DIABETIC DYSLIPIDEMIA:

PREVALENCE, CLINICAL CHARACTERISTICS AND ASSOCIATION WITH GLYCEMIC CONTROL AMONG DIABETIC PATIENTS IN DAR ES SALAAM

BY

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A dissertation submitted in partial fulfillment of the requirements for the degree of Master of Medicine (Internal Medicine) of the Muhimbili University of Health and Allied Sciences.

Muhimbili University of Health And Allied Sciences

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CERTIFICATION

The undersigned certify that he has read and hereby recommend for acceptance by the Muhimbili University of Health and Allied Sciences a dissertation entitled: ***Diabetic Dyslipidemia: Prevalence, Clinical Characteristics and Association with Glycemic Control Among Diabetic Patients in Dar es Salaam***, in partial fulfillment of the requirements for the degree of Master of Medicine (Internal Medicine).

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I, Stanley P Chattanda, declare that this dissertation is my own original work and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

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DEDICATION

In the loving memory of my parents, Margaret and Philemon;

My dear wife, Hellen, and children Rogers, Brenda and Sylvana.

ABSTRACT

Background

Diabetes is a major healthcare problem, by 2010 there will be approximately 221 million people with diabetes worldwide. Diabetes is associated with quantitative and qualitative changes in lipoproteins. Correcting lipoprotein abnormalities reduces the coronary risk in diabetes.

Objective

To determine the prevalence, clinical characteristics and association between dyslipidemia and glycaemic control among diabetic patients in Dar es Salaam.

Methodology

A descriptive cross-sectional study was conducted between November 2006 and January 2007. A total of 150 diabetic patients aged 18 years and above who were attending the diabetic clinics at Muhimbili National Hospital, Amana, Temeke and Mwananyamala Municipal Hospitals were recruited into the study. These patients were selected by simple random sampling, interviewed using a structured questionnaire, physically examined and then investigated for fasting lipid profiles and glycated hemoglobin using Konelab™ (© 2003 Thermo Electron Corporation) machine. Chi-square test, Student's t-test and multiple logistic regressions were used for data analysis. A *p-value* of < 0.05 was taken to be statistically significant.

Results

The prevalence of dyslipidemia was found to be 94.7%, with the commonest derangement being elevated triglyceride levels in 94.7% (142/150). Dyslipidemia

presented clinically as angina 11.3% (17/142), peripheral vascular disease 2.1% (3/142) and corneal arcus 21.8% (31/142). In a multivariate analysis, poor glycaemic control as determined by measurement of glycated hemoglobin was found to be independently associated with dyslipidemia ($p = 0.036$).

Conclusion and Recommendation

The prevalence of dyslipidemia among diabetic patients in Dar es Salaam is high. Because risk factors for heart disease are additive and even multiplicative, mild degrees of dyslipidemia may increase CHD risk. Therefore, controlling dyslipidemia should be given equal emphasis as controlling hyperglycemia when developing strategies for managing diabetes mellitus.

CONTENTS

1. Certification i

2. Declaration and copyright ii

3. Acknowledgement iii

4. Dedication iv

5. Abstract v

6. Contents vi

7. List of tables vii

8. Abbreviations viii

9. Introduction and Literature review..... 1

10. Problem Statement 20

11. Research Hypotheses..... 21

12. Rationale of the study..... 21

13. Objectives of the study..... 21

14. Methodology 22

15. Results 28

16. Discussion 36

17. Conclusion 40

18. Recommendations 40

19. References.....41

20. Appendices:

Appendix I: Questionnaire 51

Appendix II: Consent Form; English version 54

Appendix III: Consent Form; Swahili version 56

LIST OF TABLES

1. Social and Demographic characteristics of the study participants 29
2. Frequency distribution of dyslipidemia among diabetic patients in Dar es Salaam with respect to lipid subclasses.....31
3. Distribution of dyslipidemia among diabetic patients in Dar es Salaam with respect to sex.....31
4. Distribution of dyslipidemia among diabetic patients in Dar es Salaam with respect to diabetes treatment group 32
5. Proportion of dyslipidemic diabetic patients in Dar es Salaam with respect to site of treatment for diabetes.....33
6. Clinical Characteristics of dyslipidemia among diabetic patients in Dar es Salaam.....34
7. Multivariate analysis; association between different variables with Dyslipidemia among diabetic patients in Dar es Salaam 35

ABBREVIATIONS

ADA	– American Diabetes Association
CDC	– Centers for Disease Control and Prevention
CHD	– Coronary Heart Disease
CVD	– Cardiovascular Diseases
FPG	– Fasting Plasma Glucose
GDM	– Gestational Diabetes Mellitus
HDL-C	– High Density Lipoprotein Cholesterol
HIV	– Human Immunodeficiency Virus
IDDM	– Insulin dependent Diabetes Mellitus
IFG	– Impaired Fasting Glycemia
LDL-C	– Low Density Lipoprotein Cholesterol
MNH	– Muhimbili National Hospital
NIDDM	– Non Insulin Dependent Diabetes Mellitus
OGGT	– Oral Glucose Tolerance Test
PVD	– Peripheral Vascular Disease
TGs	– Triglycerides
VLDL-C	– Very Low Density Lipoprotein Cholesterol
WHO	– World Health Organization

INTRODUCTION AND LITERATURE REVIEW

DIABETES MELLITUS:

DEFINITION, HISTORICAL PERSPECTIVES

The term diabetes mellitus describes a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism, resulting from defects in insulin secretion, insulin action or both. [1]

Egyptian writings from as early as 1500BC described a wasting disease in which sufferers produced sweet tasting urine.[2] These early records described people with mysterious and deadly disease that caused extreme thirst, enormous urine output and wasting away of the body. The ancient Roman doctor Aretaeus of Cappadocia also gave a vivid description of diabetes, describing it as a "melting down of flesh and limbs into urine". [3] Galen, a disciple of Hippocrates, referred to the ailment as "diarrhea of the urine" and "the thirsty disease". [4]

Appolinus of Memphis probably coined the name "Diabetes" sometimes around 250BC.[2] The literal translation means to go through or siphon, reflecting the early understanding of the disease that drained people of more fluid than they would consume. Indeed, the technical term for this disease, diabetes mellitus, means "sweet flow" or "siphon". [4] Because of this hallmark of diabetes, the disease was thought to be the disorder of the kidneys and bladder for more than two thousand years. [2] Gradually, the Latin word for honey sweet "Mellitus" was appended to Diabetes because of its link with sweet urine. Since then, many physicians have remarked on the sweet taste of diabetic's urine.[5]

The effects of diabetes mellitus include long term damage, dysfunction and failure of various organ systems.[1] Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision, and weight loss.[6] In its most severe form, ketoacidosis or a non ketotic hyperosmolar state may develop and lead to stupor, coma, and, in the absence of effective treatment, death.[7]

Often the symptoms are not severe, or may be absent, and, consequently hyperglycemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made. [1,6,7] The long term effects of diabetes mellitus include progressive development of specific complications of retinopathy with potential blindness, nephropathy that may lead to renal failure, and/or neuropathy with risk of foot ulcer, amputation, Charcot joints, and features of autonomic dysfunction including sexual dysfunction. [1,7] People with diabetes are at increased risk of cardiovascular, peripheral vascular and cerebrovascular disease.[1, 6,7,8,9,10]

CLASSIFICATION OF DIABETES MELLITUS AND OTHER CATEGORIES OF GLUCOSE REGULATION

The classification encompasses both *clinical stages* and *etiological types* of diabetes mellitus and other categories of hyperglycemia, as suggested by Kuzuya and Matsuda.[11]

The clinical staging reflects that diabetes, regardless of its etiology, progresses through several clinical stages during its natural history. Moreover, individual subjects may move from stage to stage in either direction. Persons who have, or who are developing

diabetes mellitus can be categorized by stage according to the clinical characteristics, even in the absence of information concerning the underlying etiology. The classification by etiological type results from improved understanding of the cause of hyperglycemia. The etiological classification reflects the fact that the defect or process that may lead to diabetes may be identifiable at any stage in the development of diabetes - even at the stage of normoglycaemia.

