FACTORS THAT AFFECT GLYCAEMIC CONTROL AMONG TYPE 2 DIABETES MELLITUS PATIENTS IN KWAHU SOUTH DISTRICT EASTERN REGION, GHANA

BY

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10359486

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JULY, 2012
DECLARATION

This work is the result of an independent investigation under the supervision of Dr Priscillia Nortey. Where my work is indebted to the work of others, I have made acknowledgement. I declare, therefore that this dissertation has not been presented elsewhere, either in part or in whole for another degree

Resident: .................................

John Tengey

Supervisor: .................................

Dr. Priscillia Nortey
DEDICATION

This piece of work is dedicated to my loving wife Nana Yaa Nifaa Tengey for her immeasurable support, encouragement, and understanding. It is also dedicated to all diabetic patients.
ACKNOWLEDGEMENT

This work has had the support of several people.

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To Cohort four residents who made this work a success, your encouragement, support and contributions are well appreciated.
ABSTRACT

Diabetes mellitus (DM) has become one of the most common chronic diseases globally, and it has assumed an epidemic status in the past few decades especially in low and middle income countries. The most frequent form is Type 2 diabetes which represents more than 85% of cases; other forms are Type 1 (10%) and Gestational diabetes (5%).

Type 2 diabetes is a progressive disease associated with an increased risk of developing several complications which include; cardiovascular diseases, diabetic retinopathy and nephropathy. These complications are directly and strongly related to hyperglycaemia. Aggressive treatment to control hyperglycaemia is much more effective in reducing the number of complications than standard treatment. The study aims to determine the proportion of poor glycaemic control and identify the factors affecting the glycaemic control of type 2 DM.

The glycated haemoglobin (HbA1c) of 382 type 2 diabetes mellitus patients attending diabetic clinic at Atibie Government Hospital were determined. Simple random sampling was applied to select 100 patients who had HbA1c > 6.5% as cases, and 100 with HbA1c < 6.5% as controls. Using a multivariate logistic regression model, factors that are associated with poor glycaemic control were determined. Majority had well controlled glycaemic status (64%). The commonest comorbidity was hypertension (60.5%). Patients with no family support are six times more likely to have poor glycaemic control as compare to those with family support (OR 6.40, 95% CI 1.70, 23.90, p= 0.006). Income level less than Gh₵ 250, and High Density Lipoprotein were strongly associated with poor glycaemic control (OR 0.063, 95% CI 0.008, 0.505 p=0.009, OR 0.31, 95% CI 0.17, 0.56, p<0.001) respectively.
Prevalence of poor glycaemic control was 36%. Low income level and HDLP were significant risk factor for poor glycaemic control. We recommended that HbA1c assay equipment and reagents should be provided for all district hospitals, regular lipid profile should be carried out at regular short intervals. Diabetic patients with low financial status should be assisted.

**Keywords:** Type 2 diabetes mellitus, glycaemic control, Atibie hospital, Ghana
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LIST OF ACRONYMS

ACE  Angiotensin-Converting Enzyme
ADA  America Diabetes Association
AFR/RC  Africa Region/ Research Committee
BMI  Body Mass Index
BP  Blood Pressure
CVD  Cardiovascular Disease
DBP  Diastolic Blood Pressure
DM  Diabetes Mellitus
EDTA  Ethylene Diaamine Tetra-Acetic Acid
FBS  Fasting Blood Sugar
FPG  Fasting Postprandial
GDA  Ghana Diabetes Association
HbA1c  Glycated Haemoglobin
HDL  High Density Lipoprotein
HDL-C  High Density Lipoprotein- Cholesterol
HPT  Hypertension
IDF  International Diabetes Federation
JNC  Joint National Committee on Prevention
LDL  LowDensity Lipoprotein
NCD  Non Communicable Disease
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<td>National Health Insurance Scheme</td>
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<td>Oral Hypoglycaemic Agent</td>
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<td>OPD</td>
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<td>Subcutaneous Adipose Tissue</td>
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<tr>
<td>SBP</td>
<td>Systolic Blood Pressure</td>
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<td>SSA</td>
<td>Sub-Saharan Africa</td>
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<td>TG</td>
<td>Triglycaeride</td>
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<td>UKPDS</td>
<td>United Kingdom Prospective Diabetes Study</td>
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<td>VAT</td>
<td>Visceral Adipose Tissue</td>
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<td>VLDL</td>
<td>Very Low Density Lipoprotein</td>
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CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

Diabetes mellitus (DM) is a chronic non communicable disease whose global spread has given it the characteristics of a pandemic. The most frequent form is Type 2 diabetes which represents more than 85% of cases; other forms are Type 1 (10%) and gestational diabetes (5%) (WHO/IDF, 1999).

In type 1 diabetes mellitus, about 85% of the beta cells in the pancreas are destroyed due to autoimmune disorder while 35-50% are destroyed due to genetics. Typically, its onset is from childhood but it can also affect people of all ages. Onset is mostly sudden and requires daily insulin injection for survival and is prone to the development of ketoacidosis (40%) (ADA, 2002). The annual incidence of type 1 diabetes mellitus varies from 4 to 10 per 100 000 among the 0 to 19 year old population in Africa, with a high mortality rate (WHO/IDF, 1999).

Gestational diabetes first presents itself in pregnancy but may disappear after delivery and reappear shortly thereafter or follow a long-term latency period. It usually has a type 2 clinical presentation and develops in about 2-5% of all pregnancies (ADA, 2002).

Type 2 DM is the most prevalent form of diabetes forming about 85-95% of all diabetes mellitus; it is due to the combination of insulin resistance and defective secretion of insulin by the beta-cells (Grundy et al 1999). Insulin resistance is due to lifestyle disorder and causes about 80-90% while 74-100% is due to genetic disorder. It is common in adults aged over 40 years who are overweight and obese. Over 80% of people with type 2 diabetes are overweight and obese. Patients suffering from Type 2 diabetes usually control their blood
glucose by controlling their diet, carrying out regular exercises and possibly by taking drugs and or insulin injection (WHO, 2009)

Diabetes mellitus (DM) is serious due to its complications, namely: cardiovascular ailments, cerebrovascular accidents, renal insufficiency, blindness, sexual impotence and gangrene of the feet leading to amputation. It is insidious on onset and more than 50% of patients are usually not aware of the existence of the disease. Deaths from DM form about 5% of global death and it is estimated this will likely increase by 50% in the next 10 years. Of these deaths, 80% occur in Low and Middle Income Countries (WHO, 2009). There is an increased risk for Cardiovascular Disease (CVD) and 50% of CVD deaths have DM; coronary heart disease may affect 5% to 8% of type 2 diabetic patients and cardiomyopathy, up to 50% of all patients. Close to 15% of patients with stroke have diabetes, and up to 5% of diabetic patients present with cerebrovascular accidents at diagnosis (Kengne et al, 2005).

