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EXAMINING GLYCAEMIC CONTROL STATUS AND ASSOCIATED FACTORS IN DIABETES MELLITUS OUT-PATIENTS AT THE UNIVERSITY TEACHING HOSPITAL, LUSAKA, ZAMBIA

by

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A dissertation submitted in partial fulfillment of the requirements of the degree of Master of Science in Human Physiology

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DECLARATION

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CERTIFICATE OF APPROVAL

This dissertation of EMMANUEL MWILA MUSENGE on EXAMINING GLYCAEMIC CONTROL STATUS AND ASSOCIATED FACTORS IN DIABETES MELLITUS OUT-PATIENTS AT THE UNIVERSITY TEACHING HOSPITAL, LUSAKA, ZAMBIA has been approved in partial fulfillment of the requirements for the award of the Degree of Master of Science in Human Physiology by the University of Zambia.

Examiner I

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Examiner II

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ABSTRACT

Background: Diabetes mellitus is one of the main growing public health concerns worldwide. There are two major types of diabetes mellitus. Type 1 diabetes mellitus which appears early in life and Type 2 diabetes mellitus which appears in adult life. It is important to regularly monitor the glycaemic control status in diabetic patients so as to manage their condition better. This improves the patient's quality of life, delaying and preventing complications and deaths due to diabetes mellitus. Monitoring of glycaemic levels can best be achieved by HbA_{1c} test as it comprehensively evaluates glycaemic control status in the past 8 to 12 weeks. In this study, the researcher examined the glycaemic control status and associated factors in diabetic out-patients at the University Teaching Hospital in the Lusaka province of Zambia.

Methods: An institutional-based cross-sectional study was conducted at the University Teaching Hospital diabetic clinic. A simple random sampling method was used to sample diabetic out-patients from within Lusaka. The data, specimens and anthropometric measurements were collected from patients who consented for enrolment from September to November 2013. Immunoturbidimetry and Trinder colorimetric techniques were used to measure the levels of HbA_{1c} and fasting plasma glucose respectively. A structured interview schedule was used to capture data. Binary logistic regression analysis of the data was carried out using IBM® SPSS® Statistics for Windows version 20.0 to predict factors influencing glycaemic control status of diabetic out-patients.

Results: A total of 198 patients were sampled and out of these, 75 (38.7%) had good glycaemic control status ($HbA_{1c} \leq 48$ mmol/mol) and 119 (61.3%) had poor glycaemic control status ($HbA_{1c} \geq 49$ mmol/mol) in those whose data was complete. In addition, the mean of the previous and current fasting plasma glucose was 10.71 ± 7.75 mmol/L and 10.98 ± 6.22 mmol/L respectively. The glycaemic control status was associated with adherence to anti-diabetic treatment, type of anti-diabetic treatment, systolic blood pressure and fasting plasma glucose.

Conclusion: This study established that non-adherence, insulin treatment and raised blood pressure and fasting plasma glucose influenced HbA_{1c} levels. At the University Teaching Hospital, there is need to consider full scale use of HbA_{1c} for glycaemic control monitoring in diabetic out-patients alongside other tests as this will consequently improve the management of the diabetic patients. Further studies should evaluate the role of diabetic patients in the management of their disorder and consider a large scale study and compare glycaemic control status between types 1 and 2 diabetic patients and associated factors.

This dissertation is dedicated to the diabetes mellitus patients who have experienced unsound health and death due to the disorder and the important people who have been a source of inspiration in my life; my parents, Mr. Fidelis M. M. Nsofwa and Mrs. Annie Nsofwa; my wife, Constance Simooya Musenge; my daughter and son, Natasha Regina Mutale Musenge and Emmanuel Mwila Musenge Jr.

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ACRONYMS AND SYMBOLS

µL……...Microlitre

AAP…….. Aminoantipyrine

ACE……...American College of Endocrinology

ADA……..American Diabetes Association

AG…….Average Glucose

BMI…….....Body Mass Index

CAL……Calibrator

CI……....Confidence Interval

Co……Company

DBP……..…Diastolic Blood Pressure

DCCT……..Diabetes Control and Complications Trial

DM…… ……..…Diabetes Mellitus

eAG……..…estimated Average Glucose

EASD……..European Association for the Study of Diabetes

EDTA……..……Ethylenediaminetetraacetic acid

FPG…….....……Fasting Plasma Glucose

GOD.....Glucose Oxidase

H_2O…….Water

H_2O_2……Hydrogen Peroxide

Hb……Haemoglobin

HbA…….Adult Haemoglobin

HbA_{1c}…….…..……Glycosylated Haemoglobin

IBM…….International Business Machine

IDF…….……International Diabetes Federation

IEC…….……Information Education and Communication

IFCC……International Federation of Clinical Chemistry

HMIS……..…….Health Management Information System

kg…….……kilogram

Ltd…….…..Limited

m…….Metre

MEPI…….…..Medical Education Partnership Education

Milligrams/deciliter……...mg/dL

Milligrams……..…..mg

mm Hg…….…...Millimetres of Mercury

mmol/L……...……Millimoles/Litre

Mmol/mol……..……..Millimoles/mole

MoH……..……..Ministry of Health

NCDs……..…....Non Communicable Diseases

NGSP…….……..National Glycohaemoglobin Standardization Program

NHSP…….....…….National Health Strategic Plan

nm……..…….nanometre

O_2……..…….....Oxygen

OADs…….……..Oral Antidiabetic Drugs

OD…….……..Optic Density

OR……..…….Odds Ratio

p…….……..Probability

PAP……...Phenol + Aminophenazone

pH.....Potential Hydrogen

POD.....Peroxidase

RBC……..…..Red Blood Cell

RBG…….....Random Blood Glucose

Ref……Reference

RPM…….Revolutions Per Minute

SACORE……..Southern African Consortium for Research Excellence

SBGM……...Self-Blood Glucose Monitoring

SBP…….……..Systolic Blood Pressure

SD……...…..Standard Deviation

SDO……..……Staff Development Office

SE……..Standard Error

SI…….....Système International (SI) units

SPSS…….....Statistical Package for Social Scientists

STEPS……..…….STEPwise approach to Surveillance

THb……Total Haemoglobin

UK…….……United Kingdom

UNZA……University of Zambia

UNZABREC…….University of Zambia Biomedical Research Ethics Committee

USA……United States of America

UTH……...…….University Teaching Hospital

WHO……..…..……..World Health Organization

X^2.....Chi-square

CHAPTER ONE

1.0. Introduction

1.1. Background

Diabetes mellitus (DM) is one of the major causes of premature illness and death worldwide (World Health Organization [WHO], 2010). The WHO predicts that, developing countries to which Zambia belong will bear the brunt of this epidemic in the 21st century due to changes in life style. Currently, more than 70 per cent of people with DM live in low and middle income countries (Sicree et al., 2010).

Diabetes mellitus is characterised by hyperglycaemia either because of insulin deficiency from the pancreas or because of insulin resistance by the body cells or both (Gardner, 2011). There are generally two major types of DM; type 1 which accounts for fewer than 10 per cent of all cases of DM and type 2 which account for about 90 per cent of all cases of DM (Green, Flatt and Bailey, 2006). Diabetes mellitus may be associated with environmental and genetic predisposition among other factors (van Dam, 2003; Hirschhorn, 2003).

The control of DM has proved to be difficult as those already with the disease are unable to monitor and maintain near normal glycaemic levels. Among the factors which influence glycaemic control status include education, body mass index (BMI), Self-blood glucose monitoring (SBGM), diabetes duration, adherence to antidiabetic treatment regimen, type of antidiabetic treatment, physical inactivity, co-morbidity anaemias and haemoglobinopathies such as Hb S and C (Hartz et al., 2006) and certain drugs such as septrin, aspirin and antiretroviral drugs (Unnikrishnan, Anjana and Mohan, 2012). Diabetes mellitus has serious consequences especially if not controlled soon. Uncontrolled DM can put the patient at risk for a host of complications that can affect nearly every organ in the body due to damage to the blood vessels, nerves, or both such as cardiac failure, retinopathy, nephropathy, neuropathy and the gums and teeth disorders.

Glycaemic control monitoring can be achieved by fasting plasma glucose (FPG) and glycosylated or glycated haemoglobin (HbA_{1c}) among other test methods.

Fasting plasma glucose test ascertains the glucose levels for the past few days but since blood glucose levels fluctuate throughout the day, glucose records are imperfect indicators of changes in the body due to hyperglycaemia. According to the medical staff and medical record review at the University Teaching Hospital (UTH), most DM patients come to diabetic clinic for follow-up visits infrequently. For most of these patients, their blood glucose monitoring is done only on the day of visit to the clinic. The progressive nature of DM requires continuous monitoring of glycaemia and, when necessary, intensification of any existing treatment (Cox and Edelman, 2009). Thus, achieving and maintaining good glycaemic control is essential for reducing the risk of incidence and progression of DM-related complications (Jang, Guler and Shestakova, 2008).

The glycaemic control status in DM patients can be best ascertained by HbA_{1c} levels. This long-term assessment of blood glucose is advantageous not only because it eliminates the large fluctuations that occur daily in blood glucose concentrations, but in contrast to FPG, HbA_{1c} also provides an accurate result from blood drawn at any time of day without reference to prandial state (Burtis, Ashood and Bruns, 2006). The frequency of glycaemic monitoring depends on the type of DM the patient has and the treatment plan. However, it is recommended that HbA_{1c} levels be checked approximately quarterly in uncontrolled or at least half yearly in well-controlled DM patients.

Most countries in the world are using HbA_{1c} for both diagnosis and long-term monitoring of DM to establish glycaemic control status. Zambia has not been using HbA_{1c} fully in either case especially in government health facilities. Some private health facilities are using HbA_{1c} but the cost is beyond the reach of an ordinary Zambian DM patient. Glycosylated Hb is a useful indicator of how well the blood glucose levels have been controlled in the recent past and may be used to monitor the effects of diet, exercise, and drug therapy on blood glucose in DM patients.

It has been demonstrated that the complications of DM can be delayed or prevented if the HbA_{1c} levels can be kept close to 48 mmol/mol (Green, Flatt and Bailey, 2006). While diabetic patient treatment goals vary, many include a target range of HbA_{1c} values. Therefore, HbA_{1c} can help determine how well a person's DM is being controlled over time and this can help to prevent or delay DM complications.

1.2. Literature Review

The literature review in this study is focused on epidemiology of DM, description of HbA_{1c} and FPG, glycaemic control status and the factors associated with the control status in DM patients.

1.2.1. Diabetes Mellitus Epidemiology

The worldwide prevalence of DM among adults was 285 million (6.4%) in 2010 and is predicted to rise to around 439 million (7.8%) by 2030 (Sicree et al., 2010). According to WHO (2010), the percentage of deaths attributable to DM in the whole world was 5.5 per cent in 2010. The prevalence varies from 10.2 per cent in the Western Pacific to 3.8 per cent in the African region. Africa is expected to experience the highest increase in deaths due to DM in future (WHO, 2010). The sub-Saharan Africa adult population of about 296 million has an estimated adult DM prevalence of 2.4 per cent (Sicree et al., 2010). The overall DM prevalence in Zambia was at 2.6 per cent and DM contributed to over 30 per cent bed occupancy (Ministry of Health [MoH], 2010). This is slightly above the sub-Saharan Africa and below the African region DM prevalence.

1.2.2. Description of HbA_{1c} and FPG

Glycosylated Hb is formed by the non-enzymatic glycation of free amino groups at the N-terminus of the β-chain of adult Hb (HbA) by HbA's exposure to plasma glucose (Kilpatrick, Bloomgarden and Zimmet, 2009). Glycosylated Hb is a gold standard in analysis of DM patients' glycaemic control status, and is essential to ensure optimal care of DM patients (Ghazanfari et al. 2010a; ADA, 2010). Since the red blood cells (RBCs) in the human body survive for 8 to 12 weeks before renewal, measuring HbA_{1c} can be used to reflect average blood glucose levels over that duration, providing a useful longer-term gauge of glycaemic control (Roszyk et al., 2007). Nonetheless, since the half-life of RBCs is reduced to almost half in haemoglobinopathies, the HbA_{1c} levels may not reflect the true glycaemic control in such patients. The level of HbA_{1c} is proportional to the level of glucose in the blood. Thus, normal levels of glucose produce a normal amount of HbA_{1c}. As the average amount of plasma glucose increases, the fraction of HbA_{1c} increases in a predictable way (Roszyk et al., 2007).

Glycosylated Hb has traditionally been reported as a percentage of total Hb as per the Diabetes Control and Complications Trial (DCCT) of United Kingdom (UK) and the National Glycohaemoglobin Standardisation Program (NGSP) units (percentage, one decimal place) of Australia. However, since June 2011, the way HbA_{1c} values are reported has switched from a percentage to a measurement in mmol/mol.

The International HbA_{1c} Consensus Committee comprising the American Diabetes Association (ADA), European Association for the Study of Diabetes (EASD) and International Diabetes Federation (IDF) among others recommends that all HbA_{1c} levels be reported in Système International (SI) units of millimoles of HbA_{1c} per mole of Hb (mmol/mol, no decimal places), with results directly traceable to the International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) standardised reference method (Hanas and John, 2010). There is a linear relationship between results from the two methods (per cent and mmol/mol), and the following formulas are used to convert results between the two methods:

i. HbA_{1c} SI unit (mmol/mol) = 10.93 x HbA_{1c} NGSP or DCCT unit (per cent) – 23.50 mmol/mol.

ii. HbA_{1c} NGSP or DCCT per cent unit = 0.0915 x mmol/mol + 2.15 per cent

The approximate mapping between HbA_{1c} values given in DCCT percentage and estimated average glucose (eAG) measurements is also given by the following equation (Nathan et al., 2008):

i. $eAG(mg/dl) = 28.7 \times HbA_{1c} - 46.7$

ii. $eAG(mmol/l) = 1.59 \times HbA_{1c} - 2.59$

The International Diabetes Federation (IDF) and American College of Endocrinology (ACE) recommend HbA_{1c} values below 48 mmol/mol (IDF, 2010) as a therapeutic objective. Using the ABX Pentra 400 discrete photometric benchtop Clinical Chemistry Analyser, which technique has been NGSP-certified, the standard value for HbA_{1c} is between 20 to 42 mmol/mol (Burtis, Ashood and Bruns, 2006). Using an NGSP-certified method for analysis ensures that the results are standardised. The ADA's new recommendations set HbA_{1c} levels as follows: less than 39 mmol/mol normal, greater than 48 mmol/mol for diagnosis of DM and levels between 39 and 47 mmol/mol as an indication of increased risk for diabetes (ADA, 2010).

However, some studies have suggested that HbA_{1c} levels below the recommended targets may be excessive as the health benefits of reduced HbA_{1c} become smaller, and the intensive glycaemic control required to reach this level leads to an increased rate of dangerous hypoglycaemic episodes (Lehman and Krumholz, 2009).

On the other hand, a retrospective study of DM patients found that patients with an HbA_{1c} more than 48 mmol/mol had an increased mortality rate, but a later international study contradicted these findings (Roszyk et al., 2007). Research has shown that, a one per cent change in HbA_{1c} is equivalent to an approximately 1.94 mmol/L change in mean plasma glucose. Smaller values of HbA_{1c} indicate better blood glucose control. With each one per cent reduction in the value of HbA_{1c}, the risk of microvascular complications is reduced by 40 per cent (Ghazanfari et al. 2010b).

Fasting plasma glucose is the level of glucose in a person's blood plasma after a period of fasting. The normal FPG levels are between 4.1 to 5.9 mmol/L when using the ABX Pentra 400 discrete photometric benchtop Clinical Chemistry Analyser (Burtis, Ashood and Bruns, 2006). Moreover, the FPG measured in milligram per decilitre (mg/dL) can be converted into mmol/L which is the SI units by dividing the value in mg/dL by 18. Thus, 18 mg/dL = one mmol/L.