Type 1 Diabetes Mellitus

This form of diabetes, which accounts for only 5 – 15% of those with diabetes, previously encompassed by the terms insulin dependent diabetes mellitus, type 1 diabetes, or juvenile onset diabetes, is characterized by selective ß-cell destruction and severe or absolute insulin deficiency. [12] Type 1 diabetes is further subdivided into immune and idiopathic forms.[1, 12] Type 1a is immune mediated, having markers of immune destruction of ß-cells. [13] In this form of diabetes, the rate of ß -cell destruction is quite variable, being rapid in some individuals (mainly infants and children) and slow in others (mainly adults). [12,13]

Autoimmune destruction of ß-cells has multiple genetic predispositions and is also related to environmental factors that are still poorly defined. [1,12,13] Although patients are rarely obese when they present with this type of diabetes, the presence of obesity is not incompatible with the diagnosis. [1,8,12] These patients are also prone to other autoimmune disorders like Grave's Disease, Hashimoto's Thyroiditis, Addison's Disease, Vitiligo,

Celiac Sprue, Autoimmune Hepatitis, Myasthenia Gravis and Pernicious Anemia.[1, 6,8,12,13]

Type 2 Diabetes Mellitus

This form of diabetes, which accounts for about 90 – 95% of those with diabetes, previously referred to as non-insulin-dependent-diabetes (NIDDM), type 2 diabetes, or adult onset diabetes, encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency. [1,6,12,13,14] At least initially, and throughout their lifetime, these individuals do not need insulin treatment to survive. [1,12] There are probably different causes of this form of diabetes. Although the specific etiologies are not known, autoimmune destruction of ß-cells does not occur, and patients do not have any of the causes listed above.[12, 13]

Many patients with this form of diabetes are obese, and obesity itself causes some degree of insulin resistance. [1,12,13] Patients who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal regions. [12,13] This form of diabetes frequently goes undiagnosed for many years because the hyperglycemia develops gradually and at an earlier is often not severe enough for patients to notice any of the classic symptoms of diabetes. [12,13] Nevertheless, such patients are at an increased risk of developing macrovascular and microvascular complications. [1,6,8,12,13] Whereas patients with this form of diabetes may have insulin levels that appear normal or elevated, the higher blood glucose levels in these diabetic patients would be expected to result to even higher insulin values had their ß-cell

function been normal. [12,13] Thus insulin secretion is defective in these patients and insufficient to compensate for insulin resistance. [1,12,13] Insulin resistance may improve with weight reduction and/or pharmacological treatment of hyperglycemia but is seldom restored to normal. [1,12] The risk of developing this form of diabetes increases with age, obesity, and lack of physical activity. [12,13] It occurs more frequently in women with prior Gestational Diabetes Mellitus (GDM) and in individuals with hypertension or dyslipidemia, and its frequency varies in different racial/ethnic subgroups. [12,13] It has a strong genetic pre-disposition, more so than in the autoimmune form of type 1 diabetes. However, the genetics of this form of diabetes are complex and not clearly defined. [1,12,13]

Type 3 Diabetes Mellitus

The type 3 designation refers to multiple other specific causes of elevated blood glucose; such as non-pancreatic diseases, drug therapy and many others.[14]

Type 4 Diabetes Mellitus

This encompasses Gestational Diabetes Mellitus (GDM). [12,13,14] GDM is defined as any degree of glucose intolerance with onset or first recognition during pregnancy.[14] The definition applies regardless of whether insulin or only diet modification is used for treatment or whether the condition persists after pregnancy. [12,13] It does not exclude the possibility that unrecognized glucose intolerance may have antedated or begun concomitantly with the pregnancy. [12,13] The prevalence may range from 1 to 14% of pregnancies, depending on the population studied. GDM represents nearly 90% of all pregnancies complicated by diabetes. [1, 12, 13]

Impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) Also recognized is an intermediate group of subjects whose glucose levels, although not meeting criteria for diabetes, are nevertheless too high to be considered normal. This group is defined as having fasting plasma glucose (FPG) levels 100 mg/dl (5.6 mmol/l) but <126 mg/dl (7.0 mmol/l) *or* 2-h values in the oral glucose tolerance test (OGTT) of 140 mg/dl (7.8 mmol/l) but <200 mg/dl (11.1 mmol/l). Thus, the categories of FPG values are as follows:

- FPG <100 mg/dl (5.6 mmol/l) = normal fasting glucose;
- FPG 100–125 mg/dl (5.6–6.9 mmol/l) = IFG (impaired fasting glucose);
- FPG 126 mg/dl (7.0 mmol/l) = provisional diagnosis of diabetes (the diagnosis must be confirmed, as described below).

The corresponding categories when the OGTT is used are the following:

- 2-h postload glucose <140 mg/dl (7.8 mmol/l) = normal glucose tolerance;
- 2-h postload glucose 140–199 mg/dl (7.8–11.1 mmol/l) = IGT (impaired glucose tolerance);
- 2-h postload glucose 200 mg/dl (11.1 mmol/l) = provisional diagnosis of diabetes (the diagnosis must be confirmed, as described below). [12,13]

Patients with IFG and/or IGT are now referred to as having "pre-diabetes" indicating the relatively high risk for development of diabetes in these patients.[1] In the absence of pregnancy, IFG and IGT are not clinical entities in their own right but rather risk factors for future diabetes as well as cardiovascular disease.[12] They can be observed as

intermediate stages in any of the disease processes listed in. IFG and IGT are associated with the metabolic syndrome, which includes obesity (especially abdominal or visceral obesity), dyslipidemia of the high-triglyceride and/or low-HDL type, and hypertension. [1,12,13] It is worth mentioning that medical nutrition therapy aimed at producing 5–10% loss of body weight, exercise, and certain pharmacological agents have been variably demonstrated to prevent or delay the development of diabetes in people with IGT; the potential impact of such interventions to reduce cardiovascular risk has not been examined to date.

Note that many individuals with IGT are euglycemic in their daily lives.[13] Individuals with IFG or IGT may have normal or near normal glycated hemoglobin levels.[12,13] Individuals with IGT often manifest hyperglycemia only when challenged with the oral glucose load used in the standardized OGTT. [12,13]

Diagnosis of Diabetes Mellitus

The National Diabetes Data Group and the World Health Organization have issued diagnostic criteria for DM based on the following premises:

(1) The spectrum of fasting plasma glucose (FPG) and the response to an oral glucose load varies among normal individuals, and

(2) DM is defined as the level of glycemia at which diabetes specific complications occur rather than on deviations from population-based mean. [1]

Criteria for the Diagnosis of Diabetes Mellitus. [1,6,8,12,13]

- Symptoms of diabetes (polyuria, polydipsia, weight loss) plus random blood glucose concentration ≥ 11.1 mmol/L (200 mg/dL), or
- Fasting (no caloric intake for at least 8 h) Plasma Glucose ≥ 7.0 mmol/L (126 mg/dL), or
- Two-hour plasma glucose ≥ 11.1 mmol/L (200 mmol/L) during an oral glucose tolerance test.

