

# Prevalence and Associated Risk Factors of Dyslipidemia among Type Two Diabetic Patients Attending Tiko Cottage Hospital

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## ABSTRACT

Dyslipidemia is one of the major modifiable risk factors for cardiovascular disease in type 2 diabetic patients. Dyslipidemia in type 2 diabetic patients is attributed to increased free fatty acids flux secondary to insulin resistance. Despite its high prevalence and related complications of in type 2 diabetic patients, there is a paucity of data on the prevalence of dyslipidemia in type 2 diabetic patients in Tiko. The objective of this study was to determine the prevalence of dyslipidemia amongst type 2 diabetic patients attending Tiko Cottage Hospital. A cross-sectional based study was conducted from February to April 2023. A convenient sampling technique was used to recruit 179 type 2 diabetic patients into the study. Data on socio-demographic characteristics, behavioral and clinical factors were collected using a structured questionnaire through face-to-face interviews. Five milliliters of venous blood sample were collected for serum glucose and lipid analysis. Blood pressure, weight and height were measured. Data were analyzed using SPSS version 21, whereby univariate analysis using frequency and proportions described the variables, bivariate analysis with the support of Chi-Test of independence measured the association between two variable while multivariate analysis was employed to highlight critical risk factors with the support Logistic Regression. The overall prevalence of dyslipidemia among study participants was 54.7%. Isolated lipid profile abnormality of hypercholesterolemia was found in 14.0%, hypertriglyceridemia was absent, high level of High density lipoprotein (HDL-C) was found in 53.1%, and high level of low density lipoprotein (LDL-C) was found in 0.6% of study participants. Being obese was significantly associated with dyslipidemia and female were significantly more exposed. The study concluded that high prevalence of dyslipidemia was found among type 2 diabetic patients in the study area and that obesity was a critical risk factor. The findings of this study should be taken into account to conduct appropriate intervention measures on the identified risk factors and implement routine screening, treatment and prevention of dyslipidemia.

**How to cite this paper:** Fodji Praise Afuh | Moses N. Ngemenya | Lepasia Arnold Fonge | Nana Célestin "Prevalence and Associated Risk Factors of Dyslipidemia among Type Two Diabetic Patients Attending Tiko Cottage Hospital" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-8 | Issue-1, February 2024, pp.61-72, URL: [www.ijtsrd.com/papers/ijtsrd61307.pdf](http://www.ijtsrd.com/papers/ijtsrd61307.pdf)



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**KEYWORDS:** Diabetes, dyslipidemia, patients, risk factors, Hospital, Tiko

## INTRODUCTION

Lipid disorder or dyslipidemia is a state that occurs due to abnormalities in plasma lipids. It can also be defined as disorders that arise due to defects in lipid metabolism. These abnormalities can be either due to an increase in one or a combination of plasma lipids. The different abnormalities in plasma lipids include increase total cholesterol, increased low density lipoprotein cholesterol (LDL-C), increased triglycerides (TGs), and reduced high density

lipoprotein cholesterol (HDL-C) [2]. Cholesterol is any class of organic molecules called lipids which is divided into HDL-C, LDL-C, and TGs. All these constitute an atherogenic lipid profile [1]. Triglycerides are the storage form of energy in the body and also functions in insulating the body. When the level of triglycerides increases, it leads to hardening of the arteries or thickening of the artery walls which can lead to atherosclerosis. HDL-C is

known as good cholesterol because its carries LDL-C out of the arteries and back to the liver where the LDL-C are broken down and passed from the body [3]. Decrease in HDL-C levels will lead to an increase in the accumulation of LDL-C in the arteries which will lead to the hardening of the arteries (LDL-C plays an atherogenic role while HDL-C plays a protective role) [7]. The mechanisms involved in causing dyslipidemia are not well understood, but previous studies showed that, an increase Apo Lipoprotein B levels, increase density LDL levels, increase hepatic synthesis of triglyceride with very low density lipoprotein with decrease clearance of triglycerides, circulating cytokines, acute phase reactants and viral infections also serve as different mechanisms which leads to abnormal levels in plasma lipids. Notwithstanding, genetic alterations has a role too in the elevations of lipid levels (this arises due to mutation of low density lipoprotein gene), of plasma lipoprotein levels in a normal health population [2,3], thus making it autosomal dominant.

Dyslipidemia has become a major global health concern especially in Africa, with a prevalence range from 5.2% [4] to 89.9% [2], and in Cameroon, with a prevalence of 46.0%. This high prevalence and increase mortality caused by dyslipidemia is due to low living standards, low income earnings and urbanization, poor dieting as a result of increased consumption of processed foods, sweetened beverages, edible oils which increases the level of triglycerides, and dyslipidemia has an annual mortality rate of 4 million[4]. Further studies have proven that by the year 2030, infectious diseases will be overtaken by dyslipidemia. Projected increase in the death rates arising from dyslipidemia is as a result of lack of surveillance and research on non-communicable diseases [5].