1.2.3. Glycaemic Control and Associated Factors

There is a linear relationship between HbA_{1c} and FPG. In a cross-sectional study conducted by Kahlon and Pathak (2011) among 300 known DM out-patients in India on the patterns of blood glucose control, majority of the patients had FPG level $\geq$ 6.9 mmol/L (63%) and HbA_{1c} level $\geq$ 59 mmol/mol (87%). However, in another study by Liberopoulos et al. (2010) in Greece to compare HbA_{1c} and FPG for the diagnosis of diabetes among 142 individuals with metabolic syndrome, most (54.9%) and only a few (38.7%) of the patients were considered diabetic based of HbA_{1c} and FPG. Both studies showed that blood glucose control in diabetics can be better assessed with HbA_{1c} and FPG together. A positive correlation between HbA_{1c} and FPG especially in the former study allows for the periodic estimation of HbA_{1c} along with FPG in the management of DM so as to control blood glucose but the two should not be used interchangeably.

The monitoring of glycaemia in DM patients to prevent and anticipate complications early need no to be emphasised. A study conducted in Nigeria by Adebisi et al. (2009) on HbA_{1c} and blood glucose control of DM showed that most (64%) of the patients had poor glycaemic control (HbA1c $\geq$ 55 mmol/mol). In another study by Arthur et al. (2006) among 99 DM patients attending a Diabetic Clinic at the Komfo Anokye Teaching Hospital in Ghana, results showed that 64 per cent of the DM patients had poor glycaemic control. In addition, Sobngwi et al. (2012) in a cross-sectional, descriptive study of 2352 type-2 diabetes patients who were treated at specialist clinics for at least 12 months prior to the study, in six sub-Saharan countries revealed that only 29 per cent of the patients who had their HbA_{1c} assessed in the past year had good glcaemic control (HbA_{1c} < 48 mmol/mol).

Cohen et al. (2010) in a study that was done in patients attending the diabetic clinic at a teaching hospital in Malawi to describe the current status of diabetes care in an urban diabetes clinic and the prevalence of human immunodeficiency virus (HIV) in this population investigated the possible associations between HIV and diabetes. The results showed that, most (74%) of the patients had poor glycaemic control (HbA_{1c} $\geq$59 mmol/mol). In Zambia, the stepwise survey (STEPS) Non-communicable Diseases (NCDs) Risk Factors Survey which was carried out in Lusaka-Zambia in 2008 showed that, eight per cent of the studied population had raised blood glucose with three per cent diabetes prevalence in males and four per cent in females (MoH, 2010).

Most of the studies have revealed that poor management of DM can lead to an array of complications and premature death. Microvascular complications specifically nephropathy (34.7%) (Cohen et al., 2010), cataract (14%), neuropathy (48) retinopathies (46.4% and 18%) were common in these studies (Cohen et al., 2010; Sobngwi et al. 2012). These studies provide evidence to support appropriate interventions to diabetic populations early in the disease. This suggests that a relatively large proportion of DM patients could be predisposed to microvascular complications, while a small group with near-normal HbA_{1c} levels could be prone to hypoglycaemic complications. Thus, the measurement of HbA_{1c} becomes important in the assessment of blood glucose with the view to prevent complications.

The management of DM requires that the factors that influence glycaemic control are as well taken into consideration. In a study conducted by Moreira Jr. et al. (2010) in Venezuela, 4075 patients were surveyed of which 8.6 per cent had type 1 DM and 91.4 per cent had type 2 DM. Similarly, Mendes et al. (2009) in Brazil revealed that, out of the 6,701 DM patients in the study, 15 per cent had type 1 DM and 85 per cent had type 2 DM. The overall prevalence of inadequate blood glucose control status was 76 per cent. However, in the UK, Higgins, Khan and Pearce (2007) in their cross-sectional study among DM patients attending the eye clinic showed that, 25 per cent had type 1 DM and 75 per cent had type 2 DM.

The majority of the patients in these studies had type 2 DM as expected. Nonetheless, most of the patients who had type 1 DM had poor glycaemic control, 87 per cent (Moreira Jr. et al., 2010) and 90 per cent (Mendes et al., 2009). On the other hand, Higgins, Khan and Pearce (2007) reported that, five of 11 (46%) type 1 DM patients had poorly controlled diabetes (HbA_{1c} > 75 mmol/mol) compared with four of 33 (12%) type 2 DM patients. However, this study still revealed a higher percentage of poor glycaemic control among type 1 DM patients compared with type 2 DM.

Furthermore, Mahmood and Aamir (2005) in Pakistan assessed the status of glycaemic control in type 2 DM patients and results revealed that about half (51.43%) of the patients had poor control of DM and the remaining had either good (31.43%) or fair (17.14%) glycaemic control. Similarly, Bi et al. (2010) in a study to determine the status of glycaemic control and associated factors in 2966 patients with type 2 DM in China revealed that 59.8 per cent had poor glycaemic control. In another study by Otieno, Kariuki and Ng'ang'a (2003) in Kenya to determine the glycaemic control of 305 ambulatory type I and 2 diabetic patients, results showed that 60.5 per cent of the patients had poor glycaemic control (HbAlc > 64 mmol/mol) while 39.5 per cent had good glycaemic control status (HbAlc < 64 mmol/mol).

Also, Erasmus et al. (1999) in a study among type 2 black diabetics attending the diabetic clinic at a peri-urban hospital in South Africa revealed that, out of 708 patients, 79.9 per cent had poor glycaemic control (HbA_{1c}≥53 mmol/mol) while 20.1 per cent had poor glycaemic control.

In Rwanda in a study among 286 type 1 DM patients by Marshall et al. (2012), results revealed that only 15.7 per cent had good glycaemic control (HbA1c < 64 mmol/mol).

Although, a good number of the literature showed that glycaemic control status was poor among DM patients, in Japan and Germany (Arai et al. 2009; Reisig et al. 2007), the glycaemic control status of the patients was good. This is probably attributed to adequate knowledge about DM.

Several factors besides the type of DM the patient has, have been found to influence good glycaemic control status of the patients. In the studies reviewed, older age, duration of DM (Ahmad, Islahudin and Paraidathathu, 2013; Mendes et al., 2009; Bi et al., 2010), monotherapy, combination of oral and insulin (Ahmad, Islahudin and Paraidathathu, 2013; Bi et al., 2010), oral antidiabetic drugs, tertiary hospital (Bi et al., 2010), diet only because of possible fair endogenous insulin production (Otieno, Kariuki and Ng'ang'a, 2003), satisfaction with current DM treatment (Moreira Jr. et al., 2010; Mendes et al., 2009), lower BMI, more education, higher income (Bi et al., 2010) were associated with good glycaemic control status. However, good control was also found among young DM patients (Bi et al., 2010) and DM duration, insulin dose/kg and geographical location were significantly associated with poor glycaemic control status (Marshall et al., 2012).

Conversely, poor glycaemic control status was associated with oral hypoglycaemic agents-only (Otieno, Kariuki and Ng'ang'a, 2003), both gender, obesity (Erasmus et al., 1999; Moreira Jr. et al., 2010), multi-professional care, and participation in a diabetes education program (Moreira Jr. et al., 2010; Mendes et al., 2009). Nonetheless, Bi et al. (2010) reported good glycaemic control among the patients who attended DM education. Despite increased awareness of the benefits of tight glycaemic control, few DM patients in Brazil met recommended blood glucose control targets (Mendes et al., 2009). In addition, antihypertensive medication, uncontrolled blood pressure (> 150/85 mmHg treated; > 160/90 mmHg untreated) and serum cholesterol levels > 5.2 influenced glycaemic control status of the DM patients (Higgins, Khan and Pearce, 2007).

The literature suggest that behavioural changes through health educational programmes need to be instituted with both patient and medical personnel being motivated to take this process forward. Although some self-management behaviours did not appear to influence glycaemic control, DM patients should be consistently advised to restrict sugar intake, involve themselves in exercises and adhere to medication instructions. Greater effort by healthcare providers in the primary health clinics is warranted to help a greater number of patients achieve good glycaemic control.

It can be concluded therefore that, the majority of the DM patients had poor control of their glycaemia status. This implies that there are probably problems in the management of DM patients. The studies have revealed that, avoidance of the factors that influence glycaemic control status by DM patients can help improve the glycaemic control status of patients. Also, staff motivation in the management of DM patients has shown to improve glycaemic control status of DM patients. Thus, control of HbA_{1c} and paying attention to the factors associated with glycaemic control status can slow down the progression of microvascular disorders and other DM end-points. Many of the DM patients had poorly controlled DM in terms of these risk factors. The studies suggest that, many of the long-term complications of DM especially the microvascular complications, result from many years of hyperglycemia.

Zambia is undergoing significant socio-demographic and technological transition that go with urbanization and industrialization. This will cause an epidemiological transition from communicable diseases to NCDs including DM which phase the country need to start preparing for. There is paucity of data in Zambia on the determination of glycaemic control status in DM patients using HbA_{1c}. The study may assist in improving the management of DM patients in the country.

CHAPTER TWO

2.0 Research Focus

2.1. Statement of the Problem

The pattern of glycaemic control status and associated factors in DM out-patients at UTH has not been well established. A preliminary survey at diabetic clinic suggest that most of the DM out-patients who visit UTH do not monitor their blood glucose levels at home as it is not feasible for them. Only a few who can afford a glucometer are able to do so. Some rely on the nearest health centres to monitor their blood glucose levels. While others only have their blood glucose levels checked a day or two before their follow-up visit to the diabetic clinic. Currently, at UTH, glycaemic control status and monitoring among the DM patients is evaluated through random blood glucose (RBG) and FPG. A few who can afford the HbA_{1c} test have it measured at the University Teaching Hospital (UTH) high cost (Premier) laboratory and private laboratories. According to Zemlin et al. (2011) blood glucose control status can best be assessed using HbA_{1c} test as it is a more comprehensive measure of total blood glucose exposure than other glucose tests.

The DM patients have varied frequencies on their follow-up visits to the DM clinic. The controlled patients visit the clinic at least quarterly while the unstable visits the clinic whenever necessary and those who do not stabilize are admitted for purpose of controlling their glucose levels. Diabetes Mellitus is ranked number eight among the top 11 causes of morbidity and mortality at UTH (UTH, 2010). There has been an increase in the number of admissions and deaths due to DM at UTH (Table 1).

Table 1: DM Admissions and Deaths at the University Teaching Hospital

Year	Medical Conditions Admissions	Diabetes Mellitus Admissions	Diabetes Mellitus Deaths
2008	10,561	545 (5.16%)	113 (20.7%)
2009	9,367	484 (5.17%)	103 (21.3%)
2010	7,255	561 (7.7%)	114 (20.3%)

(Source: UTH, 2010)

However, the causes for DM morbidity and mortality at UTH are not fully understood. It is also not clear as to whether there is an association between glycaemic control status and morbidity and mortality. Perhaps predictably, the patients seen and finally admitted come in serious condition.

The extent to which glycaemic control status can be assessed using HbA_{1c} in Zambia is yet to be done, hence the need to carry out the study. The study would therefore shade more light on blood glucose control status in DM out-patients at UTH.

2.2. Justification of the Study

Glycosylated Hb informs health care providers of the blood glucose levels for the preceding 8 to 12 weeks (Roszyk et al., 2007). Therefore, the health care providers will be able to know the long-term glycaemic control status in the DM out-patients. This is so because the current methods of assessing glycaemic control status with the RBG or FPG at UTH look at the control for a few days without regard to daily fluctuations. This study will make it possible and assist health-care providers such as the medical officers, nurses, clinical officers, biomedical scientists among others to review their management of DM patients.

Much of the studies done in Zambia and particularly at UTH have not focused on glycaemic control status. Besides, not much is also known about the glycaemic control status of the DM out-patients at UTH. This information will be important to various actors who may be willing to intervene and assist in improving the management of DM patients at UTH and the country as a whole.

2.3. Research Question

What is the glycaemic control status and associated factors in DM out-patients at UTH?

2.4. Objectives

2.4.1. General Objective

To determine the glycaemic control status and associated factors in DM out-patients attending diabetic clinic at UTH.

2.4.2. Specific Objectives

2.4.2.1. To measure the HbA_{1c} levels in DM out-patients.

2.4.2.2. To measure the FPG levels in DM out-patients.

2.4.2.3. To establish the relationship between HbA_{1c} and FPG levels in DM out-patients.

2.4.2.4. To identify the demographic, self-management behaviours and clinical factors that may be associated with HbA_{1c} levels.

2.5.0. Operational/Conceptual Definition of Terms

2.5.1 Diabetes mellitus: A group of metabolic disorders characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both (Green, Flatt and Bailey, 2006).

2.5.2 Glycaemic control status: A medical term referring to the typical levels of blood glucose in a person with diabetes mellitus (Parving et al., 2008).

2.5.3 Glycosylated haemoglobin: Haemoglobin formed by the non-enzymatic covalent bonding of adult Hb with glucose molecule.

2.5.4 Fasting plasma glucose: Plasma glucose levels after not eating or drinking anything other than water for eight hours to twelve hours (usually overnight).

2.5.5 Immunoturbidimetry: Interaction between antigen molecules (HbA_{1c}) and HbA_{1c} specific antibodies coated on latex beads resulting in changes in the solution turbidity which is proportional to the amount of the HbA_{1c} in the samples.

2.5.6 Trinder: Colour change test resulting from the Trinder reaction.

2.5.7 Colorimetry: Analysis of chemical samples to collect information about their concentration.

2.5.8 Good glycaemic control: The HbA_{1c} levels of less than or equal to 48 mmol/mol over months or years.

2.5.9 Poor glycaemic control: The HbA_{1c} levels greater than 49 mmol/mol over months or years before severe complications occur.

2.5.10 Non-communicable diseases: The diseases of long duration and generally slow progression.

2.6.0. Research Variable and Cut-off Points

The dependent variable which was used to examine the glycaemic control status and associated factors in DM out-patients in this study is HbA_{1c} (Table 2).

Table 2: Research Variable and Cut-off Points

Variable	**Scale of measurement**	
	Indicator	**Cut-off point**
HbA_{1c}	Good	HbA_{1c} levels ≤ 48 mmol/mol.
	Poor	HbA_{1c} levels ≥ 49 mmol/mol.

(Source: IDF, 2010)

CHAPTER THREE

3.0. METHODLOGY

3.1. Study Design

An institutional based out-patient cross-sectional study design was used in this study.

3.2. Study Setting

The study was conducted at UTH clinic five in the Lusaka District of Zambia. The UTH has a bed capacity of 1800 and serves as the main tertiary referral Hospital for the country (Lumba, 2014). Clinic five comprises diabetic clinic among others at the UTH out-patient department. The out-patients department of the Hospital sees about 9600 patients a year (UTH, 2010). The site was selected purposively because of the convenience and ease accessibility to the facilities.

3.3. Study Population

The study population included all the DM out-patients who visit UTH for purposes of managing their DM.

3.3.1 Target Population

The target population included all the DM out-patients who visited UTH during the period of the study for purposes of managing their DM.

3.3.2. Accessible Population

The accessible population for this study was the target populations who met the criteria and were willing to participate in the study.

3.4. Sample Selection and Sample Size

3.4.1. Sample Selection

The sampling frame comprised about 360 DM out-patients who visited diabetic clinic during the three months of data collection. Every morning during the study period the clerk used to make a list of the already confirmed diabetic patients coming for follow-up visits to UTH diabetic clinic. This list was used as a sampling frame for that day after excluding those who did not meet the criteria.

The patients were identified and recruited consecutively for three months so as to eliminate selection biases.

During the study period, diabetic clinic used to receive on average nine DM out-patients per day. The patients in the study were selected using lottery simple random sampling method. Small and identical pieces of paper with numbers written on them from one to the number of patients on that day were put in a box after folding them. The numbers represented the patients as listed on the sampling frame. The pieces of paper were then mixed thoroughly together by shaking the box. Then one piece of paper was blindfold picked at a time without replacement until three patients were selected per day and finally the required sample size for the study was reached. The method ensured that each patient had an equal chance of being included in the sample and this was feasible in terms of time, human, financial and material resources.

3.4.1.1. Inclusion criteria

- Confirmed diagnosis of DM with out-patients follow up in diabetic clinic.
- Aged 15 years and above.
- Informed written consent given.

3.4.1.2. Exclusion Criteria

- Newly diagnosed cases of DM out-patients who visited diabetic clinic.
- Confirmed DM out-patients who were recruited in the previous month(s).
- Refusal to give consent

3.4.2. Sample Size Calculation

$s = X^2NP\ (1-P) \div d^2\ (N-1) + X^2P\ (1-P)$

Where;

s = required sample size.