GLOBAL BURDEN OF DIABETES

In the year 2000, it was estimated that the number of people with diabetes was 171 million, about two thirds of these live in the developing world.[1,15] In these countries, most of those who are diabetic are in the economically active age groups. [15]

Between 1995 and 2025, the number of adult population affected by diabetes mellitus in developing countries is projected to grow by 170%, from 84 to 228 million people.[15,16] By 2025, these countries will be home to 76% of all persons with diabetes, as compared with 62% in 1995. [15] In the same period, the developed world will see a 41% increase, from 51 to 72 million people. [15,16,17]

Worldwide, a 122% rise is projected, from the total of 135 to 300 million.[15] This more than twofold global increase will occur because of population aging and growth, as well as from obesity, unhealthy diets and a sedentary lifestyle. These later factors are closely associated with urbanization and industrialization. [1,15,18]

If the present trend persists, by 2025 most people with diabetes in developed countries will be aged 65 years and more, while the majority of diabetic persons in developing

countries will be in the 45 – 64 year age group.[15,16] This means that some 170 million men and women, who will reside in the developing regions of the world in less than 30 years from now, will be suffering from diabetes in their most productive years of life.[1,15] The regions with greatest potential increases are Asia and Africa, where diabetes rates could rise to 2 to 3 times those experienced today.[15] With increasing incidence worldwide, diabetes mellitus will be the leading cause of morbidity and mortality in the foreseeable future.[1]

THE BURDEN OF DIABETES IN AFRICA

The prevalence of diabetes in African communities is increasing with aging of the population and lifestyle changes associated with urbanization and westernization.[1,18] The traditional rural communities still have very low prevalence, at most 1 – 2%, except in some specific high risk groups, whereas 1 – 13% or more in adults in urban communities have diabetes. [19,20,21] Type 2 diabetes is the predominant form (70 – 90%), the rest being represented by typical type 1 patients and patients with atypical presentations that require more pathophysiological insight.[19]

Due to the high urban growth rate, dietary changes, reduction in physical activity and increasing obesity, it is estimated that the prevalence of diabetes is due to triple within the next 25 years. [15,19] In addition, long term complications occur early in the course of diabetes and concern a high proportion of patients, probably higher than in other ethic groups, and that could be partly explained by uncontrolled hypertension, poor metabolic control and possible ethnic predisposition. [1, 19] The combination of the rising prevalence

of diabetes and high rate of long term complications in Africans will lead to a drastic increase in the burden of diabetes on health systems of African countries. [15] The design and implementation of appropriate strategy for early diagnosis and treatment, and population based primary prevention of diabetes in these high-risk populations is therefore a public health priority. [19]

THE BURDEN OF DIABETES IN TANZANIA

Several population-based surveys have been conducted in different parts of the country to determine the burden of diabetes in Tanzania. These surveys showed that the overall prevalence of diabetes in Tanzania is rather low, but shows geographical variability and is strongly associated with age. [20] Studies done during the 1980s found a prevalence of 0.2% of diabetes in the rural population and 1.1% in ordinary urban Africans in Dar es Salaam. [21] It was also found that the highest socio-economic groups have the highest diabetes prevalence, which is 12% among high executives in government and private company positions. [22]

McLarty *et al* in a survey in rural populations of Tanzania found an overall age adjusted prevalence of diabetes and impaired glucose tolerance as 1.1% and 8.4% respectively. [23]

In 1997, a study conducted in Dar es Salaam indicated that the prevalence of diabetes in urban areas has reached 5.7%. [22]

DIABETES AND THE CARDIOVASCULAR SYSTEM

Diabetes increases cardiovascular risk, and is recognized as cardiovascular disease equivalent. [1] A large body of epidemiological and pathological data documents that diabetes is an independent risk factor for cardiovascular disease in both men and women.[24,25,26] Women with diabetes seem to lose most of their inherent protection against developing cardiovascular diseases.[24,25] Cardiovascular diseases are listed as the cause of death in about 65% of persons with diabetes. Diabetes acts as an independent risk factor for several forms of cardiovascular diseases. [1,6,8] To make matters worse, when patients with diabetes develop clinical cardiovascular diseases, they sustain a worse prognosis for survival than do cardiovascular diseases in patients without diabetes. [28,29,30] It follows that diabetes is a major risk factor in the development of cardiovascular diseases. [1]

ASSOCIATION BETWEEN HYPERGLYCEMIA AND CARDIOVASCULAR DISEASES

Studies have shown that improved glycemic control decreases the risk of microvascular conditions (e.g., retinopathy, neuropathy, nephropathy), but its effect on coronary heart disease, stroke, peripheral arterial disease, and other macrovascular diseases has been equivocal. Selvin and associates performed a systematic review of published reports to explore the relationship between long-term glycemic control and the risk of cardiovascular disease in persons with type 1 and type 2 diabetes. According to the

meta-analysis, hyperglycemia is associated with increased cardiovascular risk in persons with types 1 and 2 diabetes. It follows that improved glycemic control may lower cardiovascular risk in patients with diabetes. Cardiovascular disease is common among persons with diabetes because of factors other than hyperglycemia. [31]

In diabetes, increased blood concentrations of markers of oxidative stress, especially in patients with poor glycemic control, have been implicated in the development of vascular complications. [32,33] The finding of increased oxidative stress in newly diagnosed children and diabetic patients with no complications suggests that the increased oxidative stress in diabetes may not be attributable to the complications but could contribute to the development of complications. [32] Other studies in patients with increased concentrations of plasma lipoproteins have implicated increased oxidative stress with associated oxidation of lipoproteins as playing a role in increasing atherogenic risk and associated cardiovascular disease. [34,35,36,37,38]

Atherosclerotic Coronary Heart Disease

Both type 1 diabetes and type 2 diabetes are independent risk factors for Coronary Heart Disease. [39,40,41] Moreover, myocardial ischemia due to coronary atherosclerosis commonly occurs without symptoms in patients with diabetes.[42] As a result, multivessel atherosclerosis often is present before ischemic symptoms occur and before treatment is instituted. A delayed recognition of various forms of CHD undoubtedly worsens the prognosis for survival for many diabetic patients. [1]

Diabetic Cardiomyopathy

Patients with both diabetes and ischemic heart disease have an enhanced myocardial dysfunction leading to accelerated heart failure (diabetic cardiomyopathy). [43,44,45,46,47,48] Thus, patients with diabetes are unusually prone to congestive heart failure. Several factors probably underlie diabetic cardiomyopathy: severe coronary atherosclerosis, prolonged hypertension, chronic hyperglycemia, microvascular disease, glycosylation of myocardial proteins, and autonomic neuropathy. [1] Improved glycemic control, better control of hypertension, and prevention of atherosclerosis with cholesterol-lowering therapy may prevent or mitigate diabetic cardiomyopathy. An early clinical trial suggested that sulfonylureas used for control of hyperglycemia are cardiotoxic and may exacerbate diabetic cardiomyopathy. [49] This side effect, however, was not confirmed in a recent large clinical trial