Diabetes mellitus is a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fats, protein metabolism resulting from insulin secretion, insulin action or both. The effects of diabetes mellitus include long term dysfunction and failure of various organs [5]. Diabetes mellitus may present several symptoms like thirst, polyuria, blurring of vision, weight loss and when severe symptoms like ketoacidosis or non-ketotic hyperosmolar state may develop and lead to stupor, coma, and in the absence of treatment death [6]. Its long term effects may include progressive development of specific complications of retinopathy with potential blindness, neuropathy that may lead to renal failure and/or neuropathy with risk of foot ulcers, amputation and features of automatic dysfunction including sexual dysfunction, several pathological conditions that may

lead to diabetes may include destruction of the beta cells of the pancreas and other that results from resistance to insulin action. Diabetes is divided into three types that is type I, type II and gestational diabetes. Research has shown that majority of the world's population die from type II diabetes, even in Africa. This high mortality rate is due to poor management and unhealthy life styles.

### **Statement of the problem**

Lipid disorder or dyslipidemia is a state that occurs due to abnormalities in plasma lipids. Disorders lipid metabolism can be as a result of increase total cholesterol, increased low density lipoprotein cholesterol (LDL-C), increased triglycerides (TGs), and reduced high density lipoprotein cholesterol (HDL-C).

Lipid disorder with a prevalence of 51.5% in the Southwest region of Cameroon has become the major reasons why there are high mortality rates in diabetes mellitus as it can lead to several complications due to poor management. Lipid disorders have become one of the major conditions which accounts for the high mortality rate in diabetes mellitus as it affects organs like the heart, liver, pancreas. Very few studies have been carried out on this topic in our country setting Cameroon.

### **General objective**

The aim of this study was to determine the prevalence of dyslipidemia amongst type two diabetic patients attending Tiko Cottage hospital.

### **Specific objectives**

- To determine the prevalence of dyslipidemia amongst patients attending Tiko Cottage hospital.
- To determine the risk factors associated with dyslipidemia amongst type two diabetic patients attending Tiko cottage hospital and its associated risk factors.
- Investigate the effects of different diabetic medications and other medications like hypertensive therapy and anti-retroviral on dyslipidemia on type two diabetic patients attending the Tiko Cottage Hospital.

### **Significance of the study**

This study will go a long way to educate the public and raise awareness on what lipid profile is and the importance of checking lipid profile regularly. It will also educate the public (especially diabetic patients) on the importance of life style modifications especially when there is noticeable increase in lipid levels. The findings will also enhance the health policy of Cameroon, Africa and the world at large in order to mitigate health risk associated with dyslipidemia amongst type two diabetic patients.

## Scope of the study

Geographically, this study was limited to Tiko Cottage Hospital, one of the reference hospitals in the Southwest Region of Cameroon. Content wise, this study is limited to dyslipidemia amongst type two diabetic patients.

## Methodology

### Research Design

This was a cross-sectional descriptive survey study carried out amongst type two diabetic patients attending Tiko Cottage Hospital in the Southwest region of Cameroon.

### Area of study

The study was carried out in Tiko at the Biochemistry Department of the Tiko Cottage Hospital. Tiko is located about 18 km from Buea which is at the foot of Mount Fako. This hospital offers services like emergency room services, short term hospitalization, X-ray/radiology services, blood services, laboratory services. The laboratory services include hematology, biochemistry, microbiology and parasitology.

### Population of the study

All adult patients in the hospital aged from 21 years and above who consented to take part in the study.

### Study duration

The study was carried out for 3 months, as of February to April 2023.

### Sample

The sample size was calculated as follows using the Cochran's formula whereby at least 178 type two diabetic patients were to be included in the study.

Within the purposively sampled cluster, convenient sampling method was used to get participants for the study. Therefore, participants were chosen as they meet the inclusive criteria and according to their availability. Participants were approached during sample collections, informed about the study and recruited after obtaining the informed consent form signed. A questionnaire which includes assessment of personal information was administered and using face-to-face interview. After each participant fills his/her questionnaire, his/her venous blood was collected into a dried tube. At the end of the day, the filled questionnaire was checked and validated, then kept for data entry, exploration and analysis.

### Inclusion criteria

Adult male and female aged 21 years or more who had type two diabetes and who voluntarily accepted to take part in this study and signed the written consent form, were recruited in to the study.

### Exclusion criteria

Participants suffering from urinary tract infections, with a history of kidney transplant or dialysis, or

those with known kidney, liver and heart diseases as well as pregnant women were excluded from the study. Participants with hepatitis B and C were excluded from the study as well.

## Material and methods

### Method

#### Pre-Analytical Phase

#### Recruitment of participants

The administrative clearance (authorization to carry our research) obtained from the Faculty of Health Science of the University of Buea was presented to the authority of Tiko Cottage Hospital. After obtaining authorization from the director of the hospital, we moved to the diabetic unit where we met with the head of the unit who informed the participants about the research and the importance. Those who gave their consent were administered the questionnaires.

#### Data collection and capture

#### Administration of questionnaire

Participants that met the inclusive criteria of the study and that consented to take part were administered the questionnaire following the face-to-face approach.

#### Measurement of weight, height, body mass index and blood pressure

Analog scale with Kg reading was used to measure the weight of study subjects. Height was also measured while they were standing by lowering the horizontal scale bar snugly to the crown of the head.

#### Body mass index (BMI):

$$\text{BMI} = \text{weight (kg)} / \text{height squared (m}^2\text{)}$$

Blood pressure of the subject was measured according to the WHO recommendations using a sphygmomanometer. Both systolic and diastolic blood pressure measurements were taken in units of millimeter mercury (mmHg).

