sorobane bikokii manene xigi dili qaxomane xigi woli sukal qaxoma jabbi sidamulas errii fayaomina hakimichii beyo maramoisa isinomo.

Sorophii maxamii woshuwaa

Ayyi kaa sorobane sidamukii woshuwii daphakosine disinomo.Ayyi kaa sorobane sidamukii woshuwii ka sorrobina yakaa uwamukii anan xiggina afurohani ihukuyaa kaha soroba baxokii maninse mulli kenii la'ena xanoyoo. kaa sorophi bikaneka sidamukii woshaa bikkanchi furrmane'e itamukissa kurubellasesnse chakinsomoyo. kuhi sorobi sansawe'e woshii ihukisam ku kitabii erri higgi chakishanehe bikaneka manomato kuroni firoissa kuramakoo.

Sabimmik oddimi ae'imi urmii xanomisa kurimaa

kaa sorobane bikoki manii hundimi hundomissinemi ixii amanene hassa ittaha bikoissa kullamo. sorobanse hasukii amanene itukii belasii firim xanamoisa kullamo. kaa sorobane bikimaa sabimine illagene mahi luwamii hogobeisa fayomii quxone mahi dangoimi affoo beisaa kuramakoo

xammichi yollassi

ka sorobane ayyi xamichi yolassi kanii woronii yooki silki quxurene teimi emailena xamimaa xanamoisa chakinisamoo

Abach agaza abosse, silk +251-920993169, imalii: ageabose12@gmail.com

Hayidamako shifaraw bakale,silk+251-917233710, imalii bekeleshiferaw@yahoo.com

Hayidamako aklilu getachew, silk +251911743331 imalii :akachew.2@gmail.com

Hayidamako fanta obssa , silk +251917001033 imalii : sinbona2006@gmail.com

Annex III: Amharic Version Information Sheet

የጥናቱ ርዕስ: የደም ስብልኬት እነ የደም ስኳረ መጠን አጠበበቅ እክል ባስኳረ ህሙማን ለይ ለመጥነት ባዋቻሞ ዩንቫርሲት ንግስት ኢልን ሞሃማዲ ማታሳቢያ ሪፈራል ሆሲፒትል ፣ሆሳዕነ፡ ደቡብ ኢትዮጵያ።

ተመራማሪ፡ አገዛ አቦሴ

አማካሪ፡1. ሽፈረው በቀለ

2. አክሊሉ ጌተቸው

3. ፈንታ ኦብሳ

የተቋሙ ስም፡-ጅማ ዩኒቨርሲቲ፣ ጤና ኢንስቲትዩት ፣ሜዲካል ላቦራቶሪ ትምህርት ክፍል

ወጪውን የሚሸፍነው ተቋም፡- ዋቻሞ ዩኒቨርሲቲ

መግቢያ፡-ይህ የማብራሪያ ቅፅ አሁን እርስዎ እንዲሳተፉ የምንጠይቀዎትን ምርምር ጥናት የሚያብራራ ነው።በዚህ ጥናት ለመሰሳተፍ ከመወሰንዎ በፊት ይህንን ቅፅ መረጃ ሰብሳቢዎቹ በሚያነቡበት ጊዜ በጥሞና በማድመጥ ጥያቄ ካለዎት በመጠየቅ ትክክለኛዉን መልስ ይመልሱ። በዚህ ጥናት መሳተፍ ከጀመሩ በኃላ በማንኛውም ጊዜ ጥያቄ ካለዎት መጠየቅ ይችላሉ።

የጥናቱ ዓላማ፡ የደም ስብልኬት የደም ስኳረ መጠን አጠበበቅ እክል ባስኳረ ህሙማን ለይ ለመጥነት ባዋቻሞ ዩንቫርሲት ንግስት ኢልን ሞሃማዲ ማታሳቢያ ሪፈራል ሆሲፒትል ፣ሆሳዕነ፡ ደቡብ ኢትዮጵያ።

የጥናቱ ሂደት፡ይህን ጥናት ለማካሄድ የደም ናሙና በመውሰድ የላብራቶሪ ምርመራ ማደርግ ነው።

ከጥናቱ ጋር የተያያዘ ጉዳት/አለመመቸት፡ እርስዎ በዚህ ጥናት ውስጥ በመሳተፈዎ ለከፋጉዳት የሚጋለጡበት ሁኔታ አይኖርም።ደም በሚወሰድበት ወቅት አነስተኛ ህመም ሊሰማወት ይችላል።እንዲሁም የመቅላት፣እና የማበጥ ሁኔታ ደም ከተወሰደበት ቦታ ላይ ሊታይ ይችላል።ነገርግን እነዚህ ሁኔታዎች የከፋጉዳት የሚያስከትሉ አይደሉም።

በጥናቱ የመሳተፍ ጥቅም፡ እርስዎ በዚህ ጥናት ላይ በመሳተፍዎ ነፃ የደም ስብልኬት (lipid profile) እነ የደም ስኪረ መጠን አጠበበቅ እክል ምርመራ ያገኛሉ፡

የጥናቱ ተሳታፊ ድርሻ፡ በዚህ ጥናት ለመሳተፍ ፍቃደኛ ከሆኑ ከጤናዎ ሁኔታ ጋር የተያያዙና ሌሎች የግል መረጃዎችን እንዲሰጡ ይጠየቃሉ። በመቀጠልም የሰዉነት ክብደተዎን እና 5 ሚሊ መጠን ያለው የደም ናሙና ለተጠቀሰዉ ዓላማ እንድንወስድ ይጠየቃሉ።