X^2 = the table value of chi-square for 1 degree of freedom at the desired confidence level of 0.05 ($1.96^2 = 3.84$).

N = the population size.

P = the population proportion (assumed to be 0.50 since this would provide the maximum sample size).

d = the degree of accuracy expressed as a proportion (0.05).

Choosing

X = 1.96

N = 360

P = 0.50

d = 0.05

$s = 3.84*360*0.5\ (1-0.5) \div 0.05^2\ (360-1) + 3.84P\ (1-0.5)$

s = 186

The sample size was calculated based on the 360 DM out-patients who passed through diabetic clinic five during the period of data of data collection. Based on Krejcie and Morgan's (1970) formula for calculating sample size of a finite population, this gave a calculated sample size of 186 participants. However, a higher number was targeted in order to account for possible exclusions due to refusal to give consent, very sick and the need to carry out subgroup analysis.

3.5. Data Management

3.5.1. Data Collection Tool

A structured interview schedule was used to collect demographic data, self-management behaviour data, clinical data, and entering laboratory measurement results of all the patients under study. The tool was based on the WHO STEPS instrument (WHO, 2007).

3.5.1.1. Validity

To ensure validity, all the independent variables as well as the confounders were considered in this study by capturing them in the interview schedule during data collection and data analysis.

3.5.1.2. Reliability

The same interview schedule and method of collecting and procession the specimens and data was used on all the patients.

3.5.2. Data Collection Technique

Two Nurses were recruited as research assistants and oriented for two days on data collection using the interview schedule. However, the blood sample collection was done by other staff at UTH such as the clinic five Nurses, clinical chemistry Laboratory Technologists, Biomedical Scientists and the researcher after an informed written consent was obtained. This was done as part of the routine clinical management of DM out-patients. The data and blood samples were collected between September and November 2013 at UTH clinic five every week day from 07:00 hours to 11:00 hours.

A three step process of data collection was set up and included patient selection, interview and blood sample collection. The research assistants and other staff at clinic five were sensitised on the whole process of data collection so as to reduce the waiting time of the patients. This also ensured that the normal standard of care of the patients was not compromised in any way. This process was done every day and to complete the process, each patient was expected to pass through all the three steps.

3.5.2.1. Patient selection

On arrival of the patient at clinic five, the clerk sorted out all the DM patients visiting clinic five for follow-up visits and prepared a list. The list was scrutinized for those who met the criteria by the researcher and research assistants. The patient's anthropometric measurements were checked at the nurses' desk and those of interest included weight, height and blood pressure. Thereafter, the patients were reviewed by the DM clinic medical officer in the screening room for their usual routine follow-up visits.

3.5.2.1.1. Anthropometric Measurements

The weight and height of the patients were measured using a ZT-160 adult weighing mechanical scale model with a height rod (Wuxi Weigher Factory Company (Co), limited (Ltd), Zhejiang, China). The blood pressure was measured using the Citizen Digital Blood Pressure Monitor (Citizen Systems Japan Co., Ltd, Tokyo, Japan).

The BMI was calculated from the height and weight based on the formula developed by the Belgian astronomer, statistician, mathematician and sociologist, Lambert Adolphe Jacques Quételet in 1835 (Garabed, 2007). A scientific calculator FX-82ES (CASIO computer Co. Ltd, Tokyo, Japan) was used to obtain the actual BMI figure by dividing weight in kilograms (kg) with height squared in metres (m) which was also verified by the BMI chart (WHO, 2006). These measurements were later on entered onto the interview schedule.

3.5.2.2. Interview of Patient

After being reviewed by the medical officer for their follow-up visits, the patients were directed to the interview room. Self-introduction to the patients was done by the researcher and research assistants before the interview. In the interview room, patients were interviewed by the researcher or research assistants so as to obtain demographic, self-management data and to extract clinical data from the medical records. Each interview lasted for about 15 to 25 minutes. At the end of each interview, patients were given time to ask questions, which were answered accordingly. The patients were thanked at the end of the interview for their participation in the study.

3.5.2.2.1. Demographic and Self-Management Behaviour Data

The demographic and self-management behavior data from all the consenting patients was collected by interview. The data included age, sex, education, adherence to antidiabetic treatment, reasons for non-adherence to antidiabetic treatment, SBGM, means of SBGM and involvement in any exercise.

3.5.2.2.2. Clinical Data

The medical records of consenting patients were also reviewed to extract data on DM type, DM duration, antidiabetic treatment type, non-antidiabetic treatment type, co-morbidity, BMI, DM family history, systolic blood pressure and FPG. The data were later entered onto the interview schedule.

3.5.2.3. Blood Sample Collection and Processing

After the interview, the patients who still consented to blood collection had a blood sample collected from either the median cubital vein or cephalic vein on the left upper limb. Each patient was asked to sit comfortably on a chair.

Before drawing blood, a sterile spirited cotton wool swab was used to clean the actual site of blood collection. The total amount of blood that was collected from each patient was eight millilitres (mL); four mL for HbA_{1c} and four mL for FPG.

The venous blood sample collection was done using a sterile 21 G needle and a sterile 10 mL syringe and then transferred into potassium ethylenediaminetetraacetic acid (K2-EDTA) (lavender top tube) for HbA_{1c} and sodium fluoride/potassium oxalate (light grey top tube) for FPG blood collection tubes (Kiechle, 2005). The standard anticoagulant used for haematology is K2-EDTA because it preserves the cellular components of the blood. Sodium fluoride functions by stabilising the RBC membrane and inhibiting the enzyme systems involved in glycolysis, which prevents RBCs metabolising any glucose present in the sample. Thus, it is the only suitable sample for accurate glucose analysis. Fluoride is a potent inhibitor of many enzymes and the inhibition of glycolysis tends to cause fluid shifts. Fluoride is a weak anticoagulant on its own, so potassium oxalate; another powerful enzyme inhibitor is usually added to supplement its action (Kiechle, 2005).

After drawing blood, a sterile dry cotton wool swab was applied over the punctured site for about a minute to avoid bleeding. The sample was coded with participant's unique secret identity. The samples were held in the tube racks in a cooler box containing ice packs. The samples were later taken to the laboratory for initial processing. The HbA_{1c} samples were kept at room temperature for less than eight hours before storage. The samples were stored in the refrigerator at $5^{o}C$ for two weeks before running the test (Guder and Zawta, 2001). The FPG samples, were centrifuged for three minutes at 3000 revolutions per minute (RPM) using the CLIO IEC Centrifuge (Thermo Electron Industries, Chateau-Gontier-France). After centrifuging, the plasma was separated from the cells and transferred into a clean non-additive tube (red top tube).

If separation is delayed, the glucose value continues to decrease in whole blood after sample collection because of RBC glycolysis. Despite the use of coagulation inhibitors in plasma testing, some studies have shown that sodium fluoride takes time to work, so that at the end of the 1st hour at $25^{o}C$, glucose decrease in the plasma is similar regardless of whether sodium fluoride was used in the tube (Schwartz, Reichberg and Gambino, 2005).

It is estimated that, plasma glucose levels are reduced by 0.6 mmol/L per hour by consumption of glucose in the RBC's glycolytic pathway (Sacks et al., 2002). Thus, the plasma was separated from the cells within one hour of collection as processing time is an important factor (Nicholas, 2005). In addition, faster laboratory turnaround time is one reason that plasma has become the gold standard for glucose measurement than serum. The separated plasma was stored in the refrigerator at 4^{o}C for three days before running the test.

3.5.2.4. HbA_{1c} Measurement

In the laboratory, in vitro diagnostic method was used for the quantitative determination of HbA_{1c} percentage, a marker of DM, in human blood by colorimetry and turbidimetry on ABX Pentra 400 Automated Clinical Chemistry Analyser (HORIBA ABX SAS, 34184 Montpellier, France). In order to determine HbA_{1c} as a percentage of THb five HbA_{1c} reagents, six HbA_{1c} calibrators and three HbA_{1c} controls were used as follows:

ABX Pentra HbA_{1c} WB: Haemolysate Reagents (REF: A11A01702)

The ABX Pentra HbA_{1c} WB is a multi-reagent kit containing five reagents which include antibody reagent (R1) (1 x 23 mL) diluted with diluent I (R5) (1 x 25 mL), agglutinator reagent (R2) (1 x 23 mL), haemolysis reagent (R3) (110 mL) and THb reagent (R4) (2 x 21 mL). The HbA_{1c} WB haemolysis reagent is also sold separately under the reference A11A01730 (HORIBA ABX SAS, 34184 Montpellier, France).

Three volumes of R1 were mixed with two volumes of R5. The mixture was allowed to stabilise for approximately an hour in the refrigerated Analyser reagent compartment before the first use. However, R2, R3 and R4 are supplied as ready-to-use solutions. These reagents were carefully mixed by inverting five to 10 times, and poured into a reagent bottle. The reagents were allowed to stabilise in the refrigerated Analyser reagent compartment for half an hour before use. The reagents were later placed in the reagents racks of the Analyser before assay.

ABX Pentra HbA_{1c} WB Calibrators (REF: A11A01703)

The six calibrators (CAL) included CAL 1 (1 x 8 mL), CAL 2 (1 x 2 mL), CAL 3 (1 x 2 mL), CAL 4 (1 x 2 mL), CAL 5 (1 x 2 mL) and CAL 6 (1 x 2 mL) (HORIBA ABX SAS, 34184 Montpellier, France). The calibrators were used to calibrate the analyser results. Thus, six HbA_{1c} calibrator levels were considered. These calibration procedures are compatible with the NGSP/DCCT certification. The calibration was done on the 11^{th} day from the previous calibration. The THb was calibrated with the CAL 1 using a target THb value. The calibrators just as the reagents are not lot-dependent and are submitted to an internal quality control at HORIBA Medical. The CALs were placed on board the Analyser in quantities of 150 µl except for CAL 1 with 200 µl.

ABX Pentra HbA_{1c} WB Control (REF: A11A0174)

The Control kit has Normal control (2 x 0.25 mL) (lyophilisate), Pathological control (2 x 0.25 mL) (lyophilisate) and a buffer (1 x 2 mL) (HORIBA ABX SAS, 34184 Montpellier, France). The controls were diluted to 1:51 with R3, carefully mixed and allowed to stand for five minutes at room temperature (until a greenish colouring was obtained) before carrying out the test in the Analyser. The controls were used for internal quality control. Each control was assayed daily and after each calibration the values were recorded on the Levey Jennings chart.

The results were within the range of the defined confidence limits of the UTH clinical chemistry laboratory of HbA_{1c} 3.6 - 6% or 15.85 - 42.08 mmol/mol. The standard values were compatible with the NGSP reference values. The reconstituted latex solutions were stored in the refrigerator at temperatures between 2°C and 8°C after reconstitution for two months. Only the required quantity was used, and immediately placed the reagents in the refrigerator after use as recommended by Knowles, Haigh and Michaud (1986).

Blood Sample

The whole-blood sample was carefully manually mixed by inverting five to 10 times with the haemolysis reagent (R3) (0.05 mL of sample for 2.5 mL of R3) in a non-additive tube before placing the mixture on board the Analyser. A positive displacement pipette was used for collecting whole blood from the tube.

The RBCs were lysed and the Hb chain was hydrolysed by the action of a protease present in the reagent. The samples were analysed within 60 minutes of mixing.

HbA_{1c} is expressed as a percentage of the total haemoglobin (THb) content. In order to determine THb concentrations by spectrophotometry, the different forms of Hb were converted into a single form having a uniform absorbance spectrum. The HbA_{1c} and THb values in μmol obtained with the test were used to calculate the HbA_{1c}/THb ratio and were not used individually to establish the values (Wolf, Lang and Zander, 1984). HbA_{1c} and THb concentrations were measured, and the ratio was given as a percentage of HbA_{1c}. The HbA_{1c} in percentage form was converted to HbA_{1c} SI units of mmol/mol using the standard conversion formula as per the IFCC guidelines.

The THb reagent (R4) was used to determine THb. The method is based on the conversion of all forms of Hb into alkaline haematin in an alkaline solution of non-ionic detergent as described in the original procedure by Wolf, Lang and Zander (1984). The reaction is triggered off by the addition of a blood sample pre-treated with R4, resulting in a green colouration of the solution. The conversion of the different types of Hb into alkaline haematin with a defined absorbance spectrum allows the calculation of the THb concentration. Hb was measured using an end-point method at 550 nanometre (nm). The Hb levels are directly proportional to an increase in the optic density (OD) observed (Wolf, Lang and Zander, 1984).

The latex agglutination inhibition test was used to measure specific HbA_{1c}. An agglutinin (synthetic polymer containing multiple copies of the immunoreactive portion of HbA_{1c}) causes the agglutination of the latex particles covered with monoclonal mouse antibodies specific for HbA_{1c}. In the absence of HbA_{1c} in the sample, the agglutinin in the Agglutinator Reagent (R2) and the microparticles covered with Antibody Reagent (R1) agglutinate. The agglutination leads to an increase in the absorbance of the suspension. The presence of HbA_{1c} in the sample reduces the rate of agglutination, for HbA_{1c} enters into competition with the agglutinator reagent (R2) at the microparticles' antibody docking sites. The greater the amount of HbA_{1c} in the sample, the lower the agglutination rate. Thus, the increase in absorbance is inversely proportional to the concentration of HbA_{1c} in the sample. The reaction was measured by absorbance at 550 nm and the agglutination rate was used to calculate the concentration from a calibration curve.

The percentage of HbA_{1c} was then calculated using HbA_{1c} and THb values in µmol (Jeppsson et al., 2002).

3.5.2.5. FPG Measurement

The ABX Pentra Glucose phenol + aminophenazone (PAP) CP diagnostic reagent was used for quantitative in-vivo determination of glucose PAP in plasma by colorimetry using the ABX Pentra 400 Automated Clinical Chemistry Analyser (HORIBA ABX SAS, 34184 Montpellier, France).

ABX Pentra Glucose PAP CP Reagent (REF: A11A01668)

This is a ready-to- use reagent (1 x 90 mL) comprising phosphate buffer, pH 7.40 (13.8 mmol/L), Phenol (10 mmol/L), 4-aminoantipyrine (0.3 mmol/l), glucose oxidase (≥ 10,000 U/l) peroxidase (≥ 700 U/l) and sodium azide (< 0.1 per cent) (HORIBA ABX SAS, 34184 Montpellier, France). On removing the cap, the cassette was placed in the refrigerated Analyser reagent compartment.

ABX Pentra MultiCalibrator (REF: A11A01652)

The MultiCal (10 x 3 mL) (lyophilisate) was used for calibration (HORIBA ABX SAS, 34184 Montpellier, France). Measured amounts were placed on board the Analyser.

ABX Pentra N Control (REF: A11A01653)

The ABX Pentra N Control (10 x 3 mL) (lyophilisate) was used for internal quality control (HORIBA ABX SAS, 34184 Montpellier, France). Measured amounts were placed on board the Analyser.

ABX Pentra P Control (REF: A11A01654)

The ABX Pentra P Control (10 x 5 mL) (lyophilisate) was used for internal quality control (HORIBA ABX SAS, 34184 Montpellier, France). Measured amounts were placed on board the Analyser. Each control was assayed daily and after each calibration. The results were within the range of the defined confidence limits (3.5-5.5 mmol/L) by UTH clinical chemistry laboratory. The opened reagent cassette placed in the refrigerated analyser reagent compartment was used within 83 days (on-board stability).

The plasma sample volume for analysis on the analyser was 4 µl per test. The FPG was measured by the enzymatic determination of glucose using the Trinder method. This method determines the presence of glucose or glucose oxidase (GOD) using the Trinder reagent, and is a colour change test resulting from the Trinder reaction. The Trinder reagent, named after P. Trinder of Sunderland comprises 4-aminoantipyrine (4-AAP) and phenol (p-hydroxybenzene) (Yamagishi, Stanford, and van Ast, 2001). The Trinder reaction is the reaction between hydrogen peroxide (H_2O_2) and the phenol and AAP to form a quinone (quinoneimine), catalysed by the presence of a peroxidase (POD) (such as horseradish peroxidase) (Arvind, Rajiv and Sudhanshu, 2004).