#### Blood sample collection and handling

After putting on gloves, a tourniquet was applied to the patient's arm in order to identify a vein. The patient was instructed to make a fist in order to see the vein clearly. The venipuncture site was cleaned with cotton soaked in 70% alcohol. A vacutainer needle screwed into its holder was inserted into the vein with its bevel facing upwards. A dry vacuum tube was inserted through the other end of the needle. About 4 -5 mL of non-fasting venous blood was obtained from each participant. The tube was gently removed once the required amount of blood was obtained. The tourniquet was then untied and the needle removed. The blood sample at room temperature was allow to clot and centrifuged at 3000 revolutions per minute (rpm) for 5 minutes to obtain the sera. The sera were then aliquot into labeled

Eppendorf tubes and stored at  $-20^{\circ}\text{C}$ , and were later analyzed in batches for LDL-c, TC, TGs and HDL-c.

### Transportation

The transportation of the sample from the HIV-unit to the Biochemistry unit was done using a test tube rack which takes approximately about 15 minutes. The sample was also stored in a refrigerator at  $-20^{\circ}\text{C}$  until we obtained a large number of samples to run the test.

### Analytical phase

#### Methods of biochemical analysis

##### Determination of triglycerides

Principle: An increase in TG co-exists with a low level of HDL-C and high levels of small-dense lipoproteins. TG in a serum/plasma sample is exposed to lipoprotein lipase (LPL) which results in the release of glycerol and free fatty acids (FFE). 3-phosphoglycerol and adenosine-5-diphosphate are formed from glycerol with the involvement of glycerol kinase under favorable conditions. Dihydroxyacetone and hydrogen peroxide are formed from 3-phosphoglycerol and molecular oxygen in the presence of glycerol phosphate oxidase. Hydrogen peroxide reacts with 4-chlorophenol and 4-aminoantipyrine forming red-colored quinoneimine [15]. The intensity of this color is proportional to the amount of TG in the blood measured using spectrophotometry.

For working reagent, the contents of reagent 1 enzymes were dissolved into reagent 2 buffer to form the working reagent. Tubes were labeled in to blank, standard, control and patients sample (serum). In each of the tubes, 1 ml of the reagent was pipetted and dispensed. In the tube containing the standard 10 ul of the standard was added, same quantity with the control and participants sample. Tubes were mixed and incubated for 5 min at  $37^{\circ}\text{C}$  after which the blank and control were measured in the spectrophotometer and the sample as well as the control read after being passed through the machine. Absorbance (A) was read of the samples and calibrator, against the Blank. The color is stable for at least 30 minutes.

Normal range:  $< 150 \text{ mg/dl}$

Unit conversion:  $[\text{mg/dl}] \times 0.011 = [\text{mmol/l}]$ .

##### Determination of total cholesterol

Principle: It is determined using enzymatic methods and using auto analyzers. After the enzymatic hydrolysis of cholesterol esters by cholesterol oxidase to 4-cholestenone, with the formation of hydrogen peroxide, it reacts with 4-aminophenazone and 4-chlorophenol with the involvement of peroxidase forming a red product (Trinder Reaction). The concentration of the product formed is determined by spectrophotometry.

Reagents were ready to use from manufacturer. Tubes were labeled in to blank, standard control and patients sample (serum). In each of the tubes, 1 ml of the reagent was pipetted and dispensed. In the tube containing the standard 10 ul of the standard, was added the same quantity with the control and participants' sample. Tubes were mixed and incubated for 5 min at  $37^{\circ}\text{C}$  after which the blank and control were measured in the spectrophotometer and the sample as well as the control read after being passed through the machine. Absorbance (A) was read of the samples and calibrator, against the Blank. The color is stable for at least 60 minutes.

Normal range:  $> 200 \text{ mg/dl}$

##### Determination of HDL-Cholesterol

Principle: Using direct method, HDL is measured directly in serum. The Apo B containing lipoproteins in the specimen are reacted with a blocking reagent that renders them non-reactive with the enzyme cholesterol reagent under the conditions of the assay. The Apo B containing lipoproteins are effectively excluded from the assay and only HDL-C is detected under adequate assay conditions. This method uses sulfated alpha-cyclodextrin in the presence of magnesium, which forms complexes with the Apo B containing lipoproteins, and polyethylene glycol coupled with cholesteryl esterase and cholesterol oxidase form the oxidase for the HDL-C measurement.

For working reagent, the contents of reagent 1 enzymes were dissolved into reagent 2 buffer to form the working reagent.

Reagents 1 and 2 were ready to use.

Instrument was adjusted to zero with distilled water.

Tubes were labeled as sample, reagent, control and blank. 250 ul of sample was mixed with 25 ul of precipitate reagent, and incubated at  $37^{\circ}\text{C}$  for 5 minutes and centrifuged at 4500 rpm for 5 mins after which 25 ul of supernatant was mixed with 1000 ul of cholesterol reagent in the tubes for blank, standard, control and sample then incubated for another 5 minutes and the absorbance measured and recorded.

Normal range:  $<50 \text{ mg/dl}$  (women),  $<40 \text{ mg/dl}$  (men)

Dysfunctional HDL is caused primarily by inflammation as well as oxidative stress and glycation, increased expression of myeloperoxidase.