የጥናቱ ተሳታፊዉ መብት፡ በጥናቱ ላይ ለመሳተፍ ባይስማሙ ምንም አይነት ቅጣት የማያስከትል ሲሆን ማንኛውም እርሰዎ ሊያገኙ የሚገባውን ህክምናና ተያያዥ መብት የማያሳጣ መሆኑን እናረጋግጣለን።

የጥናቱ መረጃዎች ምስጥራዊነት፡ እርሰዎን በተመለከተ የምንናገኘውን መረጃ በጥናቱ ወቅትም ሆነ ከዚያ በኃላ ባሉት ጊዜያት እንዲሁም ከጥናቱ የተገኘው መረጃ ሚስጥራዊነት የሚጠበቅ ሲሆን መረጃዎቹም የሚያዙት በስም ሳይሆን በልዩ ኮድ ነው።ይኸው መረጃ በጥንቃቄ የሚያዝና የተፈቀደለት ተመራማሪ እና ለህክምና ባለሙያዉ ብቻ ይህም እጅግ አስፈላጊ በሆነ ጊዜ ብቻ ካልሆነ በስተቀር ለሌላ ለማንም ሰው አይሰጥም።ማንኛውም ከርስዎ ጋር የተያያዘ ውጤት በልዩ ኮድ ብቻ የሚያዝ ሲሆን ውጤቱም ለሳይንሳዊ ዓላማ ብቻ ስም በማይገልፅ ሁኔታ እንዲታተም ይደረጋል።

ስለጥናቱ መረጃ ማግኘት ቢፈልጉ፡ ጥናቱን በተመለከተ ግልጽ ያልሆነ ማንኛዉንም ጥያቄ ካለዎት ነፃ ሆነው ከዚህ በታች ባለው አድራሻ መጠየቅ ይችላሉ።

አገዛ አቦሴ

ሞበይል፡0920993169/0991369602 ኢሜል፡ageabose12@gmail.com

Hossana, Ethiopia

Thank you!!

Annex IV: Consent Form

English Version

Participants name _____

I am informed fully in the language I understand about the aim of above mentioned research. I understood the purpose of the study entitled with assessment of lipid profile and glycemic control status among diabetic patients attending Wachemo University Nigist Ellen Mohammad Memorial Referral and Teaching hospital. I have been informed there will be interview and blood sample will be taken. In addition I have been told all the information collected throughout the research process will be kept confidential. I understood my current and future medical services will not be affected if I refused to participate or with draw from the study.

Agree_____ Not agree_____

Therefore I give my consent freely for my participation in this study.

Name of participant: _____Signature:_____

Name of researcher: _____Signature_____

Date_____

Annex V: Haddiyissa Version Consent Form

Bikanchi anani xiggi _____

Bikanchi summi_____

Anni hanane summi chakisumi bikanchi sukal jabii hikimina qerral amane awono manane orachi dili qaxoma eesam xigi woli sukal qaxoma danamisa egerim hogimine waro hawuwa laimma sorrobane anii laomanine machesomi saggarine ihokii woshuwaa kuramako. Fayaaomii woshuwaa xammkamisa xiggaa mahi dangimi afonii masakamisa chakisamako. Oddimii masakamii woshuwaa hundami maxamisane amdakamisa chakisamako. Kaa sorobane bikimm urimaa shigigumlas ayii amanenem sabimaa xanomisa kuramakoo kaka isimina mahi luwamii hoggomi beisaa danamisa chakisamko.

Itaamomo_____ittamumoyyo _____

Bikanchi summi _____furma'i---------- balii ----------

Sorobanchi summi ---------- furma'i ---------- balii ----------

Annex VI: የስምምነት ቅፅ (Consent Form)

የተሳታፊው ልዩ መለያ ቁጥር _____

የተሳታፊው ስም _____

እኔ ስሜ ከላይ የተጠቀሰው ተሳታፊ ባዋቻሞ ዩንቨርስት ንግስት ኢልን ሞሃማዲ ማታሳቢያ ሪፈራል ሆሲፒትል የደም ስብልኬት እነ የደም ስኪረ መጠን አጠበበቅ እክል ባስኪረ ህሙማን ምርምር ላይ በሚገባኝ ቋንቋ በቂ መረጃ አግኝቻለሁ። የህክምና መረጃና ናሙና ምንም አይነት ጉዳት በማያደርስ መልኩ እንደሚወሰድ ተረድቻለሁ። በተጨማሪም የሚወሰዱ ማናቸውም መረጃዎች በሚስጢር እንደሚያዙ ተነግሮኛል። እንድሁም የሚጠየቀውን መረጃ ያለመስጠትና በጥናቱ ያለመሳተፍ መብት እንዳለኝ እንድሁም ከጥናቱ በማንቸውም ወቅት ራሴን ማግለል እንደምችል የተገለፀልኝ ሲሆን ይህንንም በማድረጌ ወደ ፊት ምሆንና አሁን የማገኛቸው የህክምና ግልጋሎቶች እንደማይጓደሉብኝ ተነግሮኛል።ተረድቻለሁ።በዚህ መሠረት ያለጥናት ቡድኑ አባላት ተፅዕኖ በሙሉ ፈቃደኝነት በዚህ ጥናት ውስጥ በመሣተፍ የሚጠበቅብኝን አስተዋፅዎ ለማበርከት በፊርማዬ አረጋግጣለሁ።

የታካሚ/ የተሳታፊ ስም _____ፊርማ ---------- ቀን ----------

የተማራማሪ ስም ----------ፊርማ ---------- ቀን ----------

Annex VII: Questionnaire

INSTRUCTIONS: The questionnaire contains a question, which were pertinent to the research objectives. Study participants were kindly requested to answer all questions carefully as much as possible and the interviewer should fill blank spaces and encircle appropriate choice according to given alternatives and as participants response.