The equations of the reactions are:

- Glucose + O2 Glucose $\xrightarrow{\text{oxidase}}$ Glucose acid + H_2O_2
- $2H_2O_2$ + Phenol + 4AAP (4-aminoantipyrine) $\xrightarrow{\text{Peroxidase}}$ Quinoneimine + $4H_2O$

The H_2O_2 is itself produced by an initial reaction where the glucose is oxidised in the presence of the GOD catalyst into H_2O_2 and gluconic acid or glucose acid. The formed H_2O_2, is detected by a chromogenic O_2 acceptor, phenol-aminophenazone in the presence of POD. The quinone is red-violet in colour, with the intensity of the colour formed being in proportion to the glucose concentration (Arvind, Rajiv and Sudhanshu, 2004). The increase in absorbance colour measured at 510 nm is proportional to the glucose concentration in the sample (Dosoretz and Ward, 2006). The analyser automatically computes the glucose concentration in mmol/L of each sample (Sacks et al., 2002).

3.5.3. Data Analysis

Following data collection, the pre-coded interview schedule was double checked for completeness, consistency, legibility and accuracy daily. Numerical codes were used on the interview schedule. The flaws on the interview schedule were corrected. The data collected was entered and stored into the data editor of IBM® SPSS® and statistically analysed using IBM SPSS Statistics for Windows Version 20.0 (IBM Corp. Armonk, NY, USA).

This computer software statistical package enabled the researcher to obtain a data set of HbA_{1c} and demographic, self-management behavior and clinical factors associated with glycaemic control status of the DM out-patients.

The glycaemic control status reflected by HbA_{1c} levels was later dichotomised into good and poor glycaemic control status. Univariate analysis of HbA_{1c}, FPG and the factors associated with glucose control status was carried out to describe the variables. Bivariate analysis of HbA_{1c} and each of the independent variables was carried out to ascertain association and "causality". Pearson's Chi-Squared (X^2) and Fisher's exact tests were used to determine whether there was an association between HbA_{1c} and categorical predictors and the Student's t-test was used for continuous predictors. The Paired Samples t-test was used for repeated measures of FPG to see whether there was a difference between the means. These tests primarily helped to identify the potential predictors of glycaemic control status. The assumptions of random sample, adequate sample size and cell count and approximate normality of data for these tests were met.

Multivariate Binary logistic regression was used to determine true predictors of having good and poor glycaemic control statuses. The predictors considered statistically significant were entered into the regression model to control for confounders. A *p*-value of < 0.05 was considered statistically significant. The odds ratio (OR) = 1, implied factors do not affect the odds of HbA_{1c}, OR > 1, factors associated (effect) with higher odds of HbA_{1c}, and OR < 1, implied factors associated (effect) with lower odds of HbA_{1c} and the CI of 95 per cent was set.

The assumptions of the binary logistic regression model of multicollinearity (SE > 2), sample size (> 10 cases per predictor) and Hosmer and Lemeshow test of model fitness for data (X^2 (8) = 4.440; $P = 0.815$), overall test of relationship (Nagelkerke R^2; $p = 0.420$), omnibus test of model coefficients ($X^2(26) = 67.620$; $P = 0.000$), and classification accuracy (Accuracy rate = 78.9%) were met.

3.6. Ethical and Cultural Considerations

The approval to carry out the study was sought from the University of Zambia Biomedical Research Ethics Committee (UNZABREC) and the Director, Directorate of Research and Postgraduate studies.

Permission to conduct the study was obtained from the Senior Medical Superintendent of UTH and the head of the Department of Internal Medicine at UTH. There was very negligible interference to the participants beyond the general standard of clinical care offered by UTH at clinic five.

Written informed consent was obtained from the participants before the study. The researcher/research assistants introduced themselves and explained to participant the purpose and nature of the study. The participant was assured of confidentiality and that no names or any form of identification was to appear on the information sheet and structured interview schedule. Moreover, each participant was assigned a unique confidential study number, which was used when collecting and reporting data.

CHAPTER FOUR

4.0. Results

A total of 198 patients were sampled from amongst the DM patients who visited UTH clinic five during the period of study. The results have been presented in frequency tables, figures and contingency tables according to the sequence and sections of the interview schedule.

4.1. Demographic Data

The demographic characteristics of the patients captured during recruitment are shown in Table 3.

Table 3: Demographic Characteristics of Patients

Variable	Frequency	Per cent
Age		
15-34 Years	25	12.6
35-54 Years	72	36.4
55 Years and above	101	51.0
Total	**198**	**100**
Sex		
Male	79	39.9
Female	119	60.1
Total	**198**	**100**
Education Level		
Never/Primary	74	37.4
Secondary	92	46.5
College/University	32	16.2
Total	**198**	**100**

(Source: Author's own analysis, 2014)

Table 3 shows that about half of the patients were aged 55 years and above. The age range was between 19 and 82 years. The majority of the patients were female and almost half of the patients had secondary education and the remaining had either primary or university education.

4.2. Self-Management Behaviours Data

The self-management behaviours of the patients which were considered in this study are shown in Table 4.

Table 4: Self-Management Behaviour Characteristics of Patients

Variable	Frequency	Per cent
Antidiabetic Treatment Adherence		
No	35	17.7
Yes	163	82.3
Total	**198**	**100**
Reasons for Non-Adherence to Antidiabetic Treatment		
Stock-out	32	91.4
Forget	3	8.6
Total	**35**	**100**
Self-Blood Glucose Monitoring		
No	26	13.1
Yes	172	86.9
Total	**198**	**100**
Self-Blood Glucose Monitoring Means		
Own glucometer	60	34.9
Public Health Facility	93	54.1
Private Health Facility	19	11.0
Total	**172**	**100**
Exercise		
No	111	56.1
Yes	87	43.9
Total	**198**	**100**

(Source: Author's own analysis, 2014)

Table 4 shows that most of the patients reported adherence to antidiabetic treatment while only a few reported non-adherences to the type of antidiabetic treatment they were on. The main reason for non-adherence to antidiabetic treatment was stock-out of the drugs. The majority of the patients reported monitoring of glucose at home and almost half of them monitored their glucose control at the public health facility. The remaining monitored their glucose control with own glucometer and a small proportion at the private health facilities. More than half of the patients reported involvement in some type of exercise.

4.3. Clinical Data

The clinical characteristics of the patients in this study are shown in Table 5.

Table 5: Clinical Characteristics of Patients

Variable	Frequency	Per cent
Diabetes Mellitus Type		
Type 1	14	7.1
Type 2	184	92.9
Total	**198**	**100**
Diabetes Mellitus Duration		
2-10 Years	158	79.8
11-20 Years	37	18.7
21 Years and above	3	1.5
Total	**198**	**100**
Antidiabetic Treatment Type[a]		
Oral Antidiabetic Drugs	112	56.6
Insulin	70	35.4
Oral Antidiabetics and Insulin	7	3.6
Diet Only	9	4.5
Total	**198**	**100**
Non-Antidiabetic Treatment Type[b]		
No	68	34.3
Yes	130	65.7
Total	**198**	**100**
Co-morbidity[c]		
No	70	35.4
Yes	128	64.6
Total	**198**	**100**
Body Mass Index (kg/m^2)		
Underweight (≤ 18.4)	6	3.2
Normal (18.5-24.9)	60	31.6
Overweight (25-29.9)	70	36.8
Obese (30 or greater)	54	28.4
Total	**190**	**100**
Diabetes Mellitus in Family		
No	81	40.9
Yes	117	59.1
Total	**198**	**100**
Systolic Blood Pressure (mm Hg; Mean, SD)	132.7	17.90
Diastolic Blood Pressure (mm Hg; Mean, SD)	84.7	11.18

(Source: Author's own analysis, 2014)

Note:

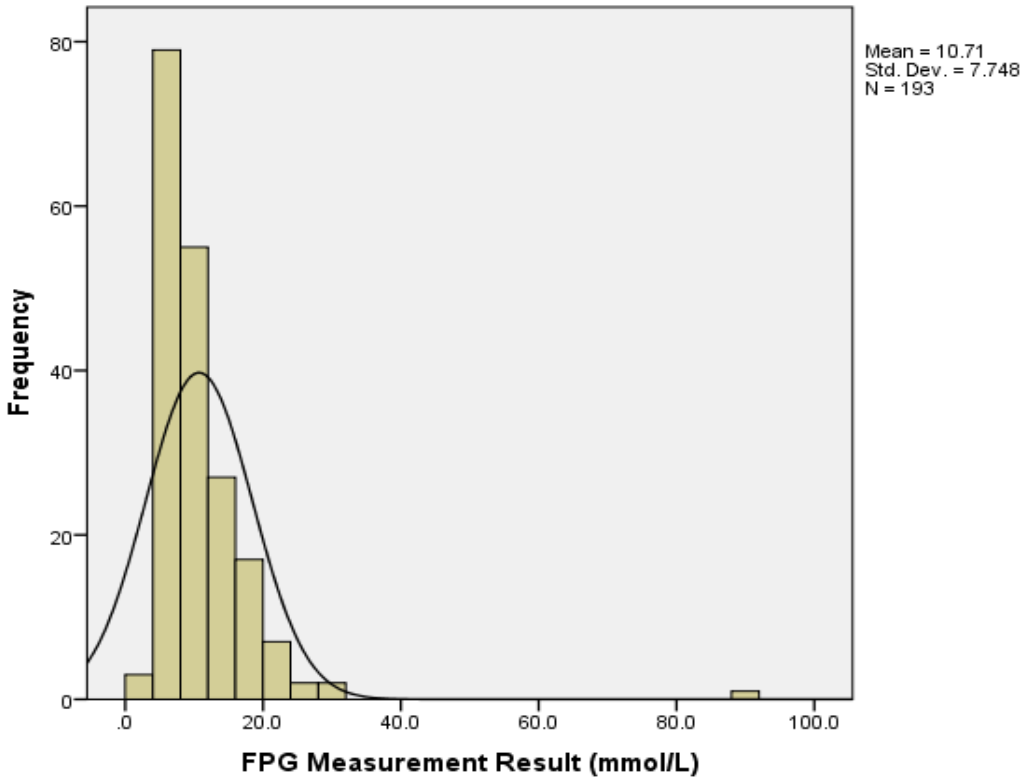
- [a]The drugs or diet the patients were on for the treatment of their DM.
- [b]Additional drugs to antidiabetic drugs which the patients where on such as antihypertensives, antiretrovirals, aspirin, septrin, antituberculous and a combination of these.
- [c]Other conditions or disorders or diabetic complications such as hypertension, cerebral vascular accident, cardiac diseases, HIV infection, Tuberculosis and a combination of these which the patients had besides DM.

Table 5 shows that nearly all of the patients had type 2 DM with most of them having been diabetic for a shorter duration. The range of the diabetic duration of the patients was between 2 and 36 years. The majority of the patients were on oral antidiabetic drugs and only a few were on both oral antidiabetic drugs and insulin. Most of the patients were on additional drugs besides the antidiabetic drugs and the majority had other disorders apart from DM.

Fewer than half 70 (36.8%) of the patients were overweight and only six (3.2%) were underweight according to the WHO classification of obesity. The minimum and maximum BMI was 16.6 and 63.2 kg/m^2. Most of the patients had a history of DM in the family. None of the patients had a seriously raised blood pressure. The minimum and maximum systolic and diastolic blood pressures were 90 and 210 mm Hg and 60 and 115 mm Hg respectively.

4.4. Laboratory Measurement Results

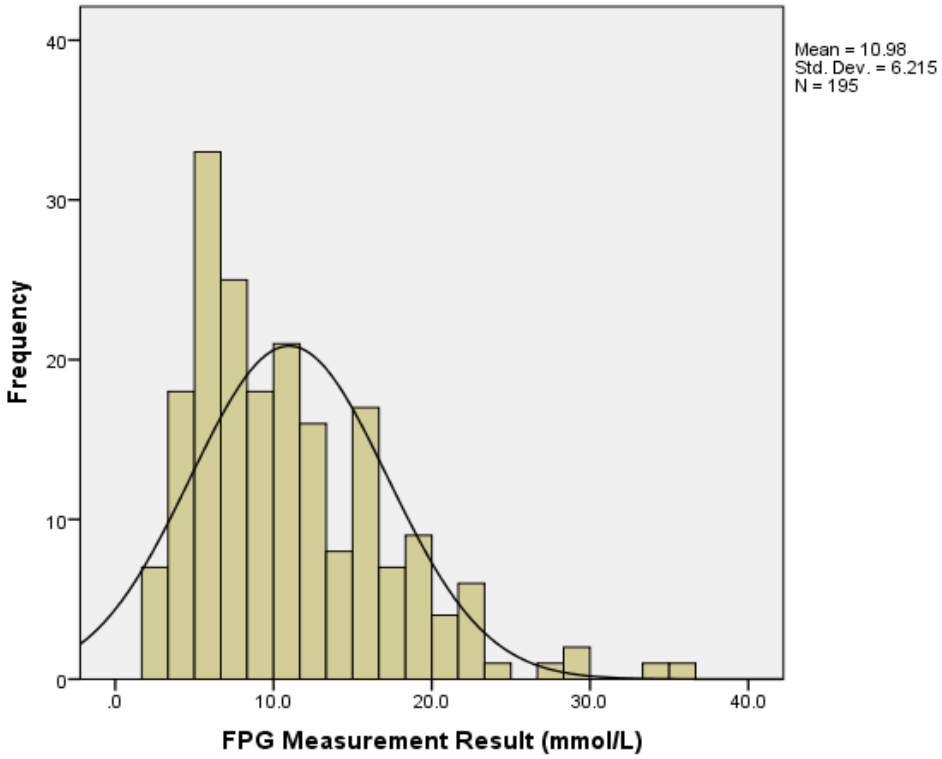
The measurement of FPG and HbA_{1c} were done in the laboratory and the results are shown in Figures 1, 2 and 3 and Table 6.



(Source: Author's own analysis, 2014)

Figure 1: FPG Measurement Result (Previous Three Months)

Figure 1 shows that the mean (SD) (10.71±7.748 mmol/L) FPG of the patients for the previous three months was higher than the normal (4.1-5.9 mmol/L).



(Source: Author's own analysis, 2014)

Figure 2: FPG Measurement Result (Current)

Figure 2 shows that the mean (SD) (10.98±6.215 mmol/L) current FPG of the patients was higher than the normal (4.1-5.9 mmol/L).

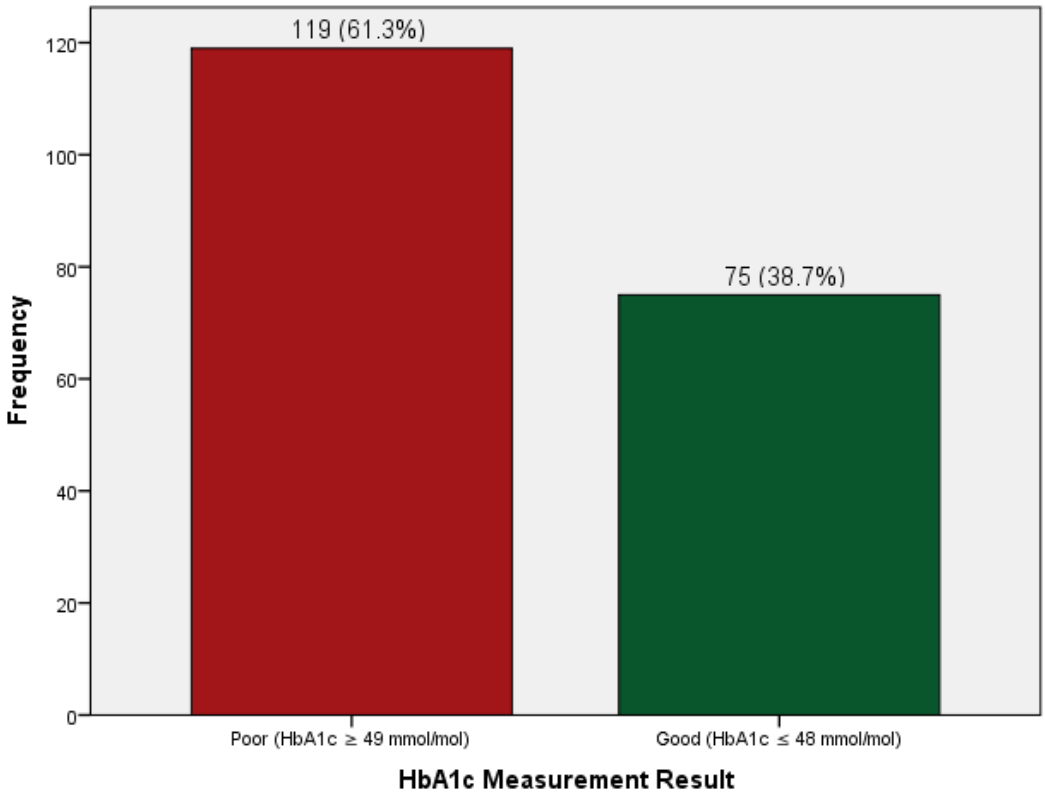
Table 6: Comparison of the Previous Three Months and Current FPG Measurement

Results

	N	Mean	Std. Deviation
Previous Six Months FPG Measurement Result (mmol/L)	191	10.753	7.7766
Current FPG Measurement Result (mmol/L)	191	11.093	6.2270

(Source: Author's own analysis, 2014) *$p < 0.05$

Table 6 shows that there was a very slight increase in the FPG from the previous (10.753 ± 7.7766 mmo/L) to the current (11.093 ± 6.2270 mmo/L). However, the difference was not statistically significant, $t(190) = 0.537$, $p = 0.592$.