##### Determination of LDL-Cholesterol

LDL-cholesterol transports about 70% of the cholesterol present in blood [37]. Cholesterol and its esters accounts for 40%-50% of the LDL-particle mass [37]. Due to the key predictive role of LDL in atherogenesis, the LDL-c levels which indirectly

reflect LDL content in the blood is a primary lipid factor of cardiovascular risk [37]. As blood samples for lipid profile do not need to be collected in a fasting state, the testing is more available and the calculation/determination of the LDL-C level is easier [2, 3, 6, 8]. Based on preparative ultracentrifugation, lipoproteins are divided into two fractions according to their density (chylomicrons and VLDL are rejected). LDL, HDL, Lp(a) and intermediate density lipoproteins (IDL) is the reference method used for calculating LDL-C. LDL levels, are usually calculated but less frequently determined by direct methods [37].

The Friedewald Formula are used to calculate LDL-C level [40].

Normal range: <50 mg/dl

#### Quality control

The reagents storage conditions, expiry dates and lots number were checked for validity of the reagents.

Established standard operating procedure (SOPs) for each step of laboratory testing process, ranging from specimen handling to instrument performance validation as presented in table 1 were followed.

**Table 1: Quality control of a spectrophotometer**

BLANK	Allows for setting the spectrophotometer to zero before measuring unknown solution. I.e. to zero out the background reading and only report values for the compound of interest.
CALIBRATOR	To ensure that the results are accurate and determine if there are issue with the spectrophotometer.
STANDARD	Commonly used to help identify and determine the concentration of a substance whose concentration is unknown.
CONTROLS	Used to maintain the variability of the results that can occur especially when there are variable that are not in question and can interfere with the results. NORMAL CONTROL; contains values within the normal range. PATHOLOGIC CONTROL: contains values below or above the norm.

#### Post analytical phase

##### Issuing of participants' results

Participant's results were printed on an A4 paper and handed to them individually. For participants that were not present, they were reached by phone call.

##### Conservation of specimens

Participant serum will be stored in the refrigerator at a temperature of -20°C for verification of test results in case of any doubt or fault.

##### Waste management

Waste were separated into infectious, non-infectious and sharps. Infectious and non-infectious wastes were kept in two separate rappens while the sharps were preserved in an enclosed and well safe guarded container. All three waste types were disposed of through the process of incineration by the janitor.

#### Data management and analysis

Codes were assigned to each participant so as to observe strict confidentiality. The results were entered in a secured log book. The questionnaire and logbook were treated with strict confidentiality. Data was entered into Microsoft excel version 16, cross-checked for errors and then transferred into Statistical Package for Social Science (SPSS) version 21 (IBM Inc., 2012) for analysis. Further exploratory statistics were performed in SPSS to check for invalid entries and outliers. Scale variables were described using Mean value and Standard Error of Mean. Data were transformed into categorical variables and described using frequency and proportions. The differences between groups were compared using Chi-Square test associated with crosstabulation. The risk factors were appraised using Odd-Ratio associated with Binary Logistic Regression. All statistics were discussed at 95% Confidence Level (CL), with Alpha set at 0.05.

#### Findings

The findings of this study highlight the socio-demographic characteristics of the participants, their dyslipidemia status and risk factors associated with dyslipidemia.

##### Socio-demographic characteristics

A total of 179 diabetic patients were screened among which 33% (n=59) were males and 67% (n=120) were females. Their mean age was  $57.1 \pm 8.4$  years.

The majority of participants were within the age group 56-65 with a proportion of 54.2% (97).

A proportion of 5.0% (9) were housewives, 50.8% (91) were self-employed, 20.1% (36) were private workers, 14.0% (25) were public civil servants while 10.1% (18) were retired workers.

With respect to their marital status, 2.8% (5) were divorced, 75.4% (135) were married, 6.1% (11) were single and 15.6% (28) were widows/widowers (table 2).

**Table 2: Socio-demographic characteristics**

Variable	Category	Frequency	Percent
Age	30-45	14	7.8
	46-55	51	28.5
	56-65	97	54.2
	>65	17	9.5
	<b>Total</b>	<b>179</b>	<b>100.0</b>
Sex	Female	120	67.0
	Male	59	33.0
	<b>Total</b>	<b>179</b>	<b>100.0</b>
Marital Status	Divorced	5	2.8
	Married	135	75.4
	Single	11	6.1
	Widow	28	15.6
	<b>Total</b>	<b>179</b>	<b>100.0</b>
Occupation	Housewife	9	5.0
	Self-employed	91	50.8
	Private	36	20.1
	Public	25	14.0
	Retired	18	10.1
	<b>Total</b>	<b>179</b>	<b>100.0</b>

### Distribution of lipid profiles of participants

As for the cholesterol class, majority had it normal 86.0% (154), all were normal for triglyceride, a weak majority 53.1% (95) was abnormal for HDL while nearly all was normal for LDL 99.4% (178) as presented on table 3.

**Table 3: Distribution of lipid profiles of participants**

Variable	Category	Frequency	Percent	Mean $\pm$ standard deviation
Cholesterol class	Abnormal	25	14.0	149 $\pm$ 40
	Normal	154	86.0	
	<b>Total</b>	<b>179</b>	<b>100.0</b>	
Triglyceride	Normal	179	100.0	52.02 $\pm$ 5.8
HDL class	Abnormal	95	53.1	75.4 $\pm$ 24.3
	Normal	84	46.9	
	<b>Total</b>	<b>179</b>	<b>100.0</b>	
LDL class	Abnormal	1	0.6	46.2 $\pm$ 5.8
	Normal	178	99.4	
	<b>Total</b>	<b>179</b>	<b>100.0</b>	

### Prevalence of dyslipidemia amongst study participants

The overall prevalence of dyslipidemia in the study was 54.7% (98) as presented in table 4.