Sub-Saharan Africa (SSA) now faces a double disease burden, with emerging non communicable diseases (NCD), such as arterial hypertension, stroke, coronary heart disease and diabetes mellitus (DM) added to the challenges in infectious diseases (Beaglehole et al 2003). In a survey conducted in 2005 in SSA, the prevalence of DM was estimated to be 16% (Longo-Mbenza et al 2008).

One significant factor which influences insulin resistance is diabetic dyslipidaemia or hyperlipidaemia which refers to an increase in concentration of one or more plasma or serum lipids, usually cholesterol and triglycerides. These lead to increased release of fatty acids from adipose tissue. Increased plasma levels of fatty acids increase production of Very Low Density Lipoprotein (VLDL), Triglycerides (TG), and cholesterol by the liver. This is a common abnormality seen in both obese and non-obese insulin-resistant subjects and those with type 2 diabetes (Goldberg, 2001). Elevated total cholesterol and higher levels of High
Density Lipoprotein-Cholesterol (HDL-C) are significantly associated with poor control of HbA1c (Longo-Mbenza et al 2008).

1.2 Problem statement

The increasing prevalence of Type 2 diabetes mellitus in the past few decades with associated complications as a result of poor glycaemic control is much more severe in low and middle income countries (WHO/AFR/RC, 2007). In Ghana, it is estimated that prevalence of diabetes is about 3% (IDF, 2012).

The glycated haemoglobin assay (HbA1c) plays a central role in the monitoring of glycaemic control largely because glucose molecules react with haemoglobin forming glycated haemoglobin. The glycated haemoglobin molecule remains that way for 3 months. This is a more robust reflection of glycaemic control than fasting blood glucose estimation. The HbA1c assay is the main method by which clinicians can relate individual blood glucose control to risk of developing complications (Sacks et al, 2002, Manley et al, 2003, Apple, 2010).

The treatment guidelines on diabetes in Ghana still uses the fasting blood glucose for the monitoring of DM, but recommends HbA1c assay two to three times a year (GNDP, 2010). Many clinicians are unable to request for HbA1c assay due to its lack of availability especially in the peripheral health facilities, even though they know its importance. Furthermore, when available some patients are reluctant in doing regular HbA1c monitoring because of cost since HbA1c is not covered by the NHIS.

Relatively, few studies have been done the in the sub-Saharan Africa region as compare to the developed countries on the risk factors that affect glycaemia. The Atibie Government hospital
is a district hospital with a very large diabetic clinic, the clinic attends to about 200 (mostly Type 2 diabetic) patients per week. This study will attempt to elucidate the proportion of patients with poor glycaemic control and the risk factors associated with poor glycaemic control in this district hospital.

1.3 Justification

This study comes at a time that WHO African Region has presented a strategy urging Member States to evaluate the magnitude of the problem of diabetes and identify primary, secondary and tertiary prevention activities. The goal of this regional strategy is to contribute to the reduction of the burden of diabetes related morbidity and mortality (WHO AFR/RC, 2007).

In a 2002 study in a Teaching Hospital in Accra, Amoah et al (2002) found that increasing age, body mass index systolic and diastolic BP were associated with poor glycaemic control. Titty (2010) observed a prevalence of poor glycaemic control of 60% among type 2 diabetic patients in a study done in the Tamale Teaching Hospital. The Atibie Government Hospital has many of its patients being indigenes with farming and trading being the main occupation. There is the need to determine the proportion of patients in this district clinic with poor glycaemic control and the factors contributing to this. The finding from the study could provide some necessary data on the factors affecting the control of Type 2 diabetes mellitus. It could also influence policy makers in the formulation of new management policies on diabetes mellitus.
1.4 Objectives

General objective:

To determine the factors that affect the control of type 2 diabetes mellitus among patients attending the diabetes clinic at Atibie Hospital in the Kwahu South District.

Specific objectives:

- To determine the proportion of glycaemic control among type 2 diabetes mellitus patients in Kwahu South District.
- To identify factors affecting the control of type 2 diabetes mellitus in Kwahu South District.
CHAPTER TWO

2.0 LITERATURE REVIEW

Diabetes mellitus (DM) presents with a disorder in the metabolism of Carbohydrate, Protein and Fat which is characterized by chronic hyperglycaemia, resulting from defective secretion or action of insulin (insulin resistance) or both. Symptoms of diabetes include polydipsia (increased thirst), polyuria (increased urine volume), recurrent infections, and unexplained weight loss. In severe cases, drowsiness, coma and high levels of glycosuria are usually present.

Insulin Pancreatic beta cells are found in the islets of Langerhans, which are of various sizes and contain a few hundred to a few thousand endocrine cells. Islets are anatomically and functionally separate from pancreatic exocrine tissue (which secretes pancreatic enzymes and fluid directly into ducts that drain into the duodenum). Normal subjects have about one million islets that in total weigh 1 to 2 grams and constitute 1 to 2% of the mass of the pancreas.

Islets vary in size from 50 to 300 micrometres in diameter. They are composed of several types of cells. At least 70% are beta cells, which are localized in the core of the islet. These cells are surrounded by alpha cells that secrete glucagon. Insulin secreted from beta cells suppresses glucagon secreted from alpha cells. It is the only pancreatic β-cell hormone known to lower blood glucose concentrations. Insulin, a small protein composed of two polypeptide chains containing 51 amino acids, is a key anabolic hormone that is secreted in response to increased blood glucose and amino acids following ingestion of a meal. Like many hormones, insulin exerts its actions through binding to specific receptors present on many
cells of the body, including fat, liver, and muscle cells. The primary action of insulin is to stimulate glucose disappearance.