Participant Identification

- Card No _____
- Code _____
- Address_____

PART I. Socio-Demographic Characteristics			
S.NO	Questions	Alternatives'	Comments
101	Age	--------years	
102	Sex	1.Male 2.Female	
103	Educational level	----------	
104	Marital status	1. Single 2. Married 3. Divorced 4. Widowed	
105	Residence	1.Urban 2.Rural	
106	Monthly income	--------Ethio birr	
107	Occupation	1.Government Employed 2. Housewife 3. Merchant 4.Farmer 5. Other specify----------	

PART-II: Diabetes Self- Care Activities			
201	How many of the last SEVEN days followed healthful eating plan	0 1 2 3 4 5 6 7	
202	On average, over the past month, how many days per week have you followed your eating plan?	0 1 2 3 4 5 6 7	
203	On how many of the last SEVEN days did you participate in at least 30 minutes of physical activity?	0 1 2 3 4 5 6 7	
204	Do you drink alcohol?	1. Yes 2. No	If no skip to Q206
205	If yes for Q203 how much do you drink per day in average?	----------ml	
206	Do you use home glucometry	1 Yes 2 No	
Part –III Clinical characteristics			
301	Type of diabetes	1. Type 1 2. Type 2	
302	Duration diabetes from time of dx	----------year	
303	Do you have history of complication	1. Yes 2. No	If No skip to Q 305
304	If yes for Q 303 select types complication	Macro complication 1.Coronary artery disease 2.peripheral artery disease 3. Cerebrovascular disease Micro complication 1 Retinopathy 2 Neuropathy 3 Nephropathy	
305	Do you have family history of diabetes	1. Yes 2. No	
306	Do you have other medal condition	1. Yes	If No skip to Q308

		2. No	
307	If yes for Q306 mention type of disease	----------	
308	Which type of medication you took for diabetes	1. Insulin only 2. OHA only 3. Both insulin and OHA in combination 4. Others specify---------	
309	FBS	First ---------mg/dl Second ---------mg/dl	
Part -IV Medication adherence			
401	Do you sometimes forget to take your medications?	1 Yes 2 No	
402	Were there any days when you did not take your medications due to other reasons rather than forgetting?	1 Yes 2 No	
403	Have you ever cut back or stopped taking your medications without telling your doctor, because you felt worse when you took it?	1 Yes 2 No	
404	When you travel or leave home, do you sometimes forget to bring along your medications?	1 Yes 2 No	
405	Did you take your medications yesterday?	1 Yes 2 No	
406	When you feel like your health condition is under control, do you sometimes stop taking you medications?	1 Yes 2 No	
407	Taking medications every day is a real inconvenience for some people. Do you ever feel hassled about sticking to your treatment plan?	1 Yes 2 No	
408	How often do you have difficulty of remembering to take all your medications?	1 Never 2 Once in a while 3 Sometimes 4 Usually 5 All the time	

Part –V Anthropometric measurements			
501	Height	----------m	
502	Body weight	----------kg	
503	Waist circumference	----------cm	
504	Hip circumference	----------cm	

Data collector name _____ Date _____ Signature_____

Annex VIII: Haddiyisaa Tirati Xamichuwaa

Xissano ananisakami ogora

- Caridik xiggi:_____
- Anannaxi quxurii:_____
- Beyyii :_____

Hundemi xamichuwina dabachi yohani ihubikina ihokii dabacha saggarine kutakoni danamisa issina xaminomo. Badoo ihuki xamichuwaa kinuwiina agukisinehe dabacha kulehe

shootii Mato: hechii kankikuwi xammichuwaa			
X.quxura	Xamichuwa	Dabacha	Kaniana ni sawit yolas
101	Umuri meo?	----------hincho	
102	Alibacha?	1.Gooncho 2. Landicho	
103	Losani gaballi?	----------	
104	Mini isata?	1.Isumoyoo 2.Issamo 3.isaa tiramo 4.isea/ixi lehako	
105	Hechi beyi?	1. gandisa 2. gaxaraa	
106	Agganane sidohik ethiopea birrane me'oo?	----------birra	
107	Baxi shotoi?	1. abulancho 2. balli baxanancho 3. adili baxancho 4. losancho 5. dadarancho 6. mulaniihulas chakisa----------	