The prevalence of dyslipidemia was 50.0% (7) among those aged 30-45 years, rose to 58.8% (30) amongst age group 56-65 years been the highest, dropped slightly to 55.7% (54) among those aged 56-65 years, before falling further to 41.2% (7) amongst those aged 65 years and above though this association was not significant ( $P=0.622$ ). Dyslipidemia was significantly ( $P=0.000$ ) preponderant among female with proportion of 73.3% (88) as against 17.0% (10) for the male (table 5).

**Table 4: Distribution of dyslipidemia**

Dyslipidemia	n	%	95% CI
No	81	45.3	38.1-52.6
Yes	98	54.7	47.4-61.9
<b>Total</b>	<b>179</b>	<b>100.0</b>	

**Table 5: Association between age, sex and dyslipidemia**

variable	category	Dyslipidemia		No dyslipidemia		$\chi^2$ -test
		n	%	n	%	
Age	30-45	7	50.0	7	50.0	$\chi^2=1.77$ P=0.622
	46-55	30	58.8	21	41.2	
	56-65	54	55.7	43	44.3	
	>65	7	41.2	10	58.8	
Total		98		81		
Sex	Male	10	17.0	49	83.0	$\chi^2=50.76$ P=0.000
	Female	88	73.3	32	26.7	
Total		98		81		

**Clinical characteristics of participants**

As for the BMI, 49.0% (88) were obese, 42.9% (77) were overweight while 8.1% (14) were normal. Majority was hypertensive 61.1% (109), 12.8% (23) do exercise among which less than majority do it more than three times a week 39.1% (9), 41.8% (75) consume alcohol, and 2.8% (5) were HIV positive. Blood group O+ was the most represented with proportion of 51.9% (93). Dyslipidemia was significantly associated with exercise (P=0.000) whereby those that did not do exercise were significantly more exposed with proportion of 85.3% (93) as against 21.7% (5) for those that do exercise; also, those that do exercise more than three days a week were significantly less exposed with proportion of dyslipidemia of 44.4% (4) as compared to 85.7% (12) for those that do it less than three days a week. Those that do not consume alcohol weekly were significantly less exposed with proportion of 45.2% (47), as compared to 68.0% (51) for those that consumed alcohol weekly (P=0.002), as presented in table 6.

**Table 6: Clinical characteristics of participants and association with dyslipidemia**

Variable	Category	Dyslipidemia	Percent	No dyslipidemia	Percent	$\chi^2$ -test
BMI	Normal	2	14.3	12	88.7	$\chi^2=10.10$ P=0.006
	Obesity	52	59.1	36	40.9	
	Overweight	44	57.1	33	42.9	
	<b>Total</b>	<b>98</b>		<b>81</b>		
Hypertension	No	23	32.9	47	67.1	$\chi^2=22.24$ P=0.000
	Yes	75	68.8	34	31.2	
	<b>Total</b>	<b>98</b>		<b>81</b>		
Exercise	No	93	85.3	64	14.7	$\chi^2=10.11$ P=0.000
	Yes	5	21.7	18	78.3	
	<b>Total</b>	<b>98</b>		<b>81</b>		
Weekly alcohol consumption	No	47	45.2	57	54.8	$\chi^2=9.15$ P=0.002
	Yes	51	68.0	24	32.0	
	<b>Total</b>	<b>98</b>		<b>81</b>		
HIV status	Negative	94	54.0	80	46.0	$\chi^2=0.48$ P=0.487
	Positive	4	80.0	1	20.0	
	<b>Total</b>	<b>98</b>		<b>81</b>		
Blood group	A+	29	63.0	17	37.0	$\chi^2=8.77$ P=0.032
	AB+	12	80.0	3	20.0	
	B+	15	75.0	5	25.0	
	O-	4	80.0	1	20.0	
	O+	38	40.9	55	59.1	
	<b>Total</b>	<b>98</b>		<b>81</b>		

Number of days of exercise per week	<3	12	85.7	2	14.3	$\chi^2=2.67$ P=0.102
	>3	4	44.4	5	55.6	
	<b>Total</b>	<b>23</b>		<b>18</b>		

Those that were HIV positive had higher proportion of dyslipidemia, 80.0% (4), as compared to 54.0% (8) for those that were HIV negative though this difference was not significant (P=0.487). As far as blood group was concerned, those with blood group ‘O’ had significantly (P=0.032) the lowest proportion of those with dyslipidemia which means they were least at risk with proportion of 40.9% (3), followed by those having blood group ‘A+’, with proportion of 73.0% (29). The obese were mostly at risk of dyslipidemia 59.1% (5), significantly higher than the 14.3% (2) for those with normal BMI (P=0.006). Those with hypertension were significantly more exposed with proportion of 68.8% (7) as compared to 32.9% (4) for those without hypertension (P=0.000), table 6.

**Effects of different diabetic medications and other medications on dyslipidemia**

There was no significant association between the dyslipidemia and diabetic medications (P=0.992) though the highest percentage of dyslipidemia was recorded among those that take insulin 56.0% (14). As for other medications taken, hypertension (HT) drug had the highest proportion with dyslipidemia 59.0% (5), though this difference was not significant (P=0.306), as presented on table 7.