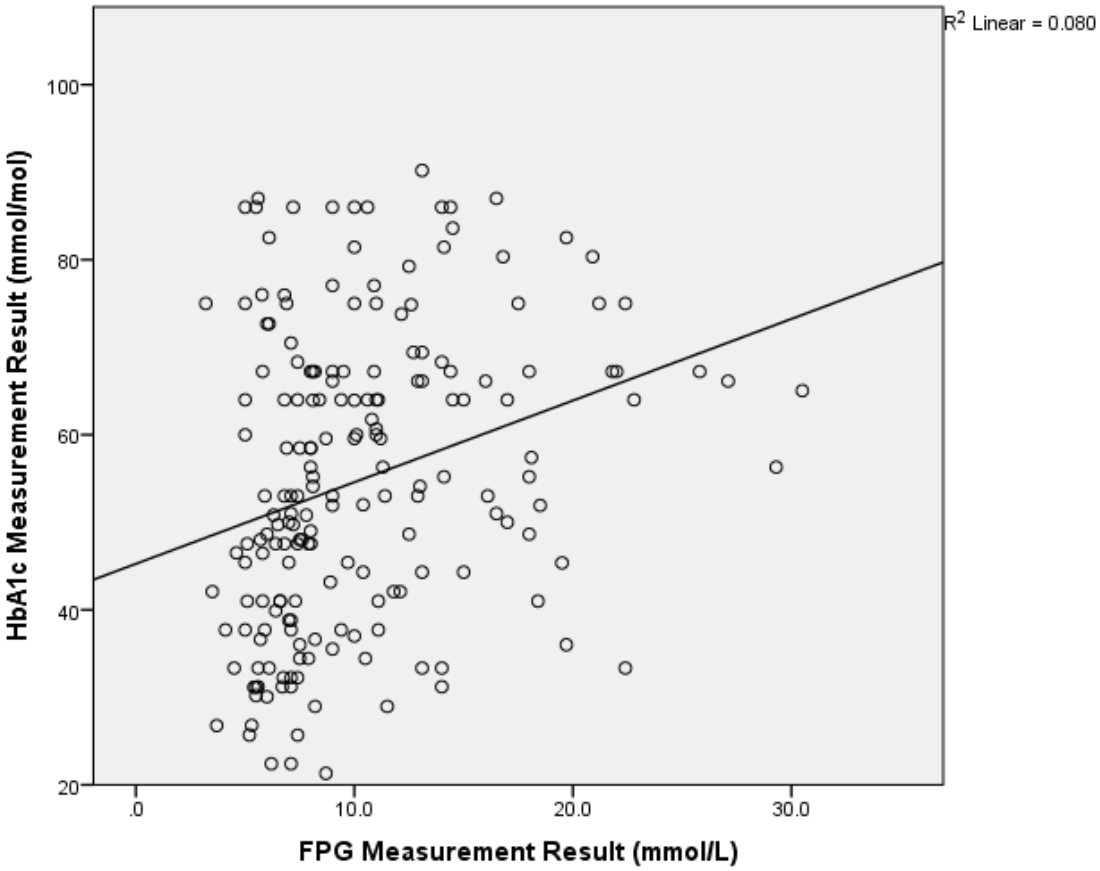
(Source: Author's own analysis, 2014)

Figure 3: HbA_{1c} Measurement Result (n=194)

Figure 3 shows that the majority (61.3%) of the patients had poor glycaemic control status while the remaining had good glycemic control among those whose data was complete. The minimum and maximum HbA_{1c} values were 21 and 90 mmol/mol.

4.5. Relationship between HbA_{1c} and FPG Levels

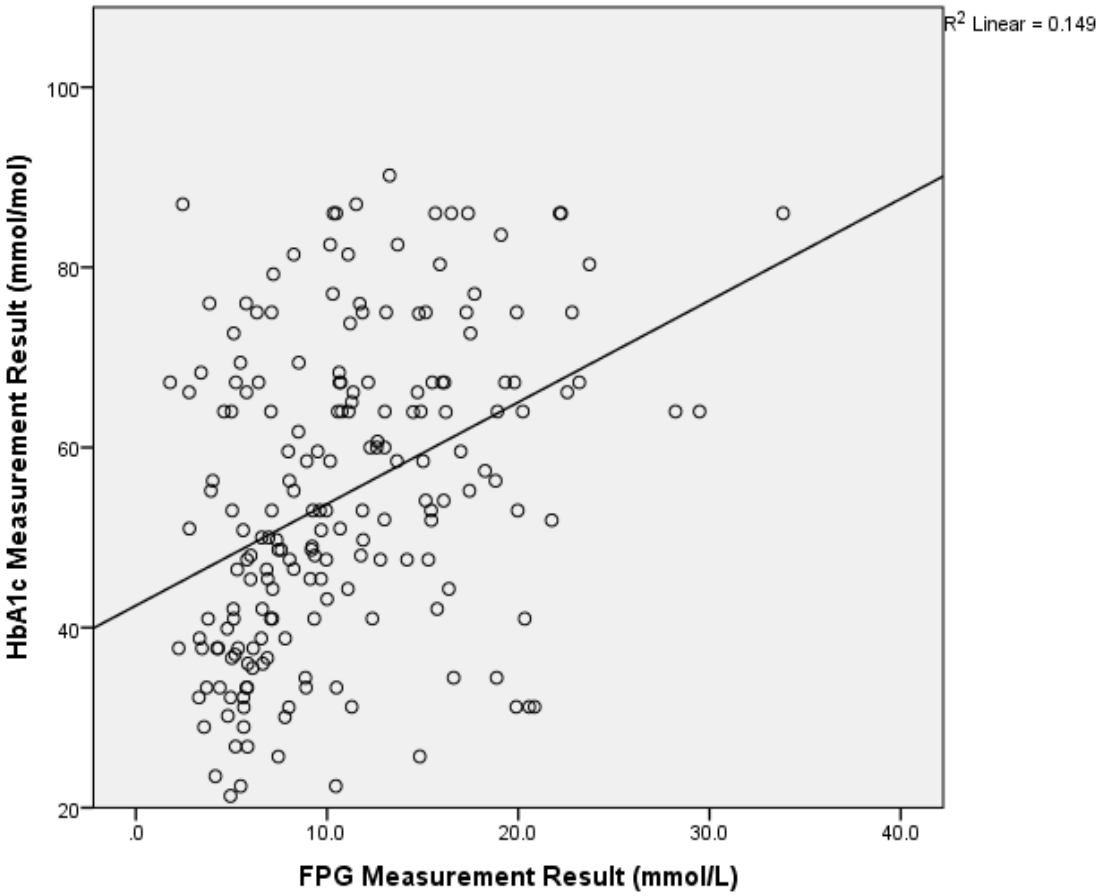
The relationship between HbA_{1c} and FPG levels was established and the results are shown in Figures 4 and 5.



(Source: Author's own analysis, 2014) *Correlation was significant at the 0.01 level

Figure 4: Scatter-Plot of Previous Three Months FPG and HbA_{1c} (n=191)

Figure 4 shows that there was a statistically significant but weak positive correlation between HbA_{1c} (54.77±17.12 mmol/mol) and the previous FPG (10.753 ± 7.7766 mmo/L) (Pearson's correlation coefficient, $r = 0.282$, $P = 0.001$).



(Source: Author's own analysis, 2014) *Correlation was significant at the 0.01 level.

Figure 5: Scatter-Plot of Current FPG and HbA_{1c} (n=191)

Figure 5 shows that there was a statistically significant moderate positive correlation between HbA_{1c} (54.77±17.12 mmol/mol) and the current FPG (11.093 ± 6.2270 mmo/L) (Pearson's correlation coefficient, $r = 0.385$, $P = 0.001$).

4.6. Glycaemic Control Status by Associated Factors

The relationship between glycaemic control status and the demographic, self-management behaviours and clinical factors of the patients was measured using Pearson's Chi-squared test, Fisher's exact test and Student's t-test and the results are presented in tables 7, 8 and 9.

Table 7: Glycaemic Control Status by the Demographic Factors of the Patients

	Glycaemic Control Status		
	Good (n = 75, $HbA_{1c} \leq$ 48 mmol/mol)	**Poor (n = 119, $HbA_{1c} \geq$ 49 mmol/mol)**	
Characteristic	**No (%)**	**No (%)**	**P-Value***
Age			
15-34 years	3 (12.5)	21 (87.5)	
35-54 years	29 (40.8)	42 (59.2)	**0.018**
55 years and above	43 (43.4)	56 (56.6)	
Sex			
Male	29 (38.2)	47 (61.8)	
Female	46 (39.0)	72 (61.0)	0 .908
Education level			
Never/Primary	27 (36.5)	47 (63.5)	
Secondary	33 (37.5)	55 (62.5)	0.053
College/University	15 (46.9)	17 (53.1)	

(Source: Author's own analysis, 2014)

Pearson's Chi-Squared Test, *Indicates significant *p*-value at $p < 0.05$.

Table 7 shows that poor glycaemic control status was more common across all ages than good glycaemic control status among the patients.

Table 8: Glycaemic Control Status by Self-Management Behaviours of the Patients

	Glycaemic Control Status		
	Good (n = 75, HbA_{1c} ≤ 48 mmol/mol)	Poor (n = 119, HbA_{1c} ≥ 49 mmol/mol)	
Characteristic	**No (%)**	**No (%)**	**P-Value***
Treatment adherence[a]			
No	61 (38.1)	99 (61.9)	
yes	14 (41.2)	20 (58.8)	**0.044**
Treatment non-adherence[b]			
Stock-out	15 (46.9)	17 (53.1)	
Forget	0 (00.0)	3 (100.0)	0.239
Not applicable	60 (37.7)	99 (62.3)	
Self-blood glucose monitoring[a]			
No	12 (46.2)	14 (53.8)	
Yes	63 (37.5)	105 (62.5)	0.399
Self-blood glucose monitoring means[a]			
Own glucometer	20 (35.1)	37 (64.9)	
Public health facility	35 (38.0)	57 (62.0)	0.793
Private health facility	8 (42.1)	11 (57.9)	
Not applicable	12 (46.2)	14 (53.8)	
Exercise[a]			
No	44 (41.1)	63 (58.9)	
Yes	31 (35.6)	56 (64.4)	0.435

(Source: Author's own analsysis, 2014)

[a]Pearson's Chi-Squared Test, [b]Fisher's Exact Test, *Indicates significant *p*-value at $p < 0.05$.

Table 8 shows that most of the patients who did not adhere to antidiabetic treatment had poor glycaemic control status compared to those who did adhere to antidiabetic treatment.

Table 9: Glycaemic Control Status by Clinical Factors of the Patients

	Glycaemic Control Status		
	Good (n = 75, $HbA_{1c} \leq 48$ mmol/mol)	Poor (n = 119, $HbA_{1c} \geq 49$ mmol/mol)	
Characteristic	**No (%)**	**No (%)**	**P-Value***
DM type[a]			
Type 1	2 (14.3)	12 (85.7)	
Type 2	73 (40.6)	107 (59.4)	0.052
DM duration[a]			
2-10 years	65 (41.9)	90 (58.1)	
11-20 years	9 (25.0)	27 (75.0)	0.160
21 years and above	1 (33.3)	2 (66.7)	
Antidiabetic Rx Type[b]			
Diet only	6 (66.7)	3 (33.3)	
Oral and Insulin	2 (28.6)	5 (71.4)	
Insulin	18 (26.9)	49 (73.1)	**0.030**
Oral antidiabetic drugs	49 (44.1)	62 (55.9)	
Non-antidiabetic Rx Type[a]			
No	19 (28.4)	48 (71.6)	
Yes	56 (44.1)	71 (55.9)	**0.032**
Co-morbidity[a]			
No	20 (29.0)	49 (71.0)	**0.040**
Yes	55 (44.0)	70 (56.0)	
BMI (kg/m2)[b]			
Underweight/Normal	18 (29.0)	44 (71.0)	
Overweight	31 (44.3)	39 (55.7)	0.178
Obese	22 (40.7)	32 (59.3)	
SBP (mm Hg; M, SD)[c]	130.64 (18.45)	136.40 (16.49)	**0.029**
DBP (mm Hg; M, SD)[c]	86.24 (9.91)	84.01 (11.80)	0.175
DM in Family[a]			
No	36 (44.4)	45 (55.6)	
Yes	39 (34.5)	74 (65.5)	0.161
Previous FPG (M, SD)[c]	8.15 (3.42)	11.64 (5.58)	**0.001**
Current FPG (M, SD)[c]	8.49 (4.63)	12.25 (6.09)	**0.001**

(Source: Author's own analysis, 2014)

Note:

- [a]Pearson's Chi-Squared Test
- [b]Fisher's Exact Test

- [c]Student's t-test
- *Indicates significant *p*-value at $p < 0.05$.
- Rx = Treatment
- BMI = Body mass index
- SBP = Systolic blood pressure
- DBP = Diastolic blood pressure
- SD = Standard deviation
- Underweight = $\leq$ 18 kg/m2
- Normal = 18.5 – 24.9 kg/m2
- Overweight = 25 – 29.9 kg/m2
- Obese = $\geq$ 30 kg/m2
- M = Mean

Table 9 shows a statistically significant association between glycaemic control status and type of DM, type of antidiabetic treatment, non-antidiabetic treatment type, co-morbidity, systolic blood pressure and FPG. Majority of the patients who had type 1 DM, on insulin treatment, did not adhere to treatment, without co-morbidity and with raised systolic blood pressure and FPG had poor glycaemic control status compared to those with type 2 DM, on other antidiabetic treatment, adhered to antidiabetic treatment, with co-morbidity and those with lower mean systolic blood pressure and FPG.

4.7. Binary Logistic Regression Determining the Demographic, Self-Management Behaviours and Clinical Factors Associated with Glycaemic Control Status

Binary logistic regression analysis was used to determine the true predictors of glycaemic control status as well as to control for confounding factors. The results of the univariate logistic regression revealed that except for the level of education and type of DM, the rest of the variables were associated with glycaemic control status of the patients as shown in table 10.