Shotti Lamo: Gaqi fayaommi egellim xammichuwaa			
201	Saantamee bal hakimuwi kuramuki icha egetakamo	0 1 2 3 4 5 6 7	
202	Higu agganane saanta mee bal hakimuwi kuramuki icha egetakamo	0 1 2 3 4 5 6 7	
203	Santa mee bal sadib daqiqa loboka orachi xoxolish baxakamo?	0 1 2 3 4 5 6 7	
204	araqe agakamone	1 Eyaa 2 aeaʿe	
205	Agakamilasbalanehinkana	----------ml	
206	Minene xiqi sukal kenacho awaxitakamonihe	1 Eyaa 2 aeaʿe	
Shotti-Saso Xissi ogolli xamichuwaa			
301	sukal jabi halata	type 1 type2	
302	Xisina hinka madanita masitakamoki	1.insulina 2. OHA 3. lamomi	
303	Kuhi xissi kemu hawiso	1 Eyaa 2 aeaʿe	
304	Eyaa yitakolasi hink orach baxacha xisokoki	Lobi xiqi hagisanchi bohe xiso 1 wodani xiso 2 loki eisam angi xiso 3 Hororle wolli xiso Hoff xiqi higisanch bohe xiso 1 ille xiso 2 muli xiso 3 assech higisanch celluwa xiso	
305	Sukal xissi mulixis exa hawiso	1 Eyaa 2 aeaʿe	
306	Eyaa yitakolasi hakimi	----------	

	maha yama kurama summa kule		
307	Kin minene kani ellageni ka xissi xisu ani esami ami yoo	1 Eyaa 2 aeaˈe	
308	Sukaljabi amadukani hikan hincho iha	----------	
309	Sukal kenati qaxoma	1Kuhi sorbi asherukani illegen agena ---------- mg/dl 2 lami agani----------mg/dl	
Shotti- Soro Sukal Qarare Massakam Duha taphanso xamicha			
401	Qarare xadako bal yoo	1 Eyaa 2 aeaˈe	
402	Mashkaine karare masitakoni utako bal yoo	1 Eyaa 2 aeaˈe	
403	Hakimuwina kutakoni qarare utako bal yoo	1 Eyaa 2 aeaˈe	
404	Bebela karare masitakahine	1 Eyaa 2aeaˈe	
405	Xummi machesamo amane karare utaka laqakamone	1 Eyaa 2 aeaˈe	
406	Gogo matakam amane karare xadaka laqakamonehe	1 Eyaa 2 aeaˈe	
407	Karare massimi kemukare utaka lakakamonihe	1 Eyaa 2 aeaˈe	
408	Me’e kore sukal jabi qarae xadakamo	1 Horiyem 2 meto matikocho 3 higahiga 4 lobakat amane 5 hundiamanenim	
Shotti-Onto Orachi Kenato			
501	Ulichi qerralomi?	----------m	
502	Orachi kemato ?	----------kg	
503	Lamaxi hararoma	----------cm	
504	Sasari hararoma	----------cm	

Annex IX: መጠይቅ

መመሪያ፡ ይህ መጠይቅ በውሰጡ ከጥናቱ ዓላማ ጋር የተያያዙ ጥያቄዎችን ይዟል እርሰዎም ትክክለኛዉን መልስ እንድሰጡን በትህትና እነጠይቆታልን በታቻለዎ መጠን በጥንቃቄ ባዶ ቦታዎችን በመሙላት ወይም ከተሰጡት አማራጮች ውስጥ ተገቢ የሆኑትን መልሶችን ለማረጀ ሰብሳቢ ይናገሩ።

የተሳታፊ መለያ

- የተሳታፊ ተራ ቁጥር----------
- የተሳታፊ መለያ ኮድ----------
- አድራሻ----------

ክፈል አንድ፡የማህበራዊእናስነ-ህዝብባህሪያት			
ተ.ቁ	***መጠይቅ***	አማራጮች	አስተያየት
101	ዕድሜ	-------በዓምት	
102	ፆታ	1. ***ወንድ*** 2. ***ሴት***	
103	የትምህርት ደረጃ		
104	የጋብቻ ሁኔታ	1. ያላገባ/ች 2. ያገባ/ች 3. የተፋቱ 4. በሞት የተለየ/ች	
105	የሚኖሩበት አካባቢ	1.ገጠር 2. ከተማ	
106	ያዋር ጋብ ኢትዮጵያ ብር	----------	
107	የሚተዳደሩበት የስራ አይነት	1. የመንግስት ሰራተኛ 2. የቤት እመቤት	

		3. ነጋዴ 4. ገበሬ 5. ሌላ ከሆነይጥቀሱ -----	
ክፈል-ሁለት ፡ባህሪያት መጠይቆች			
201	ሀክም የመከሮትን አመገ በሰምንት ለ ስንት ቀን ይከተለሉ	0 1 2 3 4 5 6 7	
202	በለፈወ ወረ በአመከይ ሀክም የመከሮትን አመገ በሰምንት ለ ስንት ቀን ተከተተሉ	0 1 2 3 4 5 6 7	
203	በሰምንት ስንት ቀን የአካል ብቃት እንቅስቃሴ ያደርጋሉ	0 1 2 3 4 5 6 7	
204	የሚያሰክርመጠጥይጣጠሉ	1. አዎን 2. አይደለም	
205	መልሶአዎንከሆናበቀንምንያህልይ ጣጣሉ	----------ሚሊ	
206	ቤት ዉስጥ ስኪረ መጠኖን ይለከለሉ	1. አዎን 2 . አይደለም	
ክፈል -ሶስት የህክምናመጠይቆች			
301	የስኪረ ህመም አይነት	1 የ ልጆች 2 የኣዋቅዎች	
302	ለ ስኪረ ህመሞየምጠቃሙት ምንድነዉ	1. መረፌ 2. ክንን 3. ሁለቱንም	
303	ህመሞኮፕልከኬት አረገወል	1. አዎ 2. አይደለም	
304	መልሶአዎንከሆና ምን አይነት ነዉ	ትልቁ የደም ቧንቧ ህማም 1 የልብ ደም ቧንቧ ህመም 2 የሰዉነት መዳረሻ ደም ቧንቧ ህመም 3 የአእምሮክፈል ደም ቧንቧ ህመም ትንሹ የደም ቧንቧ ህመም	