Stroke

Mortality from stroke is increased almost 3-fold when patients with diabetes are matched to those without diabetesThe most common site of cerebrovascular disease in patients with diabetes is occlusion of small paramedial penetrating arteries Diabetes also increases the likelihood of severe carotid atherosclerosis Patients with diabetes, moreover, are likely to suffer irreversible brain damage with carotid emboli that otherwise would produce only transient ischemic attacks in persons without diabetes. Approximately 13% of patients with diabetes >65 years old have had a stroke

Renal Disease

Renal disease is a common and often severe complication of diabetes. Approximately 35% of patients with type 1 diabetes of 18 years' duration will have signs of diabetic renal involvement. Up to 35% of new patients beginning dialysis therapy have type 2 diabetes End-stage renal disease (ESRD) appears to be especially common among Hispanics, Blacks, and Native Americans with diabetes. For patients with diabetes who are on renal dialysis, mortality rates probably exceed 20% per year When diabetes is present, CVD is the leading cause of death among patients with ESRD.

DIABETES AND DYSLIPIDEMIA

Lipoproteins are complexes of lipids and proteins that are essential for the transport of cholesterol, triglycerides, and fat-soluble vitamins. [1,6,8] Lipoproteins have been classified on the basis of their densities into: -

- Very Low Density Lipoproteins (VLDL): these carry endogenously synthesized cholesterol and triglycerides.
- Low Density Lipoproteins (LDL): these are the principal vehicles for cholesterol transport and are taken up by LDL receptors on hepatocytes and peripheral cells, thus releasing the cholesterol component for cellular use.
- High Density Lipoproteins (HDL): these mediate the reverse transport of cholesterol from peripheral tissues to the liver.

Dyslipidemias are defined as disorders in the levels of lipoproteins. [1,6,8] They are classified into hyper- or hypolipidemias. [1] Dyslipidemias are among the most common metabolic diseases seen in clinical practice. [1,6] They are important because they lead to a

number of sequelae including CHD, dermatological manifestations (xanthelasmata and xanthomata), pancreatitis, and (more rarely) neurological and ocular anomalies. [1,6,14]

The hypolipidemias are very rare but, when they occur, they present with spectacular clinical features. [1,6] They are of two main types. The first, abetalipoproteinaemia or hypobetalipoproteinaemia, results from defective secretion of apolipoprotein B-containing lipoproteins (chylomicra, VLDL, IDL, and LDL). [1] This results into fat malabsorption, ataxic neuropathy, retinitis pigmentosa and acanthocytosis. [1,6,8] The second Deficiency State, Tangier disease, is associated with defective production of apolipoprotein A-containing HDL particles. [1] As a result, transport of cholesterol to the liver for excretion is obstructed and sterol esters accumulate in unusual peripheral sites within reticuloendothelial system (e.g. tonsils, intestinal lymph nodes) and the cornea. Relapsing neuropathy is a feature of this condition. [1,6,8]

Hyperlipidemia is common, and can be divided into primary anomalies, which cannot be linked to an identifiable underlying disease, or secondary manifestations of some other conditions. The latter usually disappears when the underlying condition is treated[1]. In **Primary Hyperlipidemia**, several monogenic disorders have been identified that lead to different types of dyslipidemias, but for many cases the etiology is polygenic. [1,6,8] These disorders affect plasma lipoprotein levels by overproduction of lipoproteins and/or decreased clearance. [1]

Secondary Hyperlipidemia results from several medical conditions associated with mild or even severe hyperlipidemia even in the absence of underlying genetic disorder. [1,14] The table below summarizes common disorders causing lipid abnormalities:

Secondary causes of Lipoprotein Abnormalities[63]

Hypercholesterolaemia

Hypothyroidism; Obstructive liver disease; Nephrotic syndrome; Anorexia nervosa; Acute intermittent porphyria; Drugs: progestones, cyclosporine, thiazides, etc.

Hypertriglyceridemia

Obesity; Diabetes mellitus; Pregnancy; Chronic renal failure; Lipodystrophy; Glycogen storage disease; Alcohol; Bypass surgery; Stress; Sepsis; Acute pancreatitis; SLE; Monoclonal gammopathy; Drugs: estrogen, beta blockers, glucocorticoids, bile acid binding resins, thiazides, etc.

Low HDL

Type 2 diabetes mellitus; Rheumatoid arthritis; Malnutrition; Obesity, Cigarette smoking, Beta blockers, Anabolic steroids, etc.

Manifestations of lipid abnormalities

There may be no symptoms associated with dyslipidemia and it may come to notice only during routine check-up. [1,6,8,63] Individuals might be obese or have an early onset chest pain. [1,6,8,63] Sometimes, lipid abnormalities may be diagnosed for the first time after a

person suffers a myocardial infarction or stroke. [63] Painless nodules called xanthomas may be seen on tendons, elbows or buttocks. These are due to intra and extracellular deposition of cholesterol. [1,6,8,63]

According to the CDC, 97% of adults with diabetes have one or more lipid abnormalities. [64] The central characteristic of dyslipidemia in patients with type 2 diabetes is an elevated triglyceride level, particularly triglyceride-rich VLDL levels and decreased HDL cholesterol levels. [1, 65] In diabetic patients, the concentration of LDL cholesterol is usually not significantly different from that seen in nondiabetic individuals. However, patients with type 2 diabetes typically have a preponderance of smaller, denser, oxidized LDL particles, which may increase atherogenicity, even if the absolute concentration of LDL cholesterol is not elevated. [66, 67]

This lipid triad, referred to as atherogenic dyslipidemia, is usually present in patients with premature coronary artery disease. When this characteristic lipid profile is seen in type 2 diabetes, it is referred to as diabetic dyslipidemia and confers a risk of CVD that equals or exceeds that of a high-risk LDL cholesterol concentration of 150–220 mg/dl.[68]

According to the American Diabetes Association (ADA), the presence of increased triglyceride and decreased HDL levels is the best predictor of CVD in patients with type 2 diabetes. [69] Other predictive factors include a history of cigarette smoking and hypertension.[1]

The evidence regarding the predictive value of elevated LDL cholesterol is still accumulating. Most recently, results of the Strong Heart Study indicate that LDL cholesterol is an independent predictor of CVD in patients with diabetes, along with age,

albuminuria, fibrinogen, HDL cholesterol (inverse predictor), and percent body fat (inverse predictor). Starting with LDL levels as low as 70 mg/dl, every 10 mg/dl increase in LDL cholesterol was associated with a 12% increase in risk of CVD. This finding is supported by results of prospective, long-term clinical trials in which reduction of LDL levels was associated with a significantly reduced risk of cardiovascular events in both diabetic and non-diabetic participants.[71, 72]

There appears to be racial differences in diabetic dyslipidemia.[1] A study done in the U.S. to determine the pattern of diabetic dyslipidemia among African-Americans showed that African-Americans have generally lower LDL-C and higher HDL-C levels when compared to their Caucasian counterparts. [73] Also, diabetic dyslipidemias are more common in females than males. [1] For reasons that are not fully understood, women without diabetes are partly protected from atherosclerosis in their pre-menopausal years; however, they lose much of this protection if they develop diabetes. [65]

Dyslipidemia also occurs as a component of the metabolic syndrome, the other components being hypertension, abdominal obesity, insulin resistance and hypercoagulability. [1,6,8,65] A study done in Nigeria to determine the demographic and clinical correlates of metabolic syndrome in native African type 2 diabetic patients showed that about 72.4% of patients with metabolic syndrome have dyslipidemia. [74] Developing African nations are moving towards Westernization. [19] Affluent societies in these countries are prone to get dyslipidemias as compared to their poor socio-economic counterparts. However, a study done in South Africa to determine the lipid levels in black south Africans with type 2 diabetes showed this not to be the case; it showed that

the lipid levels of affluent and non-affluent societies were more or less the same. [75] This may be explained by the fact that these societies have easy access to quality medical care and dietary modifications, hence tight glycemic controls compared to their non-affluent counterpart.