Table 10: Univariate Binary Logistic Regression Determining Factors Associated with Glycaemic Control Status

	Glycaemic Control Status			
	Good (n = 75, HbA_{1c} ≤ 48 mmol/mol)	Poor (n = 119, HbA_{1c} ≥ 49 mmol/mol)		
Predictor Variable	**No (%)**	**No (%)**	**OR (95% CI)**	**P-Value***
Age[a]				
15-34 years	3 (12.5)	21 (87.5)	0.19 (0.05-0.67)	**0.010**
35-54 years	29 (40.8)	42 (59.2)	0.90 (0.49-1.68)	0.736
55 and above	43 (43.4)	56 (56.6)	1.00 (Ref.)	
Adherence				
No	61 (38.1)	99 (61.9)	0.29 (0.20-1.11)	**0.049**
Yes	14 (41.2)	20 (58.8)	1.00 (Ref)	
Treatment Type				
OAD	49 (44.0)	62 (55.9)	0.40 (0.09-1.66)	0.205
Insulin	18 (26.9)	49 (73.1)	0.18 (0.04-0.81)	**0.026**
OAD and Insulin	2 (18.6)	5 (71.4)	0.20 (0.02-1.71)	0.142
Diet/None	6 (66.6)	3 (33.3)	1.00 (Ref.)	
Non-antidiabetic Rx				
No	19 (28.4)	48 (71.6)	0.50 (0.27-0.95)	**0.034**
Yes	56 (44.1)	71 (55.9)	1.00 (Ref.)	
Co-morbidity				
No	20 (29.0)	49 (71.0)	0.52 (0.28-0.97)	**0.041**
Yes	55 (44.0)	70 (56.0)	1.00 (Ref.)	
SBP (mm Hg; M, SD)	130.64 (18.44)	136.40 (10.49)	1.02 (1.00-1.02)	**0.032**
Previous FPG (M, SD)	8.15 (3.42)	11.64 (5.58)	0.77 (0.69-0.86)	**0.001**
Current FPG (M, SD)	8.49 (4.63)	12.25 (6.09)	0.87 (0.82-0.93)	**0.001**

(Source: Author's own analysis, 2014)

Note:

- *Indicates significant *p*-value at $p < 0.05$
- [a]Some of the sub-variables were merged to ensure a perfect analysis.
- Adherence = Adherence to antidiabetic treatment
- Treatment type = Antidiabetic treatment type
- OAD = Oral antidiabetic drugs
- Rx = Treatment
- M = Mean

- SD = Standard deviation
- FPG = Fasting plasma glucose in mmol/L

In table 10, the patients who were young, did not adhere to antidiabetic treatment, on insulin, not on other treatment apart from antidiabetics, without co-morbidity and with raised systolic blood pressure and FPG had poor glycaemic control status while the elderly, those who adhered to antidiabetic treatment, not on other treatments besides antidiabetic treatment, with co-morbidity and with lower systolic blood pressure and FPG had good glycaemic control status.

The multivariate logistic regression model was the final analysis to be performed. All the significant factors from the univariate logistic regression were considered for entry into the multivariate logistic regression model. The results of the multivariate binary logistic regression analysis to predict whether eight variable factors; that is age, adherence to antidiabetic treatment, type of DM, type of antidiabetic treatment, non-antidiabetic treatment, co-morbidity, systolic blood pressure and previous and current FPG levels were associated with glycaemic control status showed that, adherence to antidiabetic treatment, antidiabetic treatment type, systolic blood pressure and previous and current FPG levels were statistically significantly associated with glycaemic control status (Table 11).

Table 11: Multivariate Binary Logistic Regression Model of Factors Associated with Glycaemic Control Status

	Glycaemic Control Status			
	Good (n = 75, HbA1c ≤ 48 mmol/mol)	Poor (n = 119, HbA1c ≥ 49 mmol/mol)		
Predictor Variable	**No (%)**	**No (%)**	**OR (95% CI)**	**P-Value***
Age				
15-34 years	3 (12.5)	21 (87.5)	0.34 (0.07-1.71)	0.192
35-54 years	29 (40.8)	42 (59.2)	1.04 (0.50-2.12)	0.922
55 and above	43 (43.4)	56 (56.6)	1.00 (Ref.)	
Rx Adherence				
No	61 (38.1)	99 (61.9)	0.38 (0.13-1.07)	**0.043**
yes	14 (41.2)	20 (58.8)	1.00 (Ref.)	
Rx Type				
OAD	49 (44.0)	62 (55.9)	0.20 (0.2-1.85)	0.154
Insulin	18 (26.9)	49 (73.1)	0.13 (0.01-1.41)	**0.044**
OAD and Insulin	2 (18.6)	5 (71.4)	0.50 (0.03-8.86)	0.640
Diet only	6 (66.6)	3 (33.3)	1.00 (Ref.)	
Non-DM Rx				
No	19 (28.4)	48 (71.6)	0.95 (0.26-3.46)	0.940
Yes	56 (44.1)	71 (55.9)	1.00 (Ref.)	
Co-morbidity				
No	20 (29.0)	49 (71.0)	0.74 (0.21-2.63)	0.638
Yes	55 (44.0)	70 (56.0)	1.00 (Ref.)	
SBP (M, SD)	130.64 (18.44)	136.40 (10.49)	1.04 (1.00-1.08	**0.038**
Previous FPG				**0.001**
(mmol/L; M, SD)	8.15 (3.42)	11.64 (5.58)	0.81 (0.72-0.90)	
Current FPG				**0.001**
(mmol/L; M, SD)	8.49 (4.63)	12.25 (6.09)	0.85 (0.78-0.93)	

(Source: Author's own analysis, 2014)

Note:

- *Indicates significant *p*-value at $p < 0.05$.
- Rx = Treatment
- Rx adherence = adherence to antidiabetic treatment
- Rx type = Type of antidiabetic treatment
- OAD = Oral antidiabetic drugs

- Non-DM Rx = Non-antidiabetic treatment
- SBP = Systolic blood pressure in mm Hg.
- M = Mean
- SD = Standard deviation

Table 11 shows that the patients who did not adhere to antidiabetic treatment and those on insulin were 62% and 87% less likely to achieve good glycaemic control status than those who adhered to antidiabetic treatment and those on other antidiabetic treatments. An increase in systolic blood pressure and previous and current FPG levels resulted in 5%, 16% and 15% reduction in odds of achieving good glycaemic control status among the patients.

CHAPTER FIVE

5.0. Discussion

The aim of DM management is to keep glycaemic levels as close to normal as safely as possible, while avoiding hyperglycaemia or hypoglycaemia (Dilshad, Saeed, and Farooq, 2009). The poor glycaemic control status among DM patients coupled with increase in the prevalence of DM in Zambia is a public health concern. Resources are being provided for the management of DM patients at both personal and government levels but achieving good glycaemic control status is proving to be a considerable challenge in most cases. The current study examined the glycaemic control status and the associated demographic, self-management and clinical factors in DM out-patients at UTH in the Lusaka province of Zambia.

5.1. Demographic Characteristics of the Patients

Most of the patients were aged 55 years and above and more than half were females. The results are similar to Moreira Jr. et al. (2010). Age is often times synonymous with DM as the chances of developing DM especially type 2 increases with age. This is because there is an alteration in physiological activities in the elderly including the loss of first-phase insulin release (Meneilly, 1999). Soltesz et al. (2007) reported male predominance in type 1 DM prevalence. But generally, the worldwide incidence of type 1 DM between genders does not differ (Diamond Project Group, 2006). In addition, there is no difference in gender distribution of DM (Gale, 2001) because both genders seem to be affected equally by autoimmunity and obesity.

In this study, almost half of the patients had secondary education and less than a quarter had college/university education. This is similar to findings by Ghazanfari et al. (2010a) were most of the participants had secondary education. However, Moreira Jr. et al. (2010) found that most of the patients were lowly educated. Although, literature did not show how education contributes to the prevalence of DM, there is reason to suggest that education can influence the incidence of DM. This is because non-communicable diseases such as DM are influenced by lifestyle and health education can play a major role in mitigating DM.

5.2. HbA_{1c} and FPG Measurement Results

The results showed that out of the 198 patients, most of them had poor glycaemic control status (≤ 48 mmol/mol) while less than half had good glycaemic control status (≥ 49 mmol/mol). The mean (SD) for the previous three months and current FPG levels where both higher than the normal. This implies that the glycaemic control status was poor in most patients based on both HbA_{1c} and FPG. There was also a slight increase in FPG from the previous three months to the current one.

The poor glycaemic control status is consistent with other studies in developed and developing countries. Mahmood and Aamir (2005) in Pakistan (51.4 %), Adebisi et al. (2009) in Nigeria (64%), Arthur et al. (2006) in Ghana (65%), Liberopoulos et al., (2010) 38.7% and Erasmus et l. (1999) in South Africa (79.9%). Most of the studies had results similar to this study except for Erasmus (1999) which had the highest percentage of poor glycaemic control status based on both HbA_{1c} and FPG. The poor glycaemic control status could be attributed to inadequate knowledge on treatment protocols, inactivity and poor diets among others. On the other hand, good glycaemic control status was seen in Japan and Germany (Arai et al. 2009; Reisig et al. 2007). This might be because of the higher literacy levels with consequent better knowledge levels about DM.

5.3. Relationship between HbA1c and FPG levels

The current study revealed a weak to moderate positive correlation between HbA_{1c} and the previous and current FPG. However, Silverman et al. (2008) reported a moderate to strong positive correlation between HbA_{1c} levels and FPG. Bozkaya, Ozgu and Karaca (2010) in Turkey and Rohlfing et al., (2002) in the UK revealed a strong positive correlation between FPG levels and HbA_{1c} levels. Thus, the level of HbA_{1c} is proportional to the level of glucose in the blood and normal levels of glucose produce a normal amount of HbA_{1c}.

On the other hand, as the average amount of plasma glucose increases, the fraction of HbA_{1c} increases in a predictable way and this serves as a marker for average blood glucose levels over the previous 8 to 12 weeks prior to the measurement (Roszyk et al., 2007). It is important to note that FPG test ascertains the glucose levels for the past few days but since blood glucose levels fluctuate throughout the day, glucose records are imperfect indicators of changes in the body due to hyperglycaemia.

Nonetheless, the long-term assessment of blood glucose with HbA_{1c} is advantageous not only because it eliminates the large fluctuations that occur daily in blood glucose concentrations, but it also provides an accurate result from blood drawn at any time of day without reference to prandial state (Burtis, Ashood and Bruns, 2006).

In addition, this study showed that the patients were 16% (previous three months FPG) and 15% (current FPG) less likely to achieve good glycaemic control status. The results are better than the findings by Lipska et al. (2013) in the US who reported that, the older adults with both raised FPG levels and poor glycaemic control status had a substantially increased odds of developing diabetes over 7 years. Thus combined screening with FPG and HbA_{1c} may identify older adults at very high risk for diabetes when FPG and HbA_{1c} are considered together. The addition of elevated HbA_{1c} to the model with raised FPG resulted in improved discrimination and calibration.

Kumaravel et al. (2012) in the UK also revealed that, good glycaemic control together with other factors increased the odds of impaired FPG 6.5-fold compared to the pre-test odds. This has implications for current and future diabetes prevention programmes, for vascular risk management, and for clinical advice given to people with 'pre-diabetes' based on FPG data. Inoue, Matsumoto, Kobayashi (2007) in Japan, reported a 0.5% increase in HbA_{1c} for every 0.56 mmol/L increase in FPG. Thus, the combined use of FPG and HbA_{1c} levels predicts the progression to diabetes in individuals with no apparent risk. In particular, the combination is recommended for individuals with a FPG > or =5.55 mmol/L.

Studies have shown that HbA_{1c} is an index of average glucose (AG) over the preceding weeks-to-months. The level of HbA_{1c} at any point in time is contributed to by all circulating erythrocytes, from the oldest (120 days old) to the youngest (NGSP, 2010). However, HbA_{1c} is a "weighted" average of blood glucose levels during the preceding 120 days, meaning that glucose levels in the preceding 30 days contribute substantially more to the level of HbA_{1c} than do glucose levels 90-120 days earlier. Similarly, other researchers state that the major proportion of HbA_{1c} value is weighted toward the most recent two to four weeks.

This is supported by data from actual practice showing that HbA_{1c} level improved significantly already after 20 days since glucose-lowering treatment intensification (Sidorenkov et al. 2011). This explains why the level of HbA_{1c} can increase or decrease relatively quickly with large changes in glucose; however, it does not take 120 days to detect a clinically meaningful change in HbA_{1c} following a clinically significant change in AG (NGSP, 2010).

Thus, there is a very predictable relationship between HbA_{1c} and average glucose (AG). The relationship between HbA_{1c} and FPG was defined and understanding this relationship can help patients with diabetes and their health-care providers set day-to-day targets for AG based on HbA_{1c} goals. Also, FPG should be used with caution as a surrogate measure of AG and it is important to remember that HbA_{1c} is a weighted average of glucose levels during the preceding four months.

Unless the patient's glucose levels are very stable month after month, quarterly and half yearly measurement is needed to ensure that a patient's glycaemic control remains within the target range. Reporting the estimated AG level together with the HbA_{1c} level is believed to assist patients and medical doctors determine the effectiveness of glycaemic control measures. This association between the FPG and HbA_{1c} levels depends on the extent of glycaemic control.

5.4. Self-management Behaviours and Clinical Factors Associated with Glycaemic Control Status

The current study was able to demonstrate an association between adherence to antidiabetic treatment, type of antidiabetic treatment, systolic blood pressure, previous and current FPG levels and glycaemic control status. However, there was no association between achieving glycaemic control status and age, sex, level of education, type of DM, reason for non-adherence to antidiabetic treatment, duration of DM, non-antidiabetic treatment, SBGM, means of SBGM, co-morbidity, BMI, exercise, diastolic blood pressure, and family history of DM.

This is in agreement with a study by Ghazanifari et al. (2010a) where sex, BMI, co-morbidity, exercise age, and SBGM, duration of DM and diastolic blood pressure were not associated with glycaemic control status of the patients.

However, other previous studies have found an association between age, duration of DM (Ahmad, Islahudin and Paraidathathu, 2013), SBGM and education (Bi et al. 2010) and glycaemic control status. Although, in this study, female gender, SBGM, exercise and family history of DM revealed somewhat higher HbA_{1c} values, the results were not statistically significant.

There was a statistically significant association between adherence to antidiabetic treatment and glycaemic control status of the patients in this study. The effectiveness of drug treatment depends primarily on the efficacy of the prescribed treatment regimen and adherence of the patient to the antidiabetic treatment (Knobel et al. 1998). Studies have shown that adherence to antidiabetic treatment among DM patients is poor. It is also not surprising that diabetic patients who fail to comply with the prescribed antidiabetic treatment regimen show very poor outcomes (Leichter, 2005).

This study showed that the patients who did not adhere to antidiabetic treatment had 62% decreases in the odds of achieving good glycaemic control status compared to those who adhered to antidiabetic treatment. Among the patients who did not adhere to antidiabetic treatment, most of them had poor glycaemic control while less than half had good glycaemic control status. However, more than half of those who adhered to treatment had poor glycaemic control while less than half had good glycaemic control status. This also accords with a study by Ahmad, Ramli and Paraidathathu (2013) in Malaysia and Curkendal et al. (2013) in the USA where poor glycaemic control status was associated with non-adherence to antidiabetic treatment. To the contrary, Tiv et al. (2012) in France reported good glycaemic control status associated with adherence to antidiabetic treatment.

In addition, studies have shown that, an increase in adherence by 10% can decrease the HbA_{1c} value by 0.16%. This is also supported by other previous studies in DM, which showed that an increase in patient education and adherence has been associated with good glycaemic control. In Hong Kong, a pharmacist-managed clinic for diabetic patients improved adherence and glycaemic control without any change in medication or dosage (Lee and leung, 2003). It has been suggested that greater effort should be placed in counselling and improving adherence rather than changing medication or altering the dose (Bezie et al. 2006).

In Malaysia, health personnel were specifically dedicated to the care of diabetic patients and results showed better glycaemic control (Wong and Rahimah, 2004). Ahmad, Ramli and Paraidathathu, (2013) reported that, improvement of adherence among patients results in better glycaemic control, and that achievement of glycaemic control was higher among adherent patients than among non-adherent patients. However, tackling non-adherence is not a simple matter, as it is multifactorial and might include cost, health belief, dosing frequency, personality disorders and patient-provider relationship (Leichter, 2005).

The achievement of optimal glycaemic control through strict adherence to antidiabetic treatment among other factors minimises serious long term complications of DM (Kalyango, Owino and Nambuya, 2008). This is because the antidiabetic treatment taken by DM patients assists in the metabolism of more especially carbohydrates with consequent glycaemic control. If adherence could be resolved, it is possible that the outcome of treatment would be much more satisfactory among DM patients.