Insulin helps control postprandial glucose in three ways. Initially, insulin signals the cells of insulin-sensitive peripheral tissues, primarily skeletal muscle, to increase their uptake of glucose (Koda et al, 1992) as shown in the figure 1. Secondly, insulin acts on the liver to promote glycogenesis. Finally, insulin simultaneously inhibits glucagon secretion from pancreatic α-cells, thus signalling the liver to stop producing glucose via glycogenolysis and gluconeogenesis. All of these actions reduce blood glucose (Drucker et al 2001).

Other actions of insulin include the stimulation of fat synthesis, promotion of triglyceride storage in fat cells, promotion of protein synthesis in the liver and muscle, and proliferation of cell growth (Drucker et al, 2001).

Insulin action is carefully regulated in response to circulating glucose concentrations. Insulin is not secreted if the blood glucose concentration is ≤ 3.3 mmol/l, but is secreted in increasing amounts as glucose concentrations increase beyond this threshold, (Ogawa et al, 1990). Postprandially, the secretion of insulin occurs in two phases: an initial rapid release of preformed insulin, followed by increased insulin synthesis and release in response to blood glucose. Long-term release of insulin occurs if glucose concentrations remain high (Drucker et al, 2001, Ogawa et al, 1990). Other factors that stimulate insulin secretion are the increased plasma concentrations of some amino acids released from the gut following a meal (Weyer et al, 2001).
Diabetes Mellitus is confirmed with a random venous plasma glucose higher than 2g/l (11.1 mmol), or fasting glycaemia that is higher than 1.26g/l (7.0 mmol/l) at two tests, or a fasting glycaemia higher than 2g/l (11.1 mmol) 2 hours after a glucose intake (WHO/ IDF, 2006).

During the normal 120-day life span of the red blood cell, glucose molecules react with
haemoglobin forming glycated haemoglobin. A few decades ago researchers discovered that in individual with poorly controlled diabetes, the quantities of these glycated haemoglobins are much higher than in healthy people (Apple, 2010). Once a haemoglobin molecule is glycated, it remains that way.

A build-up of glycated haemoglobin within the red blood cell therefore reflects the average level of glucose to which the cell has been exposed during its 3 month life cycle. This is called HbA1c or glycated haemoglobin and it is used by doctors (and patients) to monitor blood sugar control in diabetic patients (Apple, 2010).

Until 2010, the HbA1c test was used only for monitoring patients once they had been diagnosed with diabetes. In January 2010, however, the American Diabetes Association Standards of Medical Care in Diabetes added the measure of HbA1c 6.5% as a criterion for the diagnosis of diabetes, in addition to the fasting glucose test and the oral glucose tolerance test. Measurement of glycated proteins, primarily glycated haemoglobin, is widely used for routine monitoring of long-term glycaemic status in patients with diabetes mellitus. (Apple, 2010).

2.1 Burden of Diabetes Mellitus

Type 2 diabetes mellitus is the most prevalent form of diabetes. It appears later in life, and it is due to the combination of insulin resistance (impairment in insulin-mediated glucose disposal) and defective secretion of insulin by pancreatic β-cells (Grundy et. al, 1999). Diabetes has become one of the most common chronic diseases globally (figure 2).
Figure 2: Global Prevalence of Type 2 Diabetes in 2000 and estimated prevalence in 2030.

Credit;


In Latin America, the prevalence of Type 2 diabetes is highest among Pima Indians, followed by Hispanics, blacks, and then whites (Ismail & Gill, 1999). Ethnic group, age (≥ 40 years), dietary intake, obesity, and lack of physical activity are associated with a higher prevalence of diabetes (Choi & Shi, 2001). The prevalence of diabetes mellitus and impaired glucose tolerance were 10.5% and 16.5% in Kelantan state of north-east Malaysia (Mafauzy et. al, 1999). The prevalence of new cases of diabetes in United Kingdom were 0.2% (0 to 1.4%) and 2.8% (1.6% to 4.7%) in patients whose sole risk factor was age over 45 and in patients aged over 45 with one or more additional risk factors for diabetes, respectively (Lawrence et. al, 2001).
The estimated prevalence of diabetes in Africa is 1% in rural areas, up to 5% to 7% in urban Sub-Saharan Africa, and between 8% and 13% in more developed areas such as South Africa and in populations of Indian origin. The annual incidence of type 1 diabetes mellitus varies from 4 to 10 per 100,000 among the 0- to 19-year-old population in Africa, with a high mortality rate (Kengne et al 2005).

Up to 25% of people with diabetes have evidence of microvascular complications at diagnosis, and extrapolation of the association between the prevalence of retinopathy and the duration of disease suggests that the true onset of diabetes occurs several years before it is recognized clinically (Wareham & Griffin, 2001). The United Kingdom Prospective Diabetes Study and Kumamoto Study confirmed that, improved glucose control reduces the microvascular complication in type 2 diabetes such as retinopathy, and neuropathy (UKPDS 1998, Ohkubo et al, 1995). Because of these findings, new standard of care and new models of health care have emerged (ADA Diabetes care 2003).

Diabetes-related cardiovascular disease complications are considered to be rare in Africa but are on the rise and are regularly associated with classic cardiovascular risk factors. Coronary heart disease may affect 5% to 8% of type 2 diabetic patients and cardiomyopathy, up to 50% of all patients. Close to 15% of patients with stroke have diabetes, and up to 5% of diabetic patients present with cerebrovascular accidents at diagnosis. Peripheral vascular disease prevalence varies across sites from 4% to 28% (Kengne et al, 2005).

In Ghana the prevalence of diabetes mellitus increased from 0.4% in 1958 (Dodu, 1958) to 2-3% between 1988- 2000 (GDA, 2000) and according to a study in 2002 (Amoah et al, 2002), the crude prevalence of diabetes in all subjects was 6.3%, age standardization to the Ghanaian population yielded a diabetes prevalence of 6.1%, with higher prevalence in males (7.7) than females (5.5). Diabetes and endocrine disorder cases formed 6.3% of all admissions and 5.2%
of all deaths (1990-1997) in Korle-Bu Teaching Hospital (Amoah et al, 2004), while 44% of amputation is due to diabetes mellitus (Naaeder, 1997).