Treatment of diabetic dyslipidemia

The National Cholesterol Education Program (NCEP), Adult Treatment Panel Third report (ATP III) and the American Diabetes Association have agreed that the target level of LDL-C in patients with diabetes should be 2.6mmol/L (100mg/dL). [1,65,76] Different methods of reducing lipid levels may be employed. The first step is usually to establish the target level that apply to the patient; the second step is to optimize lifestyle and glycemic control, this lifestyle measures include appropriate physical activity and dietary adjustments. [1,65] The diets should be designed to restrict fat intake, particularly saturated fats and cholesterol, and to bring the patients to the ideal body weight. However, these dietary recommendations should consider the patient's needs for glycemic control. The practical application of these principles requires close collaboration with a dietician. [1] If these two steps are not sufficient to reach target levels, then the third step that involves the introduction of lipid lowering drugs should be considered. [1,6,65]

PROBLEM STATEMENT

After a number of diverse and successful measures that have been put forwards towards the control of infectious diseases, non-communicable diseases are emerging as the leading causes of morbidity and mortality in the developing countries. With increasing Westernization in developing countries, Tanzania being one of them, the diabetic population is projected to grow. [15,16] The major cause of death among diabetic patients is cardiovascular disease, which is a result of macrovascular complication of diabetes – the main complication of diabetes. A person with diabetes has a greater risk of heart attack than a person without diabetes. [1]

Dyslipidemia is one of the modifiable atherogenic risk factor whose intervention has been shown to reduce the risk and progression of coronary disease in type 2 diabetic patients. [1, 6,8,65] If dyslipidemia is not regularly screened for among diabetic patients, the morbidity and mortality in this group of patients will increase. This will constraint the already overburdened health services currently existing and hinder economic growth of the country because of the costs involved in treating these patients when the complications have already developed and also due to increased number of days lost from work.

The prevalence of dyslipidemia among diabetic patients in Dar es Salaam is not known; the clinical presentation of dyslipidemia among diabetic patients in Dar es Salaam is not known either. Likewise, whether there is an association between glycemic control and the development of dyslipidemia among diabetic patients in Dar es Salaam is not known.

RESEARCH HYPOTHESES

1. The prevalence of dyslipidemia among diabetic patients in Dar es Salaam is low.
2. Good glycemic control does not affect the development of dyslipidemia among diabetic patients in Dar es Salaam

RATIONALE:

Individuals with diabetes mellitus have several forms of dyslipidemia. Because of the additive and even multiplicative cardiovascular risks of hyperglycemia and dyslipidemia, lipid abnormalities should be aggressively detected and treated as part of comprehensive diabetic care. Dyslipidemias among diabetic patients in Dar es Salaam are frequently overlooked. As such, diabetic patients are not routinely screened and treated for these. The aim of this study was to determine the extent of the problem and hence to emphasize on the importance of including management of dyslipidemia as part of standard of care of diabetic patients.

OBJECTIVES:

BROAD OBJECTIVE:

To determine the prevalence, clinical characteristics and association between dyslipidemia and glycaemic control among diabetic patients in Dar es Salaam

SPECIFIC OBJECTIVES:

1. To determine the prevalence of dyslipidemia among diabetic patients in Dar es salaam

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2. To determine the distribution of lipid profiles of diabetic patients in Dar es salaam.
3. To determine the clinical characteristics of diabetic dyslipidemia among diabetic patients in Dar es Salaam
4. To determine the proportion of dyslipidemic diabetic patients on treatment for dyslipidemia
5. To determine the association between lipid profile and glycaemic control among diabetic patients in Dar es Salaam.

METHODOLOGY:

Study site

The study was conducted in Dar es Salaam, which is the biggest city of Tanzania with a population of about 4 million people. The native population comprises of the Wazaramo and Wandengereko. However, due to urbanization, Dar es Salaam has been inhabited by people from different regions of the country.

The study was conducted in four large government hospitals in Dar es Salaam; Muhimbili National Hospital which is the largest hospital in the country also incorporating the University College of Health Sciences of the University of Dar es Salaam, hence it is also a teaching hospital. There are also three municipal hospitals in which the study was conducted. These included Amana Hospital located in Ilala Municipal Council, Temeke Hospital located in Temeke Municipal Council, and, Mwananyamala Hospital located in Kinondoni Municipal Council. These municipal hospitals serve the populations in the respective Municipal Councils.

Study population: Diabetic patients in Dar es Salaam aged 18years and above

Target population: Diabetic patients attending diabetic clinics at Muhimbili National Hospital, Amana Temeke and Mwananyamala Hospitals.

Study design: Descriptive cross-sectional study

Sample size determination:

The sample size required for this study was determined from the following formula: -

$$n = [Z^2(100 - p)pd] / \varepsilon^2$$

Where, n = required sample size

Z^2 = critical value

p = proportion, according to CDC, 97% of diabetic patients have one or more dyslipidemia

ε = Margin of error (4%)

d = design effect, in this case, 2, i.e., two groups of diabetic patients, dyslipidemic versus non-dyslipidemic were compared with respect to their glycemic control.

It followed that;

$$n = [1.96^2 (100 - 97) \times 3 \times 2]/(4^2)$$

$$= 139.74$$

Therefore, the sample size was 150 patients with diabetes.

Sampling technique:

Patients were selected through simple random sampling, that is, a numbered list of all diabetic patients required to attend the clinic in the day was obtained, and this formed the sampling frame. Then, the sample size required in the day was decided. Patients were selected from this sampling frame using the lottery system of randomization.

Inclusion Criteria: -

The following patients were included in the study;

- Age 18 years and above
- All types of diabetes treatment groups
- Those who signed a written informed or gave a witnessed oral consent
- Patients residing in Dar es Salaam for at least three years

Exclusion criteria: -

The following diabetic patients were excluded from this study;

- Age below 18years
- Those who refused to sign a written informed consent or give a witnessed oral consent
- Diabetic patients referred from up-country
- Expectant mothers
- Known HIV patients on treatment
- Diabetic patients with well established renal diseases

Definition of Terms: -

Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) and the American Heart Association. In this case, derangement in **any** of the lipid component as shown below was considered to be a dyslipidemia, i.e.,

- LDL-C > 2.6mmol/L (100mg/dL)
- HDL-C < 1.1mmol/L (40mg/dL) MEN; 1.38mmol/L (50mmol/dL) WOMEN
- TGs > 1.7mmol/L (150mg/dL)
- Total cholesterol > 5.2mmol/L

Poor glycemic control was defined as any level of glycated hemoglobin $\geq$ 7%.