The type of antidiabetic treatment the patient was on predicted glycaemic control status. The present study revealed that the odds of achieving good glycaemic control status in DM patients were 87% lower among the patients who were on insulin compared to those who were on oral or combined (insulin and oral) or diet only. These results agree with Ahmad, Islahudin and Paraidathathu (2013) where patients receiving insulin treatment had poor glycaemic control status compared to those receiving monotherapy or a combination of oral antidiabetic drugs. Interestingly, DeFronzo (1999) and Chuang et al. (2002) reported good glycaemic control status among patients who were on insulin treatment.

The poor glycaemic control status among insulin users could be because the patients have a more severe form of the disease or in the late stages of the disease and therefore more difficult to control. The oral drugs are widely used as first-line antidiabetic treatment when the DM is just diagnosed. Furthermore, the procedure of insulin injection administration and the resources required for the same probably affect adherence.

The good glycaemic control status associated with insulin in other studies could be because insulin is much more effective than other antidiabetic treatments.

The administered insulin in DM patients directly helps in the metabolism of more especially glucose in the body.

The SBP of DM patients was correlated with the outcome of glycaemic control status in this study. Achievement of glycaemic control among patients with higher SBP was lower than among those with lower SBP. The current study showed that, each 1-mm Hg increase in SBP was related to a 5% reduction in the odds of achieving good glycaemic control status. Similar studies showed poor blood pressure control and glycaemic control status (Genuth et al. 1998; Higgins, Khan and Pearce, 2007). However, significant association between SBP and achievement of good glycaemic control status was observed in some studies reviewed.

Ghazanfari et al. (2010b) in Iran reported better SBP for those who achieved good glycaemic control status. According to Gunarathne et al. (2009) in the ACCORD blood pressure trial, a systolic blood pressure target of <120 mm Hg cannot be recommended for the majority of patients with type 2 diabetes but instead recommend a target blood pressure level of 130/80 mm Hg. This is similar to the guidelines for targeted systolic blood pressure levels of <130 mm Hg and diastolic blood pressure levels of <85 mm Hg recommended by ADA (1995). Based on current evidence, this is probably an appropriate blood pressure target for most DM patients.

Hypertension in people with diabetes is common, affecting 30% of people with younger onset DM and 75% with older onset DM. The efficacy of blood pressure control for retinopathy in people with hypertension and DM may be a controversial point because of the known serious systemic sequelae (for example, higher risk of cardiovascular disease, nephropathy, and amputation) of uncontrolled hypertension. However, whether lowering of blood pressures already in the normal range is beneficial, is still unknown (ADA, 1995). Thus, more attention should be addressed to primary preventative factors such as blood pressure in the management of DM patients.

CHAPTER SIX

6.0. Conclusion and Recommendation

6.1. Conclusion

The current study determined the glycaemic control status and associated factors among the DM out-patients at UTH in the Lusaka province of Zambia. Glycosylated Hb is a better predictor of glycaemic control status than most other methods of establishing the glycaemic control status of DM patients. The study revealed that the glycaemic control status was poor in most DM patients.

The FPG, DM treatment, adherence and SBP were found to impact the achievement of good glycaemic control status of the patients in this study. Because DM like most other chronic diseases is progressive, complications increase and drug therapy becomes much more complex with time. The current results suggest that if these factors are not adequately addressed, the glycaemic control status of the patients is likely to be poor. Thus, it is important for the health-care providers to pay special attention to particular groups, such as those on insulin, non-adherent and those with raised BP and FPG, to ensure good glycaemic control among diabetic patients.

Among the considerations are, the examination of all aspects of the patient and accordingly individualising the choice of glycaemic control goals, lifestyle modifications and the medications required to achieve the prescribed goals. Harmonising the potential for lowering HbA_{1c} should be carried out by taking into account the above patient characteristics to ensure long-term glycaemic control. The study, provided baseline data on glycaemic control status of the DM patients at UTH and the possible factors contributing to poor glycaemic control among the DM patients.

6.2. Recommendations

As a measure to improve the management of DM out-patients especially on glycaemic control, the researcher recommends the full scale utilisation and making available in health facilities of the HbA_{1c} test alongside other tests in monitoring glycaemic levels of the DM patients.

In addition, the staff managing DM patients should emphasise and include information Education and Communication (IEC) sessions during the follow-up visit by DM patients. The IEC should include; frequent monitoring of blood glucose levels and blood pressure and adherence to prescribed antidiabetic treatment.

6.3. Future Research

Future research should focus on evaluating the role of the diabetic patient in the management of their diabetes mellitus. There is need to carry out a large scale study and compare glycaemic control status between type 1 and type 2 diabetes mellitus patients and also ascertain the effectiveness of specific treatment regimens in diabetic patient management.

6.4. Limitations of the Study

The study had some limitations. One of the limitations was that confounding factors, such as diet, antidiabetic drugs, comorbidity, non-antidiabetic drugs and SBGM sub-variable quantification were not carried out. It would have been extremely difficult to obtain accurate data. The other limitation was that the time between the previous estimation of FPG and the second FPG was not the same for all patients. Some patients had their previous FPG reading taken 4-5 months prior to the study and others did not have the result.

In addition, there was failure to collect blood from some patients and some of the collected blood clotted while running samples. Since the study was cross-sectional, it is difficult to establish a causal relation between HbA_{1c} and the factors. The study was carried out on a limited study population. Only the patients who visited the UTH diabetic clinic during the three months of data collection where sampled. There was incomplete data on some medical records of the DM patients at UTH; as a result the researcher was not able to follow the morbidity patterns. Another issue that stands out was the cost containment especially of laboratory materials and supplies.

6.5. Dissemination and utilization of findings

The results of the study were presented to the Department of Physiological Sciences, School of Medicine, University of Zambia (UNZA). Then, the results were later presented at the postgraduate seminar week on 9th July 2014 held at UNZA main campus. The results will also be presented to various stake holders involved in the management of DM at various fora such as, workshops and conferences.

The UTH which was the study site will be given a copy of the study results report so that the hospital would use them to render evidence based care to the DM patients. The results will be published in recognised journals such as; the Zambian Medical Journal, Pan African Medical Journal and Asian Academic Research Journal of Multidisciplinary. In addition, five copies of the bound research report will be printed and submitted to the following;

1. Department of Physiological Sciences
2. UNZA Medical Library and Main Library
3. Ministry of Health
4. Ministry of Community Development Mother and Child Health
5. Researcher

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APPENDIX A

PARTICIPANT INFORMATION SHEET

1. Self-Introduction

Introduction of researcher / research assistant to the participant with regard to the name, what they do and their involvement in the research.

2. Title of Research being done

"Examining blood glucose control status and associated factors in diabetes mellitus out-patients at the University Teaching Hospital, Lusaka, Zambia"

3. Purpose of the Research

To determine the blood glucose control status and associated factors using glycosylated haemoglobin (HbA_{1c}) among diabetes mellitus (DM) out-patients.

4. Procedure

A blood sample of 8 mL will be collected from a vein using a needle and syringe and then transferred into an EDTA (lavender top tube) and fluoride/oxalate (light grey top tube) specimen containers. The specimen will then be coded and later subjected to HBA_{1c} and FPG tests respectively in the laboratory.

5. Voluntariness

Your participation in this research is entirely voluntary and you do not have to participate if you do not wish to do so. Be assured that your refusal to take part will not in any way result in penalty or loss of services to which you are otherwise entitled. If you decide to take part, you are still free to withdraw at any time without giving a reason for your withdrawal. You also have the right to end the interview at any time, and to choose not to answer particular questions that are asked in the study.

6. Guarantee of Confidentiality

Be assured that the information collected from you in this research will be kept strictly confidential and all the data collection tools used will be destroyed thereafter.

7. Risk/Benefits/Discomforts

The procedure of drawing a blood sample for blood glucose tests is considered a safe and relatively painless procedure. However, as with many medical tests, some problems can occur with having blood drawn such as fainting or feeling light headedness, hematoma (blood accumulating under the skin causing a lump or bruise) or pain associated with multiple punctures to locate a vein.

The benefits are that, after the investigations are done, your blood glucose control status will be determined and this will help the health care professionals to better manage your diabetes mellitus.

8. Compensation/Reimbursement

The participation in this research has no provision for compensation/reimbursement.

9. Consequences of Injury

In the event that the participant is injured during the procedure, the researcher will take full responsibility of the consequences to correct the situation.

If you have any questions about the study please contact the principal investigator or the chairperson for the UNZA Biomedical Research Ethics Committee at the following addresses and contact numbers;

10. Contact Details of Principal Investigator

Emmanuel Mwila Musenge
The University of Zambia
School of Medicine
Department of Physiological Sciences
P.O. Box 50110
Ridgeway Campus
Cell No: +260977885979
Email: emmasenge@yahoo.com
LUSAKA
ZAMBIA

11. Contact Details of Ethics Committee

The Chairperson

The University of Zambia

School of Medicine

Biomedical Research Ethics Committee

P. O. Box 50110

Ridgway Campus

Telephone: 260-1-256067

Telegrams: UNZA, LUSAKA

Telex: UNZALU ZA 44370

Fax: + 260-1-250753

E-mail: unzarec@zamtel.zm

LUSAKA

ZAMBIA

If you choose to participate in this research study, please sign the informed consent form below.

APPENDIX B

INFORMED VOLUNTARY CONSENT FORM

DECLARATION

I have read (or have had explained to) the information about this study as contained in the participant information sheet. I have had the opportunity to ask questions about the research and any questions I have asked have been answered to my satisfaction.

I now consent voluntarily to participate in this study and understand that I have the right to end the interview at any time if I so wish, and to choose not to answer particular questions that are asked in the study.

My signature below signifies that I am willing to participate in this study:

Name of participant (Print): ……..….....…..…

Signature of participant: ……Consent Date: ……..…..…...

Participant's right thumb print if unable to write: ……...……..……..……

Name of researcher conducting voluntary consent (Print): …..……..……..…...…

Signature of researcher: …….…….…….…… Date: …….....……

Name of witness (Print): ……..….

Signature of witness: ……..…….. Date: ……...…...…..……..

APPENDIX C

TRANSLATED INFORMATION SHEET

ICHIPEPA CHA IFYO ABALEIBIMBA MULI UKU KUFWAILISHA PA BULWELE BWA SUGAR BAFWILE UKWISHIBA

1. **Ukuilondolola**

   Kafwailisha afwile aeba abaleibimbamo muli uku kufwailisha ishina lyakwe, efyo acita elo nomulimo wakwe muli uku kufwailisha.

2. **Umutwe Wa Uku Kufwailisha**

   "Ukulengula sugar mu mulopa wabalwele ba sugar pa chipatala chikalamba icha UTH muno Lusaka mu chalo cha Zambia"

3. **Icho Tulefwailisha**

   Ukulengula sugar mumulopa wabalwele ba sugar elo nefilenga ukuti uyu sugar ebabwino

4. **Ifyo Tulechita**

   Utumulopa utulingilefye twalabulwa ukufuma mutumipaipi twamulopa elo nokutwala utu tumulopa ku kupima nabamashini pakuti tumone ubwingi bwa sugar mumulopa.

5. **Ukuipelesha**

   Ukuibimba muli uku kufwailisha kwa sugar mu mulopa kuipelesha elo ngatamulefwaya kuti mwakana. Kaili ngamwakana ukuibimbamo mulu uku kufwailisha temulandu iyoo nangula teti ababomfi bamuchipatala bamuchite wanyawanya mu kundapo. Nga chakweba ati mwachinja amano, namukwata insambu ishyakukana konkanyapo muli uku ukuibimbamo nangula namutampako kale ukwabula uku londolola ifilifyonse. Elo namukwata insambu shaku kana yasuka amepusho eyo tamulefwaya ukwasuka muli uku kufwailisha.

6. **Ichilayo Cha Nkâma**

   Ndemweba ukuti fyonse ifyo twalalandishyanya pamo namepusho mulu uku kufwailisha nalafisunga munkâma elo nama pepala yonse eyo twala lemba po tuka yocha nga twapwisha ukuyabonfya.

7. **Ubusuma No Ububi Bwa Uku Kufwailisha**

   Ukufumya umulopa mu tumipaipi twamulopa takwakwata amafya elo tachikalipa sana. Ubusuma bwa uku kufwailisha bwakeba ati, ukulengula nga kwapwa, twalaishiba imifulile ya sugar mumulopa wenu. Ichi chikankala sana mukwafwilishako bashingânga uku mundapo bwino ubulwele bwa sugar.

   Muku palanya nokulengula kumbi ukufwaya ukufumya umulopa, limo limo kuti kwaba ukumfwa ulunshingwa, ukufimba elo nokukalipa kumulandi wa kutungaulwa ne nshindano elyo bashingânga balelwisha ukufumya umulopa.

8. **Amafuto**

   Ukuibimba muli uku kufwailisha ubwingi bwa sugar mumulopa takulemipela insambu sha ku mufuta mu musango uli onse.

9. **Amasanso Muli Uku Fwailisha**

   Ngachakweba ati abaibimbile muli uku kufwailisha bachenekwa, kafwailisha aka bombesha ukumona ukuti mwaloleshewapo bwino pakuti tachitwlele ku bubi.

   Ngachakweba ati namukwata amepusho pali uku kufwailisha, kuti mwamona ka fawailisha mukalamba nangula umukalamba wakabungwe akalolesha pa milandu yakufwailisha pe sukukulu ya masambililo yakalamba pe tweyala uti pe samba;

10. **Akeyala Ka Kafwailisha Mukalamba**

Emmanuel Mwila Musenge
The University of Zambia
School of Medicine
Department of Physiological Sciences
P.O. Box 50110
Ridgeway Campus
Cell No: +260977885979

Email: emmasenge@yahoo.com

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ZAMBIA

11. Akeyala Ka Umukalamba Uwulolesha Pa Kabungwe Ka Kufwailisha

The Chairperson

The University of Zambia

School of Medicine

Biomedical Research Ethics Committee

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LUSAKA

ZAMBIA

Ngacakweba ati mwasumina ukuibimbamo saineni akapepala aka pesamba;

APPENDIX D

TRANSLATED INFORMED VOLUNTARY CONSENT FORM

ICHIPEPALA CHAKUSUMINA MU KUIPELESHYA

SOSA PALWALALA

Nimbelenga (nangula naba nondondolwela) amashiwi ayalekuma ukulengula sugar mubalwele ba bulwele bwa sugar ngafilya fine yalembelwe mu chipepa cha ifyo abaleibimba muli uku kufwailisha pa bulwele bwa sugar bafwile ukwishiba. Kaili nachipelwa akashita aka kwipusha amepusho pali uku kufwailisha elo nabanjasuka fye bwino amepusho nachipusha.

Nomba nasumina mu kuipelesha ukuti ningaibimbamo muli uku kufwailisha. Elo ninjishiba ukuti ninkwata insambu shaku kana yasuka amepusho eyo nshilefwaya ukwasuka muli uku kufwailisha.

Uku saina kwandi pe samba kulepilibula uku ninsumina ukuibimbamo muli uku kufwailisha:

Ishina lyenu (Lembeni bwino): ……..….....……

Saineni: ……..…… Ubushiku bwa kusumina: ……...……....

Fwatikeni ne chikumo cha kukulyo ngatamwaishiba ukulemba: ……..…..……

Ishina yaba kafwailisha (Lembeni bwino): …….……..…….…….……

Saineni: ……..…… Ubushiku: …….....……

Ishina yaba kamboni (Lembeni bwino): ……..…….…….

Saineni: ……..……..….. Ubushiku: ……...……..…….....…...

APPENDIX E

ASSENT FORM

Study Title: "Examining Blood Glucose Control Status and Associated Factors in Diabetes Mellitus Out-patients at the University Teaching Hospital, Lusaka, Zambia"

Investigator: Emmanuel Mwila Musenge

We are doing a research study about your blood sugar levels and factors associated with these sugar levels. A research study is a way to learn more about people. If you decide that you want to be part of this study, you will be asked some questions about your disease and there after a very small amount of blood will be drawn from your vein.