2.2 Glycaemic Control

Type 2 diabetes is a progressive disease associated with numerous serious complications that develop over time. Patients with type 2 diabetes are at increased risk for cardiovascular disease. These complications are directly and strongly related to hyperglycaemia (Stratton et. al, 2000). Hyperglycaemia affects biochemical parameters and influences the progression of coronary heart disease and mortality rates in diabetic patients. Aggressive treatment to control hyperglycaemia is much more effective in reducing the number of complications than standard treatment (Van der does et. al, 1998; Herman, 1999).

Measurement of glycated proteins, primarily glycated haemoglobin, is widely used for routine monitoring of long-term glycaemic status in patients with diabetes mellitus in Europe and United States (Apple, 2010), whereas fasting blood glucose is widely used in the routine monitoring of blood glucose in diabetes in Sub-Saharan Africa.

2.3 Factors Affecting Glycaemic Control

Female sex, longer DM duration, cigarette smoking, total hypercholesterolemia, elevated high density lipoprotein-cholesterol (HDL-C), high blood glucose and underweight were identified as independent risk factors of poor control of HbA1c. The decrease of oestrogens in female diabetics may explain their susceptibility to poor control of HbA1c. Oestrogen replacement therapy reduces weight in women and improves glycaemic control (Dalvi et al,
2010). Benoit et al (2005) found that the longer someone is diagnosed with diabetes, the more difficult it is to maintain glycaemic control. Contradictory to these findings is a research by Nichols et al (2000) who found that the duration of the diagnosis of the disease is not a significant factor for glycaemic control but rather there is poorer metabolic control among the younger age group.

2.3.1 Behavioural factors

Smoking does not affect the plasma concentrations of oral diabetes medications such as sulfonylureas and thiazolidinediones. However, the most profound impact of smoking in diabetic patients is on insulin sensitivity. Smoking decreases subcutaneous absorption of insulin, resulting in increased dosing requirements. When the action of insulin is impaired chronically in smokers, a dose-response relationship can be seen between the number of cigarettes smoked and the degree of insulin resistance (Targhar et al, 1997). The differences in insulin sensitivity may be caused by the direct effects of nicotine, carbon monoxide, or other chemicals in tobacco smoke. These factors also may alter the pathogenesis of early steps in insulin action, such as signal transduction or glucose transport. Furthermore, higher plasma triglycerides, lower HDL cholesterol, higher plasma insulin levels, and elevated systolic blood pressure levels are all typical findings in smokers and characterize the insulin resistance syndrome. Thus, smoking in patients with diabetes seems to bring out attributes of the insulin resistance syndrome, serving as a link between the use of cigarettes and cardiovascular disease (Reaven et al, 2003).

Smoking is a risk factor for the development of type 2 diabetes; it induces insulin resistance in persons with or without diabetes (Ronnemaa et al, 1996). Cigarette smokers have elevated total cholesterol (TC) and DM as reported elsewhere (Zavaroni et al, 1994). Smoking also induces insulin resistance and these cigarette smokers have elevated TC which was found to
be the main effect in predicting glucose control. This was similarly confirmed by Blaum et al (1997) in a related research on the subject.

A study carried out by Koivisto et al (1993) found an acute effect of alcohol consumption on type 2 diabetes; subjects experienced slightly elevated postprandial plasma insulin and slightly lower fasting plasma glucose levels the next morning. However, without any effect on postprandial plasma glucose and no hypoglycaemic episodes were observed. Also, there was no effect of alcoholic beverages on postprandial glucose, insulin or triglyceride levels in twelve type 2 diabetic patients (Christiansen et al, 1993).

On chronic alcohol intake, an impaired glycaemic control with higher HbA1c, fasting and postprandial plasma glucose values as compared to abstainers were observed (Ben et al, 1991). However, most studies show that longer-term alcohol intake is linked to a better glycaemic control in type 2 diabetes (Bantle et al, 2008).

Occasional episodes of alcohol consumption generally do not worsen blood sugar control in people with diabetes and may even have beneficial effects. Alcohol consumption is inversely associated with glycaemic control among diabetes patients (Ahmed et al, 2008).

2.3.2 Hypertension

Hypertension (defined as a blood pressure ≥140/90 mmHg) is an extremely common comorbid condition in diabetes, affecting ~20–60% of patients with diabetes, depending on obesity, ethnicity, and age (Arauz-Pacheco et al, 2002). Hypertension is twice as common in persons with diabetes as it is in others (Epstein et al, 1992). In sub-Saharan Africa prevalence rate of hypertension in diabetes is 66.7% (Choukem et al, 2007).

In type 2 diabetes, hypertension is often present as part of the metabolic syndrome of insulin resistance also including central obesity and dyslipidaemia. Hypertension substantially
increases the risk of both macrovascular and microvascular complications, including stroke, coronary artery disease, and peripheral vascular disease, retinopathy, nephropathy, and possibly neuropathy. Hypertensive diabetic patients are also at increased risk for diabetes-specific complications including retinopathy and nephropathy (Arauz-Pacheco et al, 2002).

In recent years, adequate data from well-designed randomized clinical trials have demonstrated the effectiveness of aggressive treatment of hypertension in reducing both types of diabetes complications (Fineberg SE, 1999, Bakris et al, 2000). In the U.K. Prospective Diabetes Study (UKPDS) epidemiological study, each 10-mmHg decrease in mean systolic blood pressure was associated with reductions in risk of 12% for any complication related to diabetes, 15% for deaths related to diabetes, 11% for myocardial infarction, and 13% for microvascular complications. Fortunately, reductions in blood pressure can decrease the risk of these complications (UKPDS, 1998).

To reduce this risk, hypertension must be diagnosed accurately and promptly, and the patient must receive adequate treatment. To confirm the diagnosis of hypertension, blood pressures measured with standard techniques should be elevated on two separate occasions (JNC VI, 1997). Because patients with diabetes and hypertension are at high risk for complications, consensus statements from the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI, 1997), the American Diabetes Association (ADA, 2002) and the National Kidney Foundation (NKF) Hypertension and Diabetes Executive Committees Working Group recommend lower blood pressure goals for patients with diabetes than for the general population.

There is a strong epidemiological connection between hypertension in diabetes and adverse outcomes of diabetes. Studies by Ferrannini et al, 1992, conclude that the presence of
hypertension is not associated with significant changes in glycaemic control in type 2 diabetes.