		1 የ ዐይን ዉስጥ ደም ቧንቧ ህመም 2 የኩለልት ደም ቧንቧ ህመም 3 የነረቨ ህመም	
305	ቤተሰቦ ውስጥ የስኪረ ህመም ተመም ሰው ነበረ	1. አዎ 2. አይደለም	
306	ከስኪረ ህመም ገረ ልለ ተጎደኚ ህመምአለቦት	1. አዎ 2. አይደለም	
307	መልሶአዎንከሆና ምን አይነት ነው	----------	
308	የስኪረ ህመም ምንአክል ግዜ ቆየቦት	---------በአመት	
309	የስኪረ መጠን ለሶስት ተከተይ ወረት	1---------ግ/ዴሌትረ 2-------ግ/ዴሌትረ	
ክፍል አረት ሜድከለ አደሄረስ			
401	መደንት እረስቶ የወቀሉ	1. አዎ 2. አይደለም	
402	በሚክነት መደንት ሰወስዱ ቀረተወየ ወቀሉ	1. አዎ 2 አይደለም	
403	ሃክም ሰየመክሩ መደንት አቆረጦ	1. አዎ 2 አይደለም	
404	መንገድ ስሄዱመደንትእረስቶ የወቀሉ	1. አዎ 2 አይደለም	
405	ትነትነ መደንት ወሰደዋሉ	1. አዎ 2 አይደለም	
406	ጤንነት ስሰሞት መደንት አቆረጦ የወቀሉ	1. አዎ 2 አይደለም	
407	መደንት መዉሰድ ዐሰልች ስሆንመደንት አቆረጦ የወቀሉ	1. አዎ 2 አይደለም	

408	ስንት ግዜ መደንት የመስተወስ ችግረ አገጥሞታል	1.ማቼም 2.አንዴ 3.አልፎአልፎ 4.ብዙ ግዜ 5.ሁሌም	
ክፍል አምስት ፡ የሰዉነት ልኬት			
501	**ቁመት**	--------ሜትረ	
502	**ክብደት**	-------ስ.ሜ	
503	የዋጋብ ልኬት መጠን	-------ስ.ሜ	
504	የዳሌ ልኬት መጠን	---------ስ.ሜ	

Data collector name _____ Date _____ Signature_____

Annex X: Laboratory Procedure

A: Determination of Fasting Blood Glucose Level

Cobas 4000 series Cobas c311 chemistry analyzer used to determine the second and third blood glucose by hexokinase method, but the first fasting blood glucose determined by mindray BS-200E chemistry analyzer by glucose oxidase method because Cobas 4000 series Cobas c311 was not available.

Principle of glucose oxidase method: Oxidation of glucose by glucose oxidase generates D-gluconolactone and hydrogen peroxide. The hydrogen peroxide generated then reacts with phenol and 4- aminoantipyrine in the presence of the enzyme, peroxidase, to form a colored quinoid dye product. Absorbance of colored product was proportional to the glucose concentration in the sample and measured at 546 nm.

Principle of hexokinase method: Hexokinase catalyzes the phosphorylation of glucose to glucose-6-phosphate by ATP. Glucose-6-phosphate dehydrogenase oxidizes glucose-6-phosphate in the presence of NADP to gluconate-6-phosphate. No other carbohydrate is oxidized. The rate of NADPH formation during the reaction is directly proportional to the glucose concentration and measured photo metrically.

Specimen: Serum

Reagents

R1: MES buffer, pH 6.0; Mg2+, ATP, NADP, preservative.

R2: HEPES buffer: pH 8.0: Mg2+, HK, G-6-PDH; preservative

Calibration	**Control:**
Calibrators: S1: H2O	PreciControl ClinChem Multi1
S2: C.f.a.s.	
Calibration mode- Linear	PreciControlClinChem Multi2
Calibration frequency - 2-point	

B: Measurement of lipid profile

Basic lipids that are measured in the laboratory include total cholesterol, triglycerides, HDL hcholesterol, and LDL cholesterol. They were measured by Cobas 4000 series Cobas c311 clinical chemistry analyzer an automated system. The electrometric (ion-selective electrode/ISE) and the photometric measurement provide a wide variety of tests.

Sop for total cholesterol

Introduction: Cholesterol is found almost exclusively in animals. It is a key membrane component of all cells. It is a steroid alcohol with 27 carbon atoms that are arranged in a tetra cyclical sterane ring system, with a C-H side chain. Determination of total cholesterol (TC) level is good for risk assessment of cardiovascular disease.