Body Mass Index (BMI) was calculated by measuring the patient's weight in kilograms and dividing by his/her height in metres squared (kg/m^2). It was classified into underweight, normal, overweight or obese by using cut-offs as[1, 6,8]: -

- Underweight: BMI < 18.50
- Normal: BMI 18.50 – 24.99
- Overweight: BMI 25.00 – 29.99
- Obese: BMI $\geq$ 30.00

Physical Activity was defined as any amount of physical work equivalent to or exceeding that of brisk walking for at least thirty minutes three times a week.

Procedure: -

All patients who agreed to participate in the study were required to sign a written or give a witnessed oral consent. They were then interviewed according to the structured questionnaire. A general physical examination was conducted to detect the presence of clinical features suggestive of dyslipidemia such as the presence of tendon xanthomas and corneal arci. A bathroom scale was used to measure weight and height was measured by tape measure.

A venous blood sample, about 4cc was taken from each subject for lipid profile and glycated hemoglobin assays. This blood was stored in hematological bottles before being sent to laboratory for lipid profile and glycated hemoglobin assays. Total Cholesterol (TC), HDL-C and Triglycerides (TGs) as well as Glycated hemoglobin was measured by Konelab™ machine (© 2003 Thermo Electron Corporation). LDL-C was calculated indirectly from TC, HDL-C and TGs according to the Friedewal formula:

$$\text{LDL-C} = [\text{TC} - \text{HDL-C} - (\text{TGs}/2.2)] \text{ (mmol/L)}$$

Disposal of Patients: -

Results of lipid profile as well as glycated hemoglobin were recorded into patients' files. Those who were found to be dyslipidemic were treated for, as well as counseled on diet, physical activity and adherence to diabetic treatment. Obese diabetic patients were counseled on weight reduction. Patients found to have clinical characteristics of

dyslipidemia such as angina (CHD), intermittent claudication (PVD), were advised for further investigations and referral to specialists in the fields.

Data analysis

Responses in the questionnaires were coded and entered into computer software EPI INFO 6; overall prevalence of dyslipidemia was calculated using frequency distributions. Chi Square test was used to test for association between categorical variables and student's t-test was used for continuous variables. Multiple logistic regression was used to determine the predictors of the dependent variables. A p-value $\leq$ 0.05 was taken to be statistically significant.

Ethical Considerations: -

The research proposal was sent to the Muhimbili University of Health and Allied Sciences Research and Ethics Committee for approval. Permission to conduct the research was obtained from the ethics boards of MNH, Kinondoni, Ilala and Temeke Municipal Councils.

RESULTS

A total of 1824 diabetic patients attended the four diabetic clinics during the study period. Of these, 150 (8.22%) diabetic patients were recruited into the study. Of the 150 diabetic patients, 36 (24%) patients were from Muhimbili National Hospital, 42(28%) from Amana Municipal Hospital, 35(23.3%) from Temeke Municipal Hospital and 37 (24.7%) from Mwananyamala Municipal Hospital (Table 1).

Table 1. Social and Demographic Characteristics of the study participants (n = 150)

Variable	Number	% of Total
Age (years)		
< 20	1	0.67
20 – 30	7	4.67
31 – 40	17	11.33
41 – 50	31	20.67
51 – 60	59	39.33
61 – 70	30	20.00
70+	5	3.33
Sex		
Males	67	44.7
Females	83	55.3
Site		
MNH	36	24.00
Amana	42	28.00
Temeke	35	23.30
Mwananyamala	37	24.70
Duration of Diabetes (years)		
1 – 10	125	83.33
11 – 20	19	12.67
21 – 30	6	4.00
BMI (Kg/m^2)		
< 18.50	7	4.70
18.50 – 24.99	43	28.70
25.00 – 29.99	59	39.30
30+	41	27.30

Of the 150 diabetic patients, 67(44.7%) were males and 83(55.3%) were females. The age range was from 18 to 77 years with a mean of 52.39 ± 11.52 years. All patients admitted a dietary history of carbohydrates, proteins, fats and vitamins. A history of past or current smoking was present in 25/150 (16.7%) patients. A history of hypertension was found in 59/150 (39.3%). The mean duration of diabetes was 6.49 ± 5.42 years with a range of one to 30 years. With regard to the treatment of diabetes, of the 150 diabetic patients, 34 (22.7%) were on insulin, 114 (76.0%) were on oral hypoglycemic agents while 2 (1.3%) patients were on diet only. Only one patient had a prior record of lipid profile. None of the patients was on treatment for dyslipidemia. History of angina was present in 17/150 (11.3%) patients. Likewise, a history of peripheral vascular disease was present in 3/150 (2%) patients. During physical examination; corneal arcus was observed in 32/150 (21.3%) patients. The mean glycated hemoglobin was 6.80 ± 2.61% ranging between 3.10 to 14.2%. The mean weight was 69.91 ± 13.36 kg with a range from 37.00 to 106.00 kg whereas the mean height was 1.60 ± 0.08 metres with a range from 1.08 to 1.81metres.

Dyslipidemia was found in 142 (94.7%) of the 150 study patients. The main dyslipidemia was elevated triglyceride levels, which was present in 142/150 (94.7%) patients. Other dyslipidemias were elevated LDL-C in 49/150 (32.7%), elevated Total Cholesterol in 31/150 (20.7%) and low HDL-C levels in 53/150 (35.3%) patients (Table 2)

Table 2. Frequency Distribution of Dyslipidemia among diabetic patients in Dar es Salaam with respect to Lipid Sub-classes (n = 150)

Lipid Sub-class	No. of Patients	%
High Total Cholesterol	31	20.7
High LDL Cholesterol	49	32.7
Low HDL Cholesterol	53	35.3
High Triglycerides	142	94.7

$\chi^2 = 2.86$; df = 2

Of the 67 male diabetic patients, dyslipidemia was present in 65 (97%) of them, while in the 83 female patients, dyslipidemia was present in 77 (92.8%) patients. This difference did not reach statistical significance (Table 3).

Table 3. Distribution of Dyslipidemia Among Diabetic Patients in Dar es Salaam with respect to sex (n = 150)

Dyslipidemia	Males (n=67)	Females(n = 83)	Total	P value
Overall	65(97.0%)	77(92.8%)	142	0.30
High Total Cholesterol	10(14.9%)	21(25.3%)	31	0.18
High LDL Cholesterol	20(29.9%)	29(34.9%)	49	0.62
Low HDL Cholesterol	28(41.8%)	25(30.1%)	53	0.19
High Triglycerides	65(97.0%)	77(92.8%)	142	0.30

Of the 142 dyslipidemic patients 58 (40.8%) were hypertensive and 25 (17.6%) admitted a history of past or current smoking. Dyslipidemia was also present in all types of diabetes treatment groups. Of the 34 patients who were on insulin, 30 (88.2%) were dyslipidemic; of the 114 patients on oral hypoglycemic agents, 110 (96.5%) were dyslipidemic, while both the two patients on diet only had dyslipidemia. There was no difference regarding the type of treatment on the development of dyslipidemia (Table 4).

Table 4. Distribution of Dyslipidemia With Respect to Diabetes Treatment Groups Among Diabetic Patients in Dar es Salaam (n = 150)

Treatment Group	No. of patients	No. with Dyslipidemia
Insulin	34	30 (88.3%)
Oral Hypoglycemic Agents	114	110 (96.5%)
Diet Only	2	2 (100.0%)

$\chi^2 = 3.65$; df = 2

Also, the site of treatment for diabetes did not affect the development of dyslipidemia (Table 5).