**Treatment of hypertension in Diabetes**

Because many studies demonstrate the benefits of angiotensin-converting enzyme (ACE) inhibitors on multiple adverse outcomes in patients with diabetes, including both macrovascular and microvascular complications, in patients with either mild or more severe hypertension and in both type 1 and type 2 diabetes, the established practice of choosing an ACE inhibitor as the first-line agent in most patients with diabetes is reasonable. However, other strategies including diuretic and β-blocker–based therapy are also supported by evidence. Treatment decisions should be individualized based on the clinical characteristics of the patient, including comorbidities as well as tolerability, personal preferences, and cost.

**2.3.3 Diabetic Dyslipidaemia**

According to American Diabetic Association, the central characteristic of dyslipidaemia in patients with Type 2 diabetes is an elevated triglycerides level, particularly triglycerides-rich very low density lipoprotein (VLDL) levels and decreased HDL cholesterol levels. In diabetic patients, the concentration of LDL cholesterol is usually not significantly different from that seen in non-diabetic individuals.

However, patients with type 2 diabetes typically have a preponderance of smaller, denser, oxidized LDL particles, which may increase atherogenicity, even if the absolute concentration of LDL cholesterol is not elevated. This lipid triad, referred to as atherogenic dyslipidaemia, is usually present in patients with premature coronary artery disease. Atherogenic dyslipidaemia (diabetic dyslipidaemia) is characterized by 3 lipoprotein abnormalities: elevated very-low-density lipoproteins (VLDL), small LDL particles, and low
high-density-lipoprotein (HDL) cholesterol (the lipid triad). Despite the high and widespread prevalence of dyslipidaemia among people without and with diabetes, only 2.2% of adults without diabetes and 32% of diabetic patients are receiving treatment with diet, exercise, or drugs to reduce lipid levels and less than one third of patients with established cardiovascular disease received such treatment (Primatesta & Poulter, 2000).

Fifty eight percent of patients with good glycaemic control had dyslipidaemia and 75% of patients with poor glycaemic control had dyslipidaemia (Titty, 2010).

### 2.3.4 Body Mass Index and Waist Circumference

Obesity and overweight are now considered to be serious health problems, and the prevalence increasing worldwide (Marcellini et al, 2009) such that the World Health Organization (WHO) reported overweight and obesity to be a rising epidemic (Gyarfas, 1996). Scheen et al, suggested that; Obesity should be considered as a chronic disease with multi-factorial etiology, and treatment must be maintained for life-first with lifestyle interventions (energy-reduced diet and increased physical activity) and then with pharmacologic approaches, when necessary (Scheen et al, 2008). Obesity is an independent risk factor for a number of chronic diseases, including hypertension Diabetic Mellitus, cardiovascular disease and some cancers (Rohrer et al, 2007).

Having obesity with the coexistence of hypertension and diabetes increases the risk for macrovascular and microvascular complications, thus predisposing patients to cardiac death, congestive heart failure, coronary heart disease, cerebral and peripheral vascular diseases, nephropathy, and retinopathy (Zanella et al, 2001). Decrease in adiposity is one of the most effective preventive measures in decreasing not only the overall cardiovascular risk but also blood pressure and increasing DM control (Khan et al, 2008).
In Type 2 Diabetes Mellitus (DM), weight loss has a beneficial effect on indices of glycaemic control and treatment requirements and those who achieve pronounced weight loss experience normalization of insulin sensitivity and blood glucose concentration (Daousi et al, 2006). Waist circumference and body mass index showed the ability to predict HbA1c level although the prediction was much lower in men (Yoshida et al, 2009), even though, body mass index is the most widely used indicator of obesity, it does not measure abdominal fat mass which relates to an increased risk of cardiovascular disease (Kaur et al, 2008). Therefore waist circumference is often used as a proxy measure of abdominal adipose tissue, in particular, visceral adipose tissue (VAT) in clinical settings. VAT has been reported to create a greater risk for developing obesity related disorders than subcutaneous adipose tissue (SAT) (Nagaretani et al, 2001, Fox et al, 2007).

The findings suggest that increased level of HbA1c is associated with waist circumference as a modifiable factor (Zeinab et al, 2010). Any modifiable factors that influence glycemic control could be important (Blaha et al, 2008). In present study waist circumference was a predictor of increased level of HbA1c and thus it is the only modifiable factor in this regard. Yoshida and Okosun argued that physiologic factors are important in diabetes control where they have showed the association between glycaemic control and waist circumference (Yoshida et al, 2001, Okosun et al, 2002). However, Hartz et al (2006) suggested that patient factors such as understanding of diabetes and adherence to recommended behaviours and not physiologic factors are primary important factors on gaining control over glycosylated haemoglobin.
2.4 Management of Type 2 diabetes mellitus

The management of type 2 diabetes focuses on lifestyle interventions, lowering other cardiovascular risk factors, and maintaining blood glucose levels in the normal range. Managing other cardiovascular risk factors including: hypertension, high cholesterol, and microalbuminuria, improves a person's life expectancy (Ripsin et al, 2009). A proper diet and exercise are the foundations of diabetic care (Vijan, 2010). Culturally appropriate education may help people with Type 2 diabetes control their blood sugar levels, for up to six months at least (Hawthorne et al, 2008).

Studies done to assess the health care services for diabetes in South Africa showed the following: short consultation time, resulting in little or no time for patient education, poorly trained or inadequately staffing levels or both, and lack of continuous education programs. Monitoring and evaluation of complications of diabetes are lacking, and even if treatment guidelines are available, they are hardly used and are not up to date. Health care systems in Sub-Saharan Africa vary widely; structured, organized diabetes health care clinics are few (Whiting et al 2003).

Oral hypoglycaemic agents (OHAs) are invariably required in addition to diet and lifestyle intervention to achieve optimal glycaemic control. Thiazolidinediones and prandial glucose regulators are available, together with sulphonylureas, biguanides and α-glucosidase inhibitors (Melanie et al, 2006). Metformin, the only available biguanide is generally recommended as a first line treatment as there is some evidence that it decreases mortality (Nathan et al., 2009; Ripsin et al, 2009).
It acts by decreasing hepatic glucose output and peripheral insulin resistance (Bailey & Turner, 1996). The advantages of metformin are a very low risk of hypoglycaemia, weight neutrality and reduced risk of cardiovascular morbidity and mortality (Holman et al., 2008).