**Table 7: Association between diabetic treatment and dyslipidemia**

Variable	Category	Dyslipidemia	Percent	No Dyslipidemia	Percent	$\chi^2$ -test
Diabetic Medications	Amareh	10	55.6	8	44.4	$\chi^2=0.09$ P=0.992
	Glucophage	45	53.3	35	43.7	
	Insulin	14	56.0	11	44.0	
	Metformin	29	51.8	27	48.2	
Total		<b>98</b>		<b>81</b>		
Other medications	ARV	1	20.0	4	80.0	$\chi^2=3.62$ P=0.306
	ARV/HT	4	57.1	3	42.9	
	HT	59	59.0	41	41.0	
	NTH	34	50.7	33	49.3	
Total		<b>98</b>		<b>81</b>		

**Risk factors of dyslipidemia**

**Table 8: Bivariate analysis of predictors of dyslipidemia among participants**

Variable	Category	COR	95% CI		P-value
			Lower limit	Upper limit	
Age group	56-65	1.794	0.631	5.104	0.273
	46-55	2.041	0.669	6.225	0.210
	30-45	1.429	0.344	5.940	0.624
	65	1			
Sex	Female	6.040	2.100	8.930	0.000
	Male				
BMI	Overweight	1.871	0.692	5.054	0.217
	Obesity	2.650	0.970	7.242	0.049
	Normal	1			
Physical exercise	yes	1.073	0.532	2.165	0.843
	No				
Number of days of exercise	3 +	1.010	0.285	3.578	0.987
	<3	1			
HIV status	Positive	0.568	0.173	1.864	0.351
	Negative	1			
Weekly alcohol consumption	Yes	1.522	0.822	2.815	0.181
	No	1			
Hypertension	Yes	1.201	0.660	2.186	0.548
	No	1			



Blood group	O+	0.811	0.411	1.603	0.547
	O-	0.345	0.029	4.065	0.398
	B+	0.575	0.154	2.144	0.410
	AB+	1.034	0.158	6.764	0.972
	A+	1			

When controlled for each other, obesity surfaced as a critical risk factor of dyslipidemia ( $P=0.049$ ;  $COR=2.650$ ;  $95\%CI: 0.908, 6.545$ ), then sex ( $P=0.000$ ;  $COR=6.040$ ;  $95\%CI: 2.100 - 8.930$ ) as depicted by table 8.

## Discussion

Dyslipidemia is one of the major modifiable risk factors for cardiovascular diseases in a type two diabetic patient which is the leading cause of morbidity and mortality in these patients [6]. Without timely and effective control, the prevalence of dyslipidemia will continue to rise, leading to a heavy burden of cardiovascular diseases, therefore it is important to identify the potential associated risk factors of dyslipidemia to manage this condition as to reduce the burden of cardiovascular diseases.

The current study attempted to assess the prevalence of dyslipidemia, associated risk factors, effects of diabetic medications and other medications on dyslipidemia among type two diabetic patients attending Tiko Cottage Hospital. The overall prevalence of dyslipidemia among type two diabetic patients in this current study was 54.7%. BMI, alcohol consumption, sex, physical exercise, hypertension and blood group were independent risk factors of dyslipidemia in type two diabetic patients in this study while obesity and sex surfaced as the critical ones while controlled for other factors. The individual lipid profile abnormality obtained were 14%, 0%, 53.1% and 0.6% for TC, TG, HDL-C and LDL-C respectively.

The 54.7% prevalence of dyslipidemia in type two diabetic patients obtained in this study was comparable to that obtained at Buea and Limbe regional hospital in Cameroon with a prevalence of 51.5% [20], 54.6% at the Yaoundé Teaching Hospital in the Center region of Cameroon [15], 63.5% at the University Teaching Medical Center of Ethiopia [20], 64.2% at Sambalpur University India [19], 64.1% at the Department of Biochemistry Institute of Health Science Nepal [20]. The reason for this high prevalence in the current study might be due to obesity, poor life style modifications such as little or no exercise and alcohol consumption which results in higher incidence of type two diabetic patients with its metabolic abnormalities, thus corroborating the work carried out in Jordan [22]. The variation in the prevalence of dyslipidemia could be attributed to dietary differences as well as variation in the genetic deposition of the population [16], thus supporting the findings of this study as alcohol consumption and

blood group were independent risk factors of dyslipidemia.

According to our findings prevalence of individual lipid abnormality for TC, TG, HDL-C was high and low LDL-C with proportions of 14%, 0%, 53.1% and 0.6% respectively which aligns with the study conducted at the Buea and Limbe regional hospital [20].

TC was the most frequent lipid abnormality in this study. The findings of this study are similar to those conducted in Ethiopia [16] and Nepal [25]. The difference in the pattern of dyslipidemia reported in type two diabetic patients might be due to different factors in the study like cultural factors and the lifestyle of the population.

The current study's finding revealed a higher prevalence of hypercholesterolemia compared to other studies conducted in Ethiopia (5.2%) [25], but its comparable with a report from China (14.7%) [25], but is lower than the study conducted in Kembata Ethiopia (27.3%) [20] and Egypt (57.3%) [28].

The prevalence of LDL-C in this study was 0.6% which is lower than the study conducted in Ethiopia (28.6%) [16], India (43.8%) [19] and Nepal (73.8%) [22]. This rise might be due to lipolysis of very low density lipoprotein (VLDL) which after triglyceride supplementation by cholesteryl esters transfer protein along with hepatic lipase mediated hydrolysis of triglyceride and phospholipid, which leads to increase production of LDL-C.