There are other things about this study you should know. The drawn blood will be taken to the laboratory for testing so as to check for the blood sugar levels. This procedure of drawing blood will only be done once and it takes less than five minutes. The procedure of drawing a blood sample is considered a safe and just slightly painful procedure. However, as with many medical tests, drawing blood can lead to fainting or feeling dizzy, some swelling at the site or pain associated with multiple punctures to locate a vein.

Not everyone who takes part in this study will benefit. A benefit means that something good happens to you. We think these benefits might be that, after the tests are done, your blood sugar levels will be known and this will help the doctors to treat your sugar disease better.

When we are finished with this study we will write a report about what was learned. This report will not include your name or that you were in the study.

You do not have to be in this study if you do not want to be and you do not have to answer questions you feel you do not want to. If you decide to stop after we begin, that's okay too. Your parents know about the study too.

If you decide you want to be in this study, please sign your name.

I, _____, want to be in this research study.

Sign your name here_____ Date _____

Signature of person obtaining assent_____ Date: _____

APPENDIX F

PARENTAL/GUARDIAN INFORMED CONSENT FORM

Study Title: "Examining Blood Glucose Control Status and Associated Factors in Diabetes Mellitus Out-patients at the University Teaching Hospital, Lusaka, Zambia"

Investigator: Emmanuel Mwila Musenge

Introduction

Your child has been invited to join a research study to look at sugar disease. Please take whatever time you need to discuss the study with your family and friends, or anyone else you wish to. The decision to let your child join, or not to join, is up to you.

In this research study, we want to know the sugar levels in your child's blood and factors associated with these sugar levels.

What Is Involved In The Study?

Your child will be asked some questions about their disease and there after a very small amount of blood will be drawn from his/her vein. The drawn blood will be taken to the laboratory for testing so as to check for his/her blood sugar levels. This procedure of drawing blood will only be done once and it takes less than five minutes.

The investigators may stop the study or take your child out of the study at any time they judge it is in your child's best interest. They may also remove your child from the study for various other reasons. They can do this without your consent.

Your child can stop participating at any time. If your child stops he/she will not lose any benefits.

Risks

This study involves the following risks;

Very likely: Pain at site of puncture

Less likely but serious: Some swelling at the site of puncture

Rare: Fainting or feeling dizzy

Benefits to Taking Part in the Study?

It is reasonable to expect the following benefits from this research: after the tests are done, your blood sugar levels will be known and this will help the doctors to treat your sugar disease better and prevent complications such as blindness, kidney diseases among others. However, we cannot guarantee that your child will personally experience benefits from participating in this study. Others may benefit in the future from the information we find in this study.

Confidentiality

Your child's name will not be used during the study and when data from this study are published. Every effort will be made to keep clinical records, research records, and other personal information confidential. After the study, research records containing personal information will be destroyed.

Incentives

The participation of your child in this research has no provision for compensation/reimbursement of whatever form.

Your Rights as a Research Participant?

Participation in this study is voluntary. Your child has the right not to participate at all or to leave the study at any time. Deciding not to participate or choosing to leave the study will not result in any penalty or loss of benefits to which your child is entitled, and it will not harm his/her relationship with the investigators and other hospital staff.

Permission for a Child to Participate in Research

As parent or legal guardian, I authorise _____ (child's name) to become a participant in the research study described in this form.

Child's date of birth: _____

Parent or Legal guardian's signature: _____ Date: _____

Signature of person obtaining consent: _____ Date: _____

Contacts for Questions or Problems?

If you have questions about the study, any problems, if your child experiences any unexpected physical or psychological discomforts, any injuries, or think that something unusual or unexpected is happening, contact the principle investigator or chairperson of the UNZA biomedical research ethics committee at the addresses below:

Contact Details of Principal Investigator

Emmanuel Mwila Musenge

The University of Zambia

School of Medicine

Department of Physiological Sciences

P.O. Box 50110

Ridgeway Campus

Cell No: +260977885979

Email: emmasenge@yahoo.com

LUSAKA

ZAMBIA

Contact Details of Ethics Committee

The Chairperson

The University of Zambia

School of Medicine

Biomedical Research Ethics Committee

P. O. Box 50110

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APPENDIX G

TRANSLATED ASSENT FORM

ICHIPEPALA CHA KUSUMINA

Umutwe Wa Uku Kufwailisha: "Ukulengula sugar mu mulopa wabalwele ba sugar pa chipatala chikalamba icha UTH muno Lusaka mu chalo cha Zambia"

Kafwailisha: Emmanuel Mwila Musenge

Tulefwailisha ubwingi bwa sugar elo nefilenga ukuti sugar uyu alefula mu mulopa wobe. Ukufwailisha ninshila eyo tusambililapo ifingi mu bantu. Ngachakweba ati ulefwaya ukuibimbamo muli uku kufwailisha, ba kafwailisha balakwipusha amepusho pa bulwele bwa sugar elo notumulopa utunonofye twalabulwa ukufuma mutumipaipi twa mulopa wenu. Kaili ufwile ukwishiba ati utumulopa utwalabulwa twalatutwala kukupima pakuti tulengula ifyo sugar ali.

Umulopa walasendwafye umukumo kaili ukubulwa umulopa kusendafye ba minute basano. Ukufumya umulopa mu tumipaipi twamulopa takwakwata amafya elo tachikalipa sana. Muku palanya nokulengula kumbi ukufwaya ukufumya umulopa, limo limo kuti kwaba ukumfwa ulunshingwa, ukufimba elo nokukalipa kumulandi wa kutungaulwa ne nshindano elyo bashingânga balelwisha ukufumya umulopa.

Ubusuma bwa uku kufwailisha bwakeba ati, ukulengula nga kwapwa, twalaishiba imifulile ya sugar mumulopa wenu. Ichi chikankala sana mukwafwilishako bashingânga uku mundapo bwino ubulwele bwa sugar.

Te bonse abaleibimba muli uku kufwailisha abalesangamo ubusuma. Tuletontonkanya ukuti, umulopa wobe ngabaupima, ubwingi bwa sugar bwalaishibikwa elo ichi chikankala sana pakundwapo bwino nabashinganga.

Ngatwapwisha ukufwailisha pa bulwele bwa sugar, tukalemba ifyo tukasambililapo. Efyo tukalemba tafyakakukume munshila iyiliyonse.

Temulandu ngataulefwaya ukuibimbamo muli uku kufwailisha elo ngataulefwaya ukwasuka amepusho ayo tausekelemo wiyasuka. Kaili ngawachinja amano nangula nauibimbamo kale muli uku kufwailisha, kuti wafumamo temulandu iyoo. Abafyashi bobe natubeba nabo pali uku kufwailisha.

Ngachakweba ati ulefwaya ukuibimbamo muli uku kufwailisha, saina ishina lyobe pe samba.

Ine, _____, ndefwaya ukuibimbamo muli uku kufwailisha.

Saina ishina lyobe apa _____ ubushiku: _____

Ukusaina kwa ka kafwailisha _____ Ubushiku: _____

APPENDIX H

TRANSLATED PARENTAL/GUARDIAN INFORMED CONSENT FORM

ICHIPEPALA CHAKUSUMINA MU KUIPELESHYA NO MUFYASHI NAGULA UMULINSHI WA MWANA

Umutwe Wa Uku Kufwailisha: "Ukulengula sugar mu mulopa wabalwele ba sugar pa chipatala chikalamba icha UTH muno Lusaka mu chalo cha Zambia"

Kafwailisha: Emmanuel Mwila Musenge

Ichantanshi

Umwana wenu naitwa ukuibimbamo muli uku ukufwailisha pabulwele bwa sugar. Fwayeni inshita iya kuti mwingalanshyanya nendupwa shyenu nangula abanenu pali uku kufwailisha. Chilikuli imwe ukuti umwana wenu engaibimbamo muli uku kufwailisha.

Muli uku kufwailisha, tulefwaya ukwuishiba ubwingi bwa sugar mu mulopa wa mwana wenu elo nefilenga ukuti sugar alefula.

Efyo Mufwile Ukuwishiba Pali Uku Kufwailisha?

Umwana wenu bala mwipushako amepusho pa bulwele bwakwe elo utumulopa utulingilefye twalabulwa ukufuma mutumipaipi twamulopa. Utu tumulopa twala tutwala ku kupima nabamashini pakuti tumone ubwingi bwa sugar mumulopa. Umulopa walasendwafye umukumo kaili ukubulwa umulopa kusendafye ba minute basano.

Bakafwailisha kuti baleka uku kuwailisha nangula kuti ba mufumyamo umwana wenu muli uku kufwailisha ngabamona ukuti chalaleta ubwafya ku mwana wenu. Kuti bachita ifi ukwabula ukumyeba.

Umwana wenu ngachakweba ati bamufumyamo muli uku kufwailisha, tachilepilibula ukuti ninshi tabamutangate bwino iyoo.

Ububi Bwa Uku Kufwailisha

Muku palanya nokulengula kumbi ukufwaya ukufumya umulopa, limo limo kuti kwaba uku kalipa no kufimba epo baletunga elo no kumfwa ulunshingwa.

Ubusuma Bwakuibimba Muli Uku Kufwailisha?

Ubusuma bwa uku kufwailisha bwakeba ati, ukulengula nga kwapwa, twalaishiba imifulile ya sugar mumulopa wenu. Ichi chikankala sana mukwafwilishako bashingânga ukumundapo bwino ubulwele bwa sugar elo nokulesha ubulwele ukuya pantanshi. Telyonse elyo umwana wenu enganonkelamo muli ububusmuma nomba bambi kuti banonkelamo umuyenshiku.

Inkama

Ishina lya mwana wenu tatwaibomye muli uku kufwailisha iyoo. Kaili tuleesha namaka ukuti fyonse ifipepala ifyotulebomya tulefisunga munkama. Ngatwapwa uku kufwailisha tukafyocha fyonse ifipepala tulebomya.

Amalipilo

Ukuibimba muli uku kufwailisha ubwingi bwa sugar mumulopa takulemipela insambu sha kumulipila mu musango uli onse.

Insambu Shenu Ngabaleibimba Muli Uku Kufwailisha?

Ukuibimba muli uku kufwailisha kuipelesha. Umwana wenu nakwata insambu shakukana yibimbamo muli uku kufwailisha. Kaili nakwata insambu shakufumamo muli uku kufwailisha inshita iiliyonse. Umwana wenu ngafumamo muli uku kufwailisha tachilepilipbula ukuti twala leka ukumundapo bwino pamo ngabakafwailisha nagula ababomfi bamuchipatala.

Insambu Shakusuminisha Umwana Wenu Ukuibimbamo Mulu Uku

Kufwailisha

Ngomufwashi nangula umulinshi wa uyu mwana, ndemipela amaka ayakuti _____ (ishina lyamwana) engaibimbamo muli uku kufwailisha ngafilyafine mulondolwele muli ichi chipepala.

Ubushiku umwana afyelwe: _____

Ukusaina kwa mufyashi nangula umulinshi: _____

Ubushiku: _____

Ukusaina kwa ka kafwailisha: _____ Ubushiku: _____

Utweyala Nganamukwata Amaepusho Nangula Amafya?

Ngachakweba ati namukwata amepusho pali uku kufwailisha nangula amafya, kuti mwamona ka fawailisha mukalamba nangula umukalamba wakabungwe akalolesha pa milandu yakufwailisha pe sukukulu ya masambililo yakalamba pe tweyala uti pe samba;

Akeyala Ka Kafwailisha Mukalamba

Emmanuel Mwila Musenge

The University of Zambia

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Akeyala Ka Umukalamba Uwulolesha Pa Kabungwe Ka Kufwailisha

The Chairperson

The University of Zambia

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ZAMBIA

APPENDIX I

STRUCTURED INTERVIEW SCHEDULE

THE UNIVERSITY OF ZAMBIA

SCHOOL OF MEDICINE

DEPARTMENT OF PHYSIOLOGICAL SCIENCES

STRUCTURED INTERVIEW SCHEDULE

TOPIC: EXAMINING GLYCAEMIC CONTROL STATUS AND ASSOCIATED FACTORS IN DIABETES MELLITUS OUT-PATIENTS AT THE UNIVERSITY TEACHING HOSPITAL, LUSAKA, ZAMBIA

DATE OF INTERVIEW…….....

PLACE OF INTERVIEW……

NAME OF INTERVIEWER……

SERIAL NUMBER ☐

INSTRUCTIONS TO INTERVIEWER

1. Introduce yourself to the respondent
2. Explain the purpose of the interview
3. Get written consent from the respondent
4. Reassure the respondent that all responses will be held in strict confidence
5. Individual names and addresses should not appear on the interview schedule
6. Ensure that all questions are answered and indicate response by ticking in the appropriate box (e.g. √) or filling in the space (s) provided
7. Thank the respondent at the end of each interview.

SECTION A

DEMOGRAPHIC DATA

FOR OFFICIAL USE ONLY

1. Age
   1) 15 - 34 Yrs [ ]
   2) 35 - 54 Yrs [ ]
   3) 55 and above [ ]

2. Sex
   1) Male [ ]
   2) Female [ ]

3. Education level
   1) Never been to school [ ]
   2) Primary school [ ]
   3) Secondary school [ ]
   4) College [ ]
   5) University [ ]

SECTION B

SELF-MANAGEMENT BEHAVIOUR DATA

4. Adherence to anti-diabetic treatment?
   1) No [ ]
   2) Yes [ ]

5. Reasons for non-adherence to anti-diabetic treatment
   1) Stock-out [ ]
   2) Forget [ ]
   3) Others [ ]
   4) Not applicable [ ]

6. Self-blood glucose monitoring at home?

1) No [ ]
2) Yes [ ]

7. Self-blood glucose monitoring means at home?

1) Own glucometer [ ]
2) Public health facility [ ]
3) Private health facility [ ]
4) Not applicable [ ]

8. Involvement in any exercise?

1) No [ ]
2) Yes [ ]

SECTION C

CLINICAL DATA

9. Type of diabetes mellitus

1) Type 1 [ ]
2) Type 2 [ ]

10. Diabetes mellitus duration …….

11. Anti-diabetic treatment type

1) Oral anti-diabetic drugs [ ]
2) Insulin [ ]
3) Oral antidiabetic drugs and insulin [ ]
4) Diet only/none [ ]

12. Non-antidiabetic treatment type

1) Antihypertensives [ ]
2) Antiretrovirals [ ]
3) Aspirin [ ]
4) Septin [ ]
5) Others, specify…….

13. Co-morbidity?

1) Infection [ ]
2) Anaemia [ ]
3) Alcoholism [ ]
4) Hypertension [ ]
5) Other, specify……

14. Body mass index

1) Underweight (< 18.5 kg/m^2) [ ]
2) Normal (18.5 – 24.9 kg/m^2) [ ]
3) Overweight (25 – 29.9 kg/m^2) [ ]
4) Obesity (30 or greater kg/m^2) [ ]

15. Any family member with DM?

1) No [ ]
2) Yes [ ]

16. Blood pressure……..

SECTION D

LABORATORY MEASUREMENT RESULTS

17. HbA1c Test Result……

1) Poor [ ]
2) Good [ ]

18. Previous three months FPG Test Result……

19. Current FPG Test Result……

END OF INTERVIEW

THANK YOU!

APPENDIX J

Department of Physiological Sciences
School of Medicine
University of Zambia
P. O. Box 50110
Ridgeway Campus
LUSAKA

24th June, 2013

The Managing Director
University Teaching Hospital
P/B RW1
LUSAKA

UFS: The Head - Department of Physiological Sciences

Dear Sir / Madam,

RE: PERMISSION TO CONDUCT RESEARCH

I am a postgraduate student pursuing Master of Science in Human Physiology (MSc. PGY) degree programme at the University of Zambia, School of Medicine, Department of Physiological Sciences. As part of the programme requirements I have to undertake a dissertation. It is in this premise that I write to seek permission to undertake a research at your institution. The title of the research is **"examining blood glucose control status in diabetes mellitus (DM) out-patients at the University Teaching Hospital (UTH), Lusaka, Zambia".** I intend to carry out the study from September, 2013 to January, 2014 at clinic five.

It is my hope that the findings will help in strengthening the management of DM patients at UTH and the country as a whole.

Your favourable response to my request will highly be appreciated.

Yours faithfully,

Musenge M. Emmanuel (Computer No. 531001882)

Contacts: Email: emmasenge@yahoo.com
Cell: +260977885979