A second oral agent of another class of OHAs; sulphonylureas which increases insulin secretion may be used if metformin is not sufficient (Qaseem et al, 2012)

Many patients with type 2 diabetes require insulin therapy. In the UKPDS, more than 50% of patients required additional insulin therapy by 6 years; this was largely attributed to the fact that β-cell function worsened from about 53% at diagnosis to about 28% after 6 years of follow-up (UKPDS, 1995), but Shoback, (2010) argued that most people do not initially need insulin. Insulin therapy, alone or in combination with oral medication, appears to be a major predictor of insufficient glycaemic control (Standl et al, 2005).

During an International Diabetes Federation survey in 2003, it was observed that 80% of the people with diabetes were unable to obtain insulin and insulin syringes because they could not afford them. The cost of insulin preparations was higher in Sub-Saharan Africa than elsewhere. Insulin and insulin syringes were accessible to only 11% of all people with diabetes in Africa. In addition, only 25% of people with diabetes monitored their blood glucose. Self-monitoring of blood glucose was rarely used, mainly because of the cost of testing supplies in 90 percent and the unavailability of testing supplies in 70 percent of the countries in Africa.

In Ghana, a study by Amoah et al (2001) revealed that, there is dearth of data on morbidity, mortality and disability caused by DM. There is also lack of trained personnel for the proper management of DM and its complications resulting in an uncoordinated care for diabetes cases.
CHAPTER THREE

3.0 METHODS

3.1 Study Area

The study was conducted at the Diabetes Clinic of the Atibie Government Hospital at Atibie in the Kwahu South District of the Eastern Region of Ghana. The District is located in the north-western part of the Eastern Region. It shares common boundaries with the Sekyere East District and Afram plains to the north, Asante-Akim North Municipal and Asante Akim South District to the West, Kwahu West and Atiwa Districts to the south, Fanteakwa District to the East.

Specifically, Kwahu South District lies between latitude $6^\circ 30'\ N$ and $7^\circ\ N$ and longitude $0^\circ 30'\ W$ and $1^\circ\ W$. It covers a total land area of 1,876 square kilometres, with a population of 216,307. The inhabitants are predominantly farmers.

There is one government hospital serving the whole district and beyond. The hospital runs some specialised clinics which include; asthmatic, HIV/AIDS, antenatal, post natal and diabetes mellitus clinics. The diabetic mellitus clinic is held once every week with a total patient attendance of about 200 patients and an average of 5 new patients per week. The clinic is headed by a physician diabetologist.
Figure 3: Map of Eastern Region showing study area
3.2 Study Design

We screened patients with type two diabetes mellitus to determine the proportion of poor glycaemic control and then used an unmatched case control to identify the risk factors associated with glycaemic control involving type 2 DM patients attending diabetic clinic at the Government hospital at Atibie. The study was carried out from May to June 2012.

3.3 Independent Variables

**Socio-Demographics** included the following; Age, sex, marital status, educational level, level of income, mode of payment of medical bills, ethnicity, and place of residence.

**Behavioural variables** include the following; Alcohol intake, cigarette smoking or the use of other tobacco products, and hospital attendance.

**Health information** is made up of the following; Family history of DM, family support, duration of disease, type of treatment (diet or on medication), herbal medication, co morbidity (hypertension, chronic liver disease, coronary heart disease)

**Physical Measurements** include; Body Mass Index (derived from Height and weight) and Blood Pressure.

**Biochemical Measurement**: Lipid profile.

3.4 Dependent Variable

Glycated haemoglobin.
3.5 Study Population

The study population was persons diagnosed as having type 2 diabetes mellitus for at least two years attending the diabetic clinic at the Atibie Government hospital.

3.5.1 Inclusion Criteria

For the purpose of this research, participants were selected based on the following criteria for both case and control patients:

i. Patients with type 2 diabetes

ii. At least two years after diagnosis,

iii. Patients attending the diabetic clinic for at least six months

iv. Patients with overnight fasting state.

3.5.2 Exclusion Criteria

Patients with missing lipid profile results or who had not done lipid profile test for the past one year were excluded from the study.

3.5.3 Definition of cases, controls and factors

A case patient: including the criteria stated above had a glycated haemoglobin >6.5% (poor/ uncontrolled glycaemia) or two consecutive fasting blood glucose >7.0mmol/l.

A control patient: had a glycated haemoglobin <6.5% (good/ controlled glycaemia) or two consecutive fasting blood glucose <7.0mmol/l (WHO/IDF, 2006).
3.6 Definition of terms

**Herbal medication:** The use of any herbal preparation for any type of medical condition irrespective of the number, quantity, or duration.

**Co-morbidity:** Hypertension; patients with arterial hypertension of systolic blood pressure (SBP) ≥ 140mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg (WHO/ISH, 1999) or undergoing treatment with antihypertensive drugs. Other diseases of concern includes; cardiac disease, chronic liver disease and renal failure.

**Dyslipidaemia:** The levels of TC ≥ 200 mg/dL (HDL-C < 40 mg/dL (<1.034 mmol/L) for men or < 50 mg/dL (< 1.29 mmol/L) for women and triglycerides ≥150 mg/dL (≥1.7 mmol/dL) was defined as hypercholesterolemia, low HDL-C, and hypertriglyceridemia, (Alberti et al, 2005).

**Smoking status:** Currently consuming tobacco or its products irrespective of the quantity and duration.

**Alcohol consumption:** The use of any type of alcoholic beverage.

**Body mass index (BMI):** BMI defined by weight in kg, divided by height in meters squared, participants were classified as underweight (<18.5 kg/m$^2$), normal weight (18.5-24.9 kg/m$^2$), overweight (25-29.9 kg/m$^2$) and obese (≥30 kg/m$^2$) based on WHO classification (WHO, 2006).

**Waist circumference:** This was classified as normal (<94 cm), marginally increased (94-101 cm) and abdominal obesity (≥102 cm) for men and normal (<80 cm), marginally increased (80-87 cm) and abdominal obesity (≥88 cm) for women (WHO, 1995).