Principle: Cholesterol is an enzymatic, colorimetric test method for the quantitative determination of cholesterol in human serum and plasma. Cholesterol esters are cleaved by the action of cholesterol esterase to yield free cholesterol and fatty acids. Cholesterol oxidase then catalyzes the oxidation of cholesterol to cholest-4-en-3-one and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide effects formed the oxidative coupling of phenol and 4-aminophenazone to form a red quinoneimine dye. The color intensity of the dye formed is directly proportional to the cholesterol concentration. It is determined by measuring the increase in absorbance.

Specimen: Serum

Reagents: PIPES buffer: pH 6.8; Mg2+, sodium cholate, 4-aminophenazone: phenol, fatty alcohol polyglycol ether: 3 %cholesterol esterase, cholesterol oxidase, peroxidase preservative

Calibration	**Control:**
Calibrators: S1: H2O	PreciControl ClinChem Multi1
S2:C.f.a.s.lipid	
Calibration mode- Linear	PreciControlClinChem Multi2
Calibration frequency - 2-point	

Sop for triglycerides

Introduction: Triglycerides constitute 95% of fat stocked in tissues and their main role is to provide energy to cells. They are synthesized in the intestine from food and in liver from ingested saccharides, then transported in the blood by chylomicrons and very low-density lipoproteins (VLDL). High levels of triglycerides are associated with atherosclerosis.

Principle: Triglycerides are hydrolyzed by lipoprotein lipase to glycerol and fatty acids. Glycerol is the phosphorylated to glycerol-3-phosphate by ATP in a reaction catalyzed by glycerol kinase. The oxidation of glycerol-3-phosphate is catalyzed by glycerol phosphate oxidase to form dihydroxyacetone phosphate and hydrogen peroxide. In the presence of peroxidase, hydrogen peroxide affects the oxidative coupling of 4-chlorophenol and 4-aminophenazone to form a red-colored dye. The increase in absorbance is directly proportional to the concentration of triglycerides in the sample.

Specimen: Serum

Reagents: PIPES buffer: pH 6.8; Mg2+: sodium cholate: ATP, 4-aminophenazone; 4-chlorophenol; lipoprotein lipase; glycerokinase, glycerol phosphate oxidase; peroxidase preservative.

Calibration	**Control:**
Calibrators: S1: H2O	PreciControl ClinChem Multi1
S2:C.f.a.s.lipid	
Calibration mode- Linear	PreciControlClinChem Multi2
Calibration frequency - 2-point	

Sop for high density lipoprotein cholesterol

Introduction: The principal role of HDL in lipid metabolism is the uptake and transport of cholesterol from peripheral tissues to the liver through a process known as reverse cholesterol transport.

Principle: Homogeneous enzymatic colorimetric method is used. In the presence of magnesium ions, dextran sulfate selectively forms water-soluble complexes with LDL, VLDL, and chylomicrons which are resistant to PEG-modified enzymes. The cholesterol concentration of HDL-cholesterol is determined enzymatically by cholesterol esterase and cholesterol oxidase coupled with PEG to the amino groups. Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by cholesterol esterase. In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to delta 4-cholestenone and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-amino-antipyrine and HSDA (Sodium N-(2-hydroxy-3-sulfopropyl)-3, 5-dimethoxyaniline) to form a purple-blue dye. The color intensity of the blue dye formed is directly proportional to the HDL-cholesterol concentration. It is determined by measuring the increase in absorbance photo metrically.

Specimen: Serum

Reagents

R1 TAPSO buffer; pH 7.77; polyanion, EMSE, ascorbate oxidase, peroxidase detergent; BSA, preservative.

R2 Bis-Trisc buffer, pH 6.70; cholesterol esterase, cholesterol oxidase, cholesterol oxidase, peroxidase, 4-amino-antipyrine, BSA, detergents; preservative.

Calibration	**Control:**
Calibrators: S1: H2O	PreciControl ClinChem Multi1
S2:C.f.a.s.lipid	
Calibration mode- Linear	PreciControlClinChem Multi2
Calibration frequency - 2-point	

Sop for low density lipoprotein

Introduction: LDL primarily contains apo B-100 and is more cholesterol rich than other apo B–containing lipoproteins. It is atherogenic lipoprotein which transports cholesterol from liver to tissue.

Principle: Homogeneous enzymatic colorimetric assay used for determination of LDL-cholesterol. LDL-cholesterol selectively solubilized by a nonionic detergent. In the presence of Mg++ a sugar compound markedly reduces the enzymatic reaction of the cholesterol measurement in VLDL and chylomicrons. The combination of a sugar compound with detergent enables the selective determination of LDL-cholesterol in serum. In the presence of cholesterol esterase cholesterol esters quantitatively break down into free cholesterol and fatty acids. In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to delta 4-cholestenone and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-aminoantipyrine and HSDA to form a purple-blue dye. The color intensity of this dye is directly proportional to the cholesterol concentration and is measured photo metrically.

Specimen: Serum

Reagents

R1 MOPS (3-morpholinopropane sulfonic acid) buffer: pH 6.5; HSDA, ascorbate oxidase; peroxidase; preservative.

R2 MOPS (3-morpholinopropane sulfonic acid) buffer: pH 6.8; $MgSO4 \cdot 7H2O$; 4-aminoantipyrine:; cholesterol esterase; cholesterol oxidase; peroxidase; detergent; preservative.