Table 5. Proportion of Dyslipidemic Diabetic patients in Dar es Salaam with respect to the site of treatment for diabetes (n = 150)

Site of Treatment	No. of Patients	No.with dyslipidemia
Muhimbili National Hospital	36	35 (97.2%)
Amana	42	38 (90.5%)
Temeke	35	34 (97.1%)
Mwananyamala	37	35 (94.6%)
Total	150	142 (94.7%)

$\chi^2 = 2.35$; df = 3

All 17/150 (11.3%) patients with angina and all three patients with features of peripheral vascular disease were dyslipidemic. 32 patients presented with corneal arci, out of these, 31 (96.9%) were dyslipidemic. None of the patients presented with tendon xanthomas. 147 patients agreed to have been carrying out their normal physical activities, out of these, 139 (94.6%) had dyslipidemia. On the other hand, all three patients who had no physical activities were dyslipidemic.

Table 6. Clinical Characteristics of Dyslipidemia Among Diabetic Patients in Dar es Salaam (n = 142)

Variable	Number of Patients	%
Hypertension	58	40.8
Smoking	25	16.7
Type of Treatment of Diabetes		
(i)Insulin	30	21.1
(ii)Oral Hypoglycemic Agents	110	77.5
(iii)Diet Only	2	1.4
Angina	17	11.3
PVD	3	2.1
Corneal Arcus	31	21.8
Physical Inactivity	3	2.1

Only one of the 150 diabetic patients studied had a prior record of lipid profile. She had elevated total cholesterol (5.6 mmol/L) and Triglycerides (2.16 mmol/L). She was not on treatment for dyslipidemia. Likewise, of the 142 patients who were found to be dyslipidemic in this study, none of them was on treatment for dyslipidemia.

The mean level of glycated hemoglobin among the dyslipidemic patients was higher (6.926 $\pm$ 2.610%) compared to that of non-dyslipidemic patients (4.538 $\pm$ 1.265%), p = 0.01. In multivariate analysis, even after adjusting for hypertension, smoking, physical

inactivity, duration of diabetes, age and Body Mass Index, the level of glycated hemoglobin was independently associated with the development of dyslipidemia (p = 0.036) (Table 7).

Table 7. Multivariate Analysis; Association Between Different Variables with the Development of Dyslipidemia Among Diabetic Patients in Dar es Salaam (n = 150)

Variable	Regression Coefficient, β	P value
Glycated Hemoglobin	0.618	0.036
Physical Inactivity	7.345	0.957
Smoking	8.044	0.862
Hypertension	1.277	0.277
Duration of Diabetes	0.229	0.215
Age	0.015	0.686
BMI	5.067	0.361

DISCUSSION

The prevalence of dyslipidemia among diabetic patients attending diabetic clinics in Dar es Salaam in this study was 94.7%. The commonest dyslipidemia was elevated triglyceride levels, which occurred in all dyslipidemic diabetic patients in this study. Derangement in other lipid subclasses also was found in this study, the second commonest type of dyslipidemia being depressed HDL–C levels (Table 2). In this study, dyslipidemia occurred equally in both males and females (Table 3). The development of dyslipidemia was also not affected by the type of treatment (Table 4) or the site of treatment for diabetes (Table 5). Dyslipidemia manifested itself clinically by causing angina, peripheral vascular disease and corneal arci. It was present in all patients who were not carrying out physical activity, high Body Mass Indices, patients with a history of smoking, hypertensive patients and in all diabetic treatment groups (Table 6). Only one of study patients had a prior record of lipid profile, and this was done as part of comprehensive chemistry panel when she was admitted at Muhimbili National Hospital. She had elevated total cholesterol (5.6mmol/L) and triglycerides (2.16mmol/L). She was not on treatment for dyslipidemia. Likewise, none of the diabetic patient in this study was on treatment for dyslipidemia. Poor glycemic control as measured by the level of glycated hemoglobin was found to be associated with the development of dyslipidemia. That is, the higher the glycated hemoglobin level the poorer the glycemic control and the higher the derangement in lipid profiles. Even after adjusting for other predictors of dyslipidemia in diabetic patients like physical inactivity, smoking, hypertension, type of

treatment for diabetes, duration of diabetes and Body Mass Index, poor glycemic Control was found to be independently associated with the development of dyslipidemia (Table 7).

The high prevalence of dyslipidemia among diabetic patients found in this study is close to that which was found by Fagot-Campagna *et al* in the U.S., which was 97%[64]. This prevalence appears to be higher than that found by Isezuo *et al* when they were determining the demographic and clinical correlates of metabolic syndrome in native African type 2 diabetics, in which the prevalence of dyslipidemia was 72.4%[74]. This difference could be explained by the higher cut-off values used by Isezuo *et al.* The commonest type of dyslipidemia in this study was elevated triglyceride levels, which occurred in 94.7% of patients. The second common dyslipidemia was lowered levels of HDL-C, which occurred in 35.3%. This type of dyslipidemia occurred as expected, the common derangement in lipid profiles among diabetic patients is usually elevated triglycerides and depressed HDL-C levels[1, 6,8,65].

The lipid levels did not differ regardless of the site of treatment for diabetes. This can be extrapolated to mean there is no difference in the distribution or levels of lipid profiles between diabetic patients residing in urban and rural areas of Dar es Salaam. This finding is very similar to that found in the study by Barry I. Joffe *et al* when they were determining the lipid levels among black South Africans with type 2 diabetes mellitus. It showed that the lipid levels of affluent and non-affluent societies were more or less the same [75].

In this study, there was no gender difference with respect to dyslipidemia. This contradicts with some studies done in the Middle East. For example, Abdul Rahman Al Nuaim *et al* in a study among Saudi diabetic patients found that females were more dyslipidemic than males[77]. M. Nakhjavani *et al* in Iran found out that all types of dyslipidemia were significantly more prevalent in females[78]. This could be explained by the racial and cultural differences between the two populations.

An appreciable proportion of diabetic patients had hypertension. This, together with dyslipidemia increases the cardiovascular diseases risk of Coronary Artery Disease, left ventricular dysfunction and even lead to heart failure[1,6,65]. Dyslipidemia may not present itself clinically, but in this study, some patients presented with angina, features of peripheral vascular disease, cornear arci.

Only one of the 150 patients in this study had a prior record of lipid profile. This was done as part of comprehensive chemistry profile when she was admitted in the ward. She had elevated total cholesterol and triglycerides. She however, was not on any treatment for this. This shows that diabetic patients in Dar es Salaam are not routinely screened for dyslipidemia. Likewise, none of the 142 dyslipidemic patient was on treatment for dyslipidemia. These findings are the same as those in developed countries. A study by *et al* in the U.S. found that only about 28.2% of dyslipidemic diabetic patients were on treatment for dyslipidemia and that only 3% were controlled to target levels [79].