**Rural residence:** is a population with less than 5000 inhabitants (Waugh, 2009).
**Urban residence**: is a settlement that has a population of at least 5000 inhabitants (Waugh, 2009).

**Low education**: no formal education or had some formal education up to the Junior High level.

**High education**: had education to the Senior High level or any form of education.

**Family history of DM**: Any person who have blood relation (family member) with the participant and had a medical history of DM.

**Family support**: Any type of help from family members given to the patient in relation to the disease.

### 3.7 Data collection Techniques and Tools

#### 3.7.1 Training of research assistance

Research assistants were trained for two days before the pre-testing. Two of the research assistants were senior nursing officers who were drawn from the diabetic clinic of the Atibie Government Hospital. They were oriented on the correct interpretation and administering of the questionnaire, and on good communication skills.

#### 3.7.2 Pre-testing

Questionnaires were pre-tested in the diabetic clinic of the Brong Ahafo Regional hospital, Sunyani. The outcome was used to sharpen the data collection tools before data collection begun.
3.7.3 Sampling procedure

3.7.3.1 Sampling size determination;

The sample size was calculated using OpenEpi Version 3.03.17 calculation;

Two-sided confidence level (1-alpha): 95%

Power (% chance of detecting): 80%

Ratio of Controls to Cases: 1

Hypothetical proportion of controls with exposure: 40%

Least extreme Odds Ratio to be detected: 2.00

Using 60% of poor glycaemic control (Titty, 2010); sample size of 188 was calculated and we approximated to 200; 100 cases and 100 controls for the determination of the risk factors that affect glycaemic control.

3.7.4 Selection of participants

On diabetic clinic days, clients who consented to being part of the study were interviewed with a structured interviewer-administered questionnaire after the purpose of the research was explained to them and the consent forms signed or thumb printed. The questionnaire assessed factors that could affect the glycaemic control and that included socio-demographic characteristics; age, sex, income level, educational level, type of occupation, type of residence, and the mode of payment of hospital bills. The clinical histories assessed the; family history of DM, family knowledge and support, duration of disease, smoking habit and the use of any tobacco product at the time of the study in the past, alcohol intake, the use herbal preparations for any condition.
Participants’ folders were reviewed to know the type and duration of treatment, to identify the existence of co-morbidities, and to record the results of their lipids profile done in the last one year.

For physical measurement; blood pressure, weight and height measurement were taken and BMI calculated. Even though patients are required to attend the diabetic clinic in a fasting state, patients were screened and fasting persons were sent to the laboratory where blood samples were taken to determine the FBS. Blood samples for the Glycated haemoglobin were put in EDTA tubes in a cold chain and immediately transported to the Eastern Regional hospital Koforidua for the laboratory analysis.

We found 136 patients with uncontrolled glycaemia (HbA1c >6.5) and a total number of 246 patients had controlled glycaemia (HbA1c <6.5). After excluding patients with missing lipid profile, we had a total of 120 and 200 patients with uncontrolled and controlled glycaemia respectively. A simple random sampling was done to select 100 cases and 100 controls.

3.7.5 Physical Measurements

3.7.5.1 Clinical BP Measurements

Clinical BP was measured (Systolic and diastolic BP values, Korotkoff phase I and phase V, respectively) on the right arm following the British Hypertension Society guidelines (Williams et al, 2004) in a quiet environment with a calibrated mercury sphygmomanometer with the patient in a sitting position after 5 min of rest. Two different BP readings were measured at 5-min intervals and the mean calculated. In all patients, sphygmomanometric measurements were obtained by the same medical doctor (Perloff D et al, 1993).
3.7.5.2 Weight measurement

Participants wearing light cloths without shoes stood on a level placed measuring scale with no support. He or she stood still with the body weight evenly distributed between both feet in centre of the weighting scale. Salter 200 Academy Professional Mechanical scale was used, the scale was check to make sure it is set at zero before each participant was weighted. Weight was recorded to the nearest 100 g (WHO, 1995).

3.7.5.3 Height measurement

A vertical board with an attached metric rule and a horizontal mobile headboard that could be brought into contact with the uppermost point on the head was used. The individuals to be measured were barefooted or in thin socks and wore light clothing so that the positioning of the body could be seen. He or she stood on a flat surface, with weight distributed evenly on both feet, heels together, and the head positioned so that the line of vision is perpendicular to the body. The arms hung freely by the sides, and the head, back, buttocks, and heels were in contact with the vertical board. Those who could not stand straight in this position were positioned vertically so that only the buttocks and the heels or the head were in contact with the vertical board. The individual was asked to inhale deeply and maintain a fully erect position. The mobile headboard was brought onto the topmost point on the head with sufficient pressure to compress the hair. For consistency with methods used to collect the recommended reference data, no additional upward pressure was exerted on the mastoid processes. The height was recorded to the nearest 0.1 cm (WHO, 1995). Then the BMI was calculated using the formula; the weight in kilogram divided by the square of the height in meters. (Weight (kg)/Height (m$^2$)).
3.7.5.4 Waist circumference measurement

The participant stood comfortably with his or her weight evenly distributed on both feet, and the feet about 25-30 cm apart. The measurement was taken midway between the inferior margin of the last rib and the crest of the ilium, in a horizontal plane. Each landmark was palpated and marked, and the midpoint determined with a tape measure and marked. The observer sat by the side of the participant and fit the tape snugly but not so tightly as to compress underlying soft tissues. The circumference was then measured to the nearest 0.1 cm at the end of normal expiration (WHO, 1995).

3.7.6 Blood Sampling and Analysis

After a fasting period of 8 to 14 hours or overnight, peripheral venous blood samples were collected for determination of FPG, 2-hour PG and HbA1c. The samples were collected in evacuated EDTA-coated specimens for HbA1c and in evacuated Jodactetat coated specimens for FPG, (Otieno C.F. et al, 2005).

3.7.6.1 Laboratory procedures

Measurement of Haemoglobin A1c (HbA1c) in whole blood

Principle: The HbA1c was determined using an antigen-antibody reaction between HbA1c in whole blood adsorbed on latex particles and mouse anti-human HbA1c. Total haemoglobin and HbA1c have the same unspecific absorption rate to latex particles. The reagent one (R1) contains the latex particles. When mouse anti-human HbA1c monoclonal antibody is added (R2), latex-HbA1c-mouse anti-human HbA1c antibody complex is formed. Agglutination is formed, when goat anti-mouse IgG polyclonal antibody interacts with the monoclonal antibody. The amount of agglutination measured is proportional to the amount of HbA1c.
adsorbed on the surface of the latex particles. The amount of agglutination is measured as absorbance at 660nm. The HbA1c value is obtained from a calibration curve.