Calibration	**Control:**
Calibrators: S1: H2O	PreciControl ClinChem Multi1
S2:C.f.a.s.lipid	
Calibration mode- Linear	PreciControlClinChem Multi2
Calibration frequency - 2-point	

Sop for hemoglobin A1c test:

Introduction: Hemoglobin consists of four protein subunits, each containing a heme moiety, and is the red-pigmented protein located in the erythrocytes. HbA_1c is sub fraction of adult hemoglobin A which formed by the attachment of sugars to the N-terminal amino group of the beta-chain.

Principle: The HbA1c determination is based on the turbidimetric inhibition immunoassay (TINIA). This method uses TTAB (Tetradecyltrimethylammonium bromide) as the detergent in the hemolyzing reagent to eliminate interference from leukocytes. Sample pretreatment to remove labile HbA1c is not necessary. All hemoglobin variants which are glycated at the β-chain N-terminus and which have antibody-recognizable regions identical to that of HbA1c are determined by this assay.

Specimen: whole blood collected on EDTA tube and mix thoroughly. The minimum volume required for analysis directly from collection tubes is 1 mL of whole blood**.**

Reagents: R1 Antibody Reagent: MES buffer: 0.025 mol/L, TRIS buffer: 0.015 mol/L, pH 6.2, HbA1c antibody > 0.5 mg/mL, detergent, stabilizers and preservatives (liquid)

R2 Polyhapten reagent: MES buffer: 0.025 mol/L, TRIS buffer: 0.015 mol/L, pH 6.2; HbA1c Polyhapten: > 8 µg/mL, detergent, stabilizers and preservatives (liquid)

Calibration	**Control:**
Calibrators: S1- S6 C.f.a.s.Hba1c	NormN
Calibration mode- spline	PathP
Calibration frequency - full	

Table 8:ATP III Classification of LDL-C, TC, HDL-C and TG (mg/dL)(11)

Lipid profile	Interpretation
LDL- Cholesterol	
<130	Normal
130-159	Borderline high
160-189	High
>190	Very high
Total Cholesterol	
<200	Desirable
200-239	Borderline high
>240	High
HDL- Cholesterol	
<40	Low
>60	High
Triglyceride	
<150	Normal
150-199	Borderline high
200-499	High
>500	Very high

C: Anthropometric measurements

Anthropometry is the study of the measurement of the human body in terms of the dimensions of bone, muscle, and adipose tissue. Several indexes and ratios can be derived from anthropometric measurements. In this study the following anthropometric measurements are included

Body mass index (BMI) is a measure of weight adjusted for height, calculated as weight in kilograms divided by the square of height in meters (kg/m2). BMI is a simple, inexpensive, and noninvasive surrogate measure of body fat (59).

Table 9: Body mass index classification used to perform research on lipid profile and glycemic control status among diabetes mellitus in WCUNEMMH, 2020.

BMI	OBSETIY CLASS	INTERPRETATION
<18.5		Underweight
18.5–24.9		Normal
25.0–29.		Overweight
30.0–34.9	Class –I	High Obesity
35.0–39.9	Class-II	Very high obesity
>40.0	Class-III	Extreme obesity

Waist circumference is a measure of abdominal or visceral fat. Increased waist circumference is associated with the risk of diabetes and cardiovascular disease. It can be used by its own or in combination with BMI to determine increased risk of morbidity and mortality due to excessive abdominal fat(60).

Table 10: Ethnic specific waist circumference measurement standard used to perform research on lipid profile and glycemic control status among diabetes mellitus in WCUNEMMTH, 2020.

Country or ethnic group	Sex	**Waist circumference (cm)**
	Male	≥ 94 cm
	Female	≥ 80 cm
European		
Sub-Saharan Africans	Use European data until more specific data are available	

Bivariate logistic regression analysis of sociodemographic factors associated to dyslipidemia among DM patients attending at Wachemo University Nigist Elleni Mohammed Memorial Referral and Teaching Hospital, Hossana, Southern, Ethiopia, May 1 to June 30, 2020

Variables		N	Dyslipidemia		COR(95% CI)	P-Value
			Yes (%)	No (%)		
Age in years	<25	20	15(75)	5(25)	Ref	0.481
	25-44	102	89(87.3)	13(12.7)	2.282(0.71-7.33)	0.166
	45-64	149	131(87.9)	18(12.1)	2.426(0.787-7.477)	0.123
	≥65	36	31(86.1)	5(13.9)	2.067(0.518-8.251)	0.304
Sex	Male	201	174(86.6)	27(13.4)	Ref	
	Female	106	92(86.8)	14(13.2)	1.139(.563-2.304)	0.718
Educational status	Noformal education	66	54(81.8)	12(18.2)	0.556(.229-1.348)	0.194
	Primary(1-8)	79	67(84.8)	12(15.2)	0.690(.287-1.659)	0.407
	Secondary(9-12)	62	56(90.3)	6(9.7)	1.154(.404-3.294)	0.790
	Diplomaand above	100	89(89)	11(11)	Ref	0.439.
Marital status	Single	34	26(76.5)	8(23.5)	Ref	0.042
	Married	252	224(88.9)	28(11.1)	2.462(1.016-5.961)	0.046
	Divorced	14	12(85.7)	2(14.3)	1.846(0.339-10.043)	0.478
	Widowed	7	4(57.1)	3(42.9)	0.41(.075-2.232)	0.303
Residence	Urban	213	188(88.3)	25(11.7)	Ref	
	Rural	94	78(82.9)	16(11.1)	0.648(0.328-1.281)	0.212
Occupation	Government	95	83(87.4)	12(12.6)	Ref	0.432
	Employed					
	Housewife	65	58(89.2)	7(10.8)	1.198(0.445-3.226)	0.721
	Merchant	69	58(84)	11(16)	0.762(0.315-1.846)	0.547
	Farmer	53	48(90.5)	5(9.5)	1.388(0.461-4.179)	0.560
	Other	25	19(76)	6(24)	0.458(0.152-1.375)	0.164
Monthly income	≤1000ETB	30	24(80)	6(20)	Ref	
	>1000ETB	268	235(87.7)	33(12.3)	1.780(0.678-4.677)	0.242
Type of DM	Type1	27	21(77.8)	6(22.2)	Ref	
	Type2	280	245(87.5)	35(12.5)	2(0.755-5.297)	0.163