In this study, poor glycaemic control was associated with development of abnormal lipid profiles. These findings are similar to those by A. Caixas et al in which normalization of blood glucose was associated with improvement in the levels of lipids, particularly LDL-

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C[80]. They are also similar to those found by Joffe *et al*, that the prevalence of dyslipidemia rose with worsening glycemic control.[81]

The type of diet, smoking, physical inactivity, hypertension, and duration of diabetes, age and the body mass index (BMI) also affect the development of dyslipidemia. In a study by Stern MP *et al*, glycemic control alone does not suffice to control lipid profile and that a significant number of dyslipidemic patients will need direct lipid management.[81]

In this study, poor glycemic control was independently associated with the development of dyslipidemia even after adjusting for the above predictors in multivariate analysis. The lack of association between these predictors and dyslipidemia in this study can be explained by the fact that in this study, there were very few patients without dyslipidemia, 8/150 (5.3%).

CONCLUSION

1. The prevalence of dyslipidemia among diabetic patients in Dar es Salaam is high
2. The commonest type of dyslipidemia among diabetic patients in Dar es Salaam is elevated triglyceride levels
3. There is lack of awareness and treatment of dyslipidemic diabetic patients among practicing physicians inspite of the propagated knowledge on the decreased rate of coronary events and other cardiovascular diseases with treatment of dyslipidemia.
4. Poor glycaemic control is independently associated with the development of abnormal lipid profiles among diabetic patients in Dar es Salaam.

RECOMMENDATIONS

1. There is a need to periodically screen the diabetic population and implement vigorous therapy for dyslipidemic ones.
2. Treatment of dyslipidemia should be taken as part of the standard of care for diabetic patients. Practicing physicians should be continuously reminded on the importance of including the management of dyslipidemia among diabetic patients.
3. Large studies should be conducted to include patients attending private hospitals for their diabetic care.

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APPENDIX I : QUESTIONNAIRE

THE PREVALENCE, CLINICAL CHARACTERISTICS AND ASSOCIATION BETWEEN DYSLIPIDEMIA AND GLYCEMIC CONTROL AMONG DIABETIC PATIENTS IN DAR ES SALAAM.

1. Study site....(1. MNH 2. Amana 3.Temeke 4. Mwananyamala)
2. Questionnaire No. .....
3. Patient file No.....

SOCIAL AND DEMOGRAPHIC DATA:

4. Name.....
5. Age.....
6. Sex.....(1. Male 2. Female)
7. Residence.....
8. Diet .....

    1.carbohydrates

    2. Proteins

    3. Fats

    4. Vitamins

9. Physical activities..... (1.Yes 2. No)
10. Smoking history..... (1.Yes 2. No)
11. History of Hypertension .....(1.Yes; 2.No)

TREATMENT FOR DIABETES:.

12. Duration of Diabetes……

13. Type of Treatment……..(1.insulin 2.OHA 3.Diet Only 4.Other……)

14. **PRIOR RECORD OF LIPID PROFILE (IF ANY):**

Total cholesterol ……mmol/L

HDL-C ……..mmol/L

LDL-C ……...mmol/L

TGs ……mmol/L

TREATMENT OF DIABETIC DYSLIPIDEMIA (IF ANY):

15. Type of treatment …….

1. "Statins" (mention)……
2. Fibrates (mention)……..
3. Diet only
4. Other (mention)……

16. Duration of Treatment of Dyslipidemia …… months

SYMPTOMS OF DIABETIC DYSLIPIDEMIA:

17. Have you ever experienced the following symptoms…? (1.Yes 2. No)

a) Central/Left sided chest pain radiating to left arm, neck, jaws or back

b) Rest pain in the feet, pain on calf muscles on walking

SIGNS OF DYSLIPIDEMIA

18.Corneal Arcus .....(1. Present; 2. Absent)

19. Tendon Xanthomas .....(1.present; 2. Absent)

PHYSICAL MEASUREMENT:

20.Weight..... Kg

21. Height.....m

22. Body Mass Index (BMI).....Kg/m^2

LABORATORY:

23. Glycated Hb.....%

24. Lipid Profile: -

Total Cholesterol .....mmol/L

LDL-C .....mmol/L

HDL-C .....mmol/L

TGs.....mmol/L

THANK YOU FOR PARTICIPATING IN THIS STUDY!

APPENDIX II: CONSENT FORM, ENGLISH VERSION

Greetings madam/ Sir,

My Name is Dr Stanley Chattanda of the Muhimbili National Hospital.

I am doing a study on Diabetic Dyslipidemia: Prevalence, Clinical Characteristics and association with glycemic control among Diabetic Patients in Dar es Salaam. I am recruiting patients from the four big Government hospitals in Dar es Salaam, Muhimbili National Hospital, Amana, Mwananyamala and Temeke Hospitals.

To consented or witnessed consented diabetic patients, face to face interview and questionnaire filling, general examination will be done which will include weight and height measurement. Then, a blood sample will be taken and sent to the laboratory for lipid profile and glycated hemoglobin estimation.

You will benefit from the study by knowing your blood lipid levels and your glycemic control. However, you will feel some discomfort when a blood sample is taken from your vein.

All information we collect from you will be confidential and will only be used for the purpose of better care and treatment. Your participation is voluntary and you have the right to discontinue from participating in our study at any time. However your decision may be, it will not affect in any way your rights to care and treatment.

I would appreciate if you will agree and take part in this study.

Dr Stanley Chattanda

Investigator.

I _____

Have understood the above information and my questions have been answered to my satisfaction. Willing and without any force, I agree to take part in this study.

Subject:_____

Witness_____

Date:_____

APPENDIX II: CONSENT FORM, SWAHILI VERSION

Habari za saa hizi,

Jina langu ni Dk. Stanley Chattanda, nafanya kazi katika hospitali ya taifa ya Muhimbili. Ninafanya utafiti kwa watu wenye ugonjwa wa kisukari, kuangalia ukubwa wa tatizo la kiwango kikubwa cha mafuta katika damu zao. Utafiti hu unafanyika kwa wagonjwa wa kisukari wanaohudhuria kliniki za kisukari za Hospitali ya Taifa ya Muhimbili, Amana, Mwananyamala na Temeke Hospitali.

Watakaokubali kushiriki itawabidi wasaini fomu kuonyesha wazi kukubali kwao. Pia, tutawahoji maswali kadhaa na kuwapima kwa ajili ya utafiti huu.

Faida utakayopata katika utafiti huu ni kujua kiwango cha mafuta katika damu yako kinachoambatana na kisukari na hivyo kuweza kujifahamu afya yako kwa vizuri zaidi. Mbali na faida hiyo utapata usumbufu kidogo wakati wa kujibu maswali na kutolewa damu kidogo kwaajili ya kupima kiwango cha mafuta.

Taarifa zote utakazotupa ni siri na zitatumika tu kwa ajili ya kuboresha huduma ya afya na utabibu kwa wagonjwa na wananchi kwa ujumla. Ushiriki wako ni kwa hiari kabisa na pia unayo haki ya kujitoa katika utafiti huu wakati wowote ule utakapojisikia kufanya hivyo. Uamuzi wako wa kushiriki ama la, katika utafiti huu, hautaadhiri hata kidogo haki yako ya kupata huduma nzuri za afya kama raia.

Nitafurahi ukikubali kushiriki katika utafiti huu.

Dr Stanley Chattanda

Mtafiti.

Mimi _____

Nimeelewa maelezo yaliyoandikwa hapo juu na kuridhika na majibu niliyopewa kwa maswali yangu yote. Mimi kwa hiari yangu mwenyewe, bila kushurutishwa na mtu, ninakubali kushiriki kwenye utafiti huu.

Mgonjwa:_____

Mdhamini_____

Tarehe:_____

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