**Method:** About 1ml of venous blood was obtained from subjects into Ethylene diamine tetracetic acid (EDTA) tubes. Samples that could not be processed immediately were stored at (2-8)°c overnight. Exactly 20µL of the whole blood was added to 1ml of haemolysate reagent. The haemolysate was then used for the analysis using the BT 3000 Plus Chemistry analyzer.

- **Reagents**
  
  Reagent one (R1) - Contains 0.13% latex particles and 20mmol/L glycine buffer
  
  Reagent two (R2) - Contains mouse anti-human HbA1c monoclonal antibody, Goat anti-mouse
  
  Polyclonal antibody. Haemolysate Reagent: Water and stabilizers

**Measurement of fasting blood glucose**

Five (ml) of whole blood was taken from each consenting participant into a Becton Dickson (BD) fluoride test-tube. The test-tube containing the blood was well mixed on a blood mixing roller for 5minutes. The samples were then spun in a centrifuge at 3000g for 5minutes.

3ml of each plasma sample was aliquot in to a clean plain test-tube and then place in the probe of the Biotecnica (BT) Chemistry Analyser. The measurement of the glucose was done at a wavelength of 512nm.

**Principle of Glucose Estimation (Glucose Oxidase – Peroxidase method)**

Glucose oxidase converts Glucose to hydrogen peroxide and Gluconic Acid.
The hydrogen peroxide in the presence of peroxidase enzyme is broken down to release oxygen which reacts with 4-aminophenazone and phenol to give a pink Quinoneimine which is measured spectrophotometrically at 512nm. The amount of glucose in the blood is directly proportional to the intensity of the pink colour produced.

3.8 Data management and analysis

All hard copy data were stored in a safety cabinet, whilst soft copy data was stored with codes in *Epi info version 3.5.1* and SPSS version 16. Data were analysed using *Epi info version 3.5.1* and SPSS version 16 to obtain descriptive statistics. Categorical variables were compared using Chi square test whilst student t-test was used for quantitative variables. Multivariate analysis (Odds Ratio and logistic regression) was used to determine the odds of poor and good glycaemic control and the association between the glycaemic control and age, income level and diastolic blood pressure.

3.9 Quality control measures

Each data was checked for consistency and completeness. Universal safety measures were employed in the collection and processing of the blood sample in the laboratory. There was regular supervision to check on the quality of completed data. Standardise measuring scale and sphygmomanometer were used.

The pre-testing was done by the principal investigator and the research assistants in Sunyani which is outside the catchment area of the research area.
3.10 Ethical considerations

Approval was sought from the Ghana Health Service Ethical Committee and permission from the hospital authorities. Participants were fully informed about the purpose, procedures, risks, and benefits of participating in the study. Those who agreed to participate signed or thumb printed the inform consent forms. Data collected were used for the intended purpose only, kept confidential, and stored securely in a lockable cabinet.
CHAPTER FOUR

4.0 RESULTS

4.1 General Overview of Results

Over the period of four clinic days, except patient with type 1 diabetes, gestational diabetes and newly diagnosed type 2 diabetes, all patients attending diabetic clinic at Atibie Government Hospital had their blood sample obtained for the determination of their glycated haemoglobin (HbA1c) in May 2012. Out of 382 patients, 136 patients with uncontrolled glycaemia (HbA1c >6.5) and a total number of 246 patients had controlled glycaemia (HbA1c <6.5). After excluding patients with missing lipid profile, we had a total of 120 and 200 patients with uncontrolled and controlled glycaemia respectively. A simple random sampling was done to select 100 cases and 100 controls.

Table 1 shows the number of patients with poor glycaemic control per week in the month of May 2012. The first diabetic clinic day of the month had a total 36 poor glycaemia, rising in the second week to 57, and then decreased to 30 in third week. There was a further decrease in the fourth week to 13. The total number of patients screened did show a different trend. The first week had 110 attendances which decreased to 84, then increased again in the third week to 102 and then decreased again in the four week to 86.
Table 1: Distribution of Poor Glycaemic control (HbA1c >6.5%) per week, May 2012.

<table>
<thead>
<tr>
<th>Week</th>
<th>*Number of patients screened</th>
<th>Number with HbA1c &gt; 6.5% (%)</th>
<th>Number with HbA1c &lt; 6.5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Week</td>
<td>110</td>
<td>36 (33%)</td>
<td>74</td>
</tr>
<tr>
<td>2nd Week</td>
<td>84</td>
<td>57 (68%)</td>
<td>27</td>
</tr>
<tr>
<td>3rd Week</td>
<td>102</td>
<td>30 (29%)</td>
<td>72</td>
</tr>
<tr>
<td>4th Week</td>
<td>86</td>
<td>13 (15%)</td>
<td>73</td>
</tr>
<tr>
<td>Total</td>
<td>382</td>
<td>136 (36%)</td>
<td>246</td>
</tr>
</tbody>
</table>

*: Number excluded type 1, gestational and the newly diagnosed diabetes

4.2 General characteristics of cases and controls

The general distribution of cases and controls is shown in table 2. Females formed the majority of respondents for both cases and controls, cases were 73% females, and 80% for controls. For both cases and controls, majority of them 68 (34%) were in the 65+ age group. Eight percent of the cases were less than 45 years old. Majority of the case 38% were in the 55-64 age group, as compared to the controls which had majority of patients in the greater than 64 age group.

Majority of respondents earned less than Gh₵ 250 per month. Most of the patients both cases and control mode of payment of their medical bills is by the National Health Insurance Scheme (NHIS). Employers paid for 4 percent of cases and 5 percent controls patients, whilst patients themselves formed 3 percent of cases and 1 percent of controls.
Table 2: General characteristics of study participants (n=200)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (%)</th>
<th>Controls (%)</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex Distribution</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>27</td>
<td>20</td>
<td>47(23.5)</td>
</tr>
<tr>