The prevalence of low HDL-C was high as compared to studies carried out in Yaoundé Cameroon (44.3%) [26] and Ethiopia (51.3%) [25] while a higher result was obtained from Ethiopia [15].

Socio-demographic factors can play a role in determining dyslipidemia for diabetic patients. In the current study, dyslipidemia was significantly associated with sex but not significantly with age; this finding is in agreement with the study done in Ethiopia [19], China [25] and Thailand [27].

In this study, some known risk factors such as hypertension, alcohol consumption, exercise and HIV status, showed a high prevalence with dyslipidemia though HIV was not a significant risk factor. A

similar study was carried in Jordan indicating that type two diabetic patients who fall under these categories of risk factors have some chances of developing dyslipidemia.

In this study, dyslipidemia was significantly associated with obesity, as opposed to the study carried out at the Buea and Limbe regional hospital [20]. Study participants who had obesity were 5.6 times more likely to develop dyslipidemia compared to their underweight counterparts. A similar observation was reported in Kenya [16] and China [25]. Hypertension was not significantly associated with dyslipidemia from the current study as compared to studies carried in Ethiopia [20] and Nepal [22].

### Conclusion

The prevalence of dyslipidemia in cottage hospital Tiko was high (54.7%), and was statistically critically associated with obesity and sex. Patients taking insulin were seen to be mostly suffering from dyslipidemia as compared to participants taking other diabetic medication. Other risk factors like alcohol consumption, physical exercise and blood group though significantly associated with dyslipidemia, did not statistically surface as significant risk factors, when controlled for other factors. The findings of this study should be taken into account to conduct appropriate intervention measures on identified risk factor reduction and implement routine screening, treatments and prevention measures to curb dyslipidemia.

### Recommendations

1. Intervention strategies addressing dietary, lifestyle and behavioral factors should be enhanced especially for females, those who take ART.
2. Similar studies should be conducted with a larger sample size and over a long period of time to get more representative results.

### Limitations to the study

Refusal of patients to participate in the study was a major challenge to reckon with.

### Acknowledgements

My sincere thanks to my Supervisor Dr. Ngemenya Moses (Associate Professor), to Mr. Lepasia Arnold Fonge for proofreading the manuscript and to Nana Celestin (Prof.), Executive Director at FASTDAM (Foundation of Applied Statistics and Data Management) for collaborating in the statistical analysis of the data. I am equally grateful to the Tiko Cottage Hospital for the services and support giving to me for the accomplishment of this work, not leaving out the study participants who gave their data and blood sample for laboratory analysis. Special

thanks to my father, Mr. Lekeaka Dickson Fodji and the Anawa's Family for their endless love, financial, spiritual moral support given to me in order to accomplish this work.

### References

- [1] De Backer G, Jankowski P, Kotseva K, Mirrakhimov E, Reiner Ž and Rydén L. Management of dyslipidaemia in patients with coronary heart disease: results from the ESC-EORP EUROASPIRE V survey in 27 countries. *Atherosclerosis*. (2019) 285(1):135–46. doi:10.1016/j.atherosclerosis.2019.03.014.
- [2] Tilahun H, Masyuko SJ, Mogaka JN, Temu T, Kinuthia J and Osoi AO. Prevalence and correlates of dyslipidemia in HIV positive and negative adults in Western Kenya: a cross-sectional study. *Medicine*. 2021 1009(2):e24800. doi:10.1097/MD.00000000000024800.
- [3] Ríos-González BE, Luévano-Ortega KE, Saldaña-Cruz AM, González-García JR, Magaña-Torres MT. Polymorphisms of seven genes involved in lipid metabolism in an unselected Mexican population. *J Genet*. (2011) 90:1–6. doi(3): 10.1007/s12041-011-0118-2.
- [4] Chang M-h, Yesupriya A, Ned RM, Mueller PW, Dowling NF. Genetic variants associated with fasting blood lipids in the US population: third national health and nutrition examination survey. *BMC Med Genet*. (2010).
- [5] Kemal A, Teshome MS, Ahmed M, Molla M, Malik T, Mohammed J, Abate KH. Dyslipidemia and associated factors among adult patients on antiretroviral therapy in armed force comprehensive and specialized hospital, Addis Ababa, Ethiopia. *HIV/AIDS (Auckland, NZ)*. 2020(5);12:221.
- [6] Feeney ER, Mallon PW. HIV and HAART-associated dyslipidemia. *The open cardiovascular medicine journal*. 2011;5:49.
- [7] Buchacz K, Weidle PJ, Moore D, Were W, Mermin J, Downing R, Kigozi A, Borkowf CB, Ndazima V, Brooks JT. Changes in lipid profile over 24 months among adults on first-line highly active antiretroviral therapy in the home-based AIDS care program in rural Uganda. *JAIDS Journal of Acquired Immune Deficiency Syndromes*. 2008 Mar 1;47(7):304-11.
- [8] Wu L, Gerard NP, Wyatt R, Choe H, Parolin C, Ruffing N, Borsetti A, Cardoso AA, Desjardin E, Newman W, Gerard C. CD4-induced interaction of primary HIV-1 gp120