Bivariate logistic regression analysis of clinical ,behavioral and anthropometric factors associated to dyslipidemia among DM patients attending at wachemo university Nigist Elleni Mohammed Memorial Referral and Teaching Hospital , Hossana , Southern, Ethiopia, May 1 to June 30, 2020.

Variables		N	Dyslipidemia		COR(95% CI)	P-Value
			Yes (%)	No (%)		
History of complication	Yes	87	75(86.2)	12(13.8)	1.054(0.511-2.173)	0.887
	No	220	191(86.8)	29(13.2)	Ref	
Dietary plan adherence	Yes	80	71(88.7)	9(11.3)	Ref	
	No	227	195(85.9)	32(14.1)	1.295(0.589-2.846)	0.521
Physical Exercise	Yes	18	12(66.7)	6(33.7)	Ref	
	No	289	251(86.9)	38(13.1)	1.321(0.365-4.778)	0.671
Glucometer usage	Yes	13	8(61.5)	5(38.5)	Ref	
	No	294	258(87.8)	36(12.2)	4.479(1.389-14.45)	0.012
Alcohol Drinking	Yes	8	8(100)	0(0)	NA	
	No	299	258(86.2)	41(13.8)		
Duration of DM	<7 Years	213	185(86.9)	28(13.1)	Ref	
	≥7 Years	94	81(86.2)	13(13.8)	0.943(0.465-1.914)	0.871
Familyhistory of DM	Yes	54	45(83.3)	9(16.7)	Ref	
	No	253	221(87.4)	32(12.6)	1.381(0.617-3.093)	0.432
Hypertension as comorbidity	Yes	58	51(87.9)	7(12.1)	1.152(0.483-2.747)	0.749
	No	249	215(86.3)	34(13.7)	Ref	
Typeof medication	Insulin only	96	81(84.4)	15(15.6)	1.08(0.278-4.193)	0.911
	OHA	193	170(88)	23(12)	1.478(0.397-5.5)	0.560
	Insulin and OHA	18	15(83.3)	3(16.7)	Ref	0.626
Medication Adherence	Good	164	139(84.8)	25(15.2)	Ref	0.583
	Moderate	90	80(89.9)	10(11.1)	1.439(0.657-3.149)	0.363
	Poor	53	47(88.7)	6(11.3)	1.409(0.545-3.644)	0.480
BMI	18.5-24.9	159	28(17.6)	131(82.3)	Ref	0.007
	25-29.99	118	22(18.6)	96(81.4)	3.33(1.47-7.545)	0.004
	≥30	30	4(13.3)	26(86.7)	3.391(0.766-15)	0.108
HC					1.037(1.010-1.066)	0.008
WC					1.053(1.025-1.082)	0.000
Glycemic control	Good	54	41(76)	13(24)	Ref	
	Poor	253	225(89)	28(11)	2.548(1.219-5.325)	0.013

Annex XI: Laboratory result reporting format

Laboratory Request Form for lipid profile, Blood glucose and Hemoglobin A1c

ID _____Age_____ Sex_____

Physician Name _____

Test	Result
TC	mg/dl
TG	mg/dl
HDL-C	mg/dl
LDL-C	mg /dl
FBS	mg/dl
HgbA1c	%

Annex XII: Declaration Form

I, the undersigned, hereby declare that this MSc thesis is my original work, and has never been presented for any degree in Jimma University or any other institutions of higher learning in Ethiopia. I also declare the duly acknowledgement of all material sources used for this thesis.

Name of the student: Ageze Abose (MSc candidate)

Signature: _____ Date of submission: _____/_____/_____

Approval of the advisors

This research thesis will be approved by the supervision of university advisors:

External Examiner: Professor. Esayas Kebede(MD,DTM&H,PHD)

Signature: _____ Date of submission: _____/_____/_____

Internal Examiner: Mr.Waqtola Cheneke (MSc, Assi.prof)

Signature: _____ Date of submission: _____/_____/_____

1. Name of 1st advisor: Mr. Shiferaw Bekele (MSc, Assi.prof)

Signature: _____ Date of submission: _____/_____/_____

2. Name of 2nd advisor: Mr. Aklilu Getachew (BSc, MSc)

Signature: _____ Date of submission: _____/_____/_____

3. Name of 3rd advisor: Mr. Fanta Obsa (BSc, MSc)

Signature: _____ Date of submission: _____/_____/_____

Name of School head: _____

Signature: _____ Date of submission: _____/_____/_____