- glycoproteins with the chemokine receptor CCR-5. *Nature*. 1996 Nov;384 (6605):179-83.
- [9] Lampejo T, Pillay D. HIV virology, testing and monitoring. *Medicine*. 2013 Aug 1; 41(8):420-436.
- [10] dosSantos Coura R, Nardi NB. The state of the art of adeno-associated virus-based vectors in gene therapy. *Virology journal*. 2007 Dec;4(9):1-7.11:62. doi:10.1186/1471-2350-11-62.
- [11] Tsai F-J, Cheng C-F, Lai C-H, Wu Y-C, Ho M-W and Wang J-H. Effect of antiretroviral therapy use and adherence on the risk of hyperlipidemia among HIV-infected patients, in the highly active antiretroviral therapy era. *Oncotarget*. (2017) 8(12):106369. doi:10.18632/oncotarget.22465.
- [12] Gebreyes YF, Goshu DY, Geletew TK, Argefa TG, Zemedu TG and Lemu KA. Prevalence of high bloodpressure, hyperglycemia, dyslipidemia, metabolic syndrome and their determinants in Ethiopia: evidences from the National NCDs STEPS Survey, 2015. *PLoS ONE*. (2018) 13(13):e0194819. doi:10.1371/journal.pone.0194819.
- [13] Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI and Donato KA. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. *Circulation*. (2009) 120(26):1640-5. doi: 10.1161/CIRCULATIONAHA.109.192644
- [14] Gebreegziabihier G, Belachew T, Mehari K, Tamiru D. Prevalence of dyslipidemia and associated risk factors among adult residents of Mekelle City, Northern Ethiopia. *PLoS ONE*. (2021) 16(17):e0243103. doi:10.1371/journal.pone.0243103.
- [15] Gebreegziabihier G, Belachew T, Mehari K, Tamiru D. Prevalence of dyslipidemia and associated risk factors among adult residents of Mekelle City, Northern Ethiopia. *PLoS ONE*. (2021) 16(12):e0243103. doi:10.1371/journal.pone.0243103.
- [16] Dave JA, Levitt NS, Ross IL, Lacerda M, Maartens G, Blom D. Anti-retroviral therapy increases the prevalence of dyslipidemia in South African HIV-infected patients. *PLoS ONE*. (2016) 11(14):e0151911. doi:10.1371/journal.pone.0151911.
- [17] Tripathy JP, Thakur J, Jeet G, Chawla S, Jain S and Pal A. Burden and risk factors of dyslipidemia-results from a STEPS survey in Punjab India. *Diabetes Metab Syndr*. (2017) 11(11):S21-7. doi:10.1016/j.dsx.2016.08.015.
- [18] Yamamura T, Sudo H, Ishikawa K, Yamamoto A. Familial type I hyperlipoproteinemia caused by apolipoprotein C-II deficiency. *Atherosclerosis*. 1979 Sep 1;34(31):53-65.
- [19] Azah S.C.C And Pokam B.D.T. Dyslipidemia In Type II Diabetic Patients In South West Region Of Cameroon.
- [20] Arslan, N.(2014) obesity, fatty liver diseases and intestine microbiota. *World J. gastroenterol*. 44, 16452-16463.
- [21] Nelson, R. H. (2013) hyperlipidemia as a risk factor for cardiovascular diseases. *Prim. Care* 40, 195-211.
- [22] pischon, T., Nothlings, U, and boeing, H. obesity and cancer. *Proc, Nutr. Soc*. 67, 128-145.
- [23] Ahmed, S, Shah, P, and Ahmed, O. biochemistry, lipids, statpearls, pp. 11-19. Statpearls publishing, Treasure Island
- [24] Preshant Regmi, prajal Gyawali, Rojeet Shrestha, Mano sigdeF. Pattern of dyslipidemia in type two diabetic subject is eastern Nepal. *JNAMLS* 10.1371/journal.pone.0126166.
- [25] Doupa D, Mbengue AS, Diallo FA, Jobe M, Ndiaye A, Kane A. Lipid profile frequency and the prevalence of dyslipidaemia from biochemical tests at Saint Louis University Hospital in Senegal. *Pan Afr Med J*. (2014) 17:75. doi:10.11604/pamj.2014.17.75.3577.
- [26] Asiki G, Murphy GA, Baisley K, Nsubuga RN, Karabarinde A, Newton R. Prevalence of dyslipidaemia and associated risk factors in a rural population in South-Western Uganda: acommunity based survey. *PLoS ONE*. (2015) 10:e0126166. doi:10.1371/journal.pone.0126166
- [27] Ayoade OG, Umoh I, Amadi C. Dyslipidemia and associated risk factors among Nigerians with hypertension. *Dubai Med J*. (2020) 3:155-61. doi:10.1159/.
- [28] Mach F, Baigent C, Catapano AL. ESC Scientific Document Group. 2019 ESC/EAS

- Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J* 2020(18); 41: 111-88.
- [29] NCD Risk Factor Collaboration. National trends in total cholesterol obscure heterogeneous changes in HDL and non-HDL cholesterol and total-to-HDL cholesterol ratio: a pooled analysis of 458 population-based studies in Asian and Western countries. *Int J Epidemiol* 2019(45): doi:10.1093/ije/dyz099.
- [30] Sygitowicz G, Filipiak KJ, Sitkiewicz D. Czy nie-HDL cholesterol lepiej niż cholesterol frakcji LDL odzwierciedla ryzyko sercowo-naczyniowe? *Folia Cardiol* 2018(44); 13: 435-41.
- [31] Quispe R, Hendrani A, Elshazly MB. Accuracy of low-density lipoprotein cholesterol estimation at very low levels. *BMC Medicine* 2017; 15: 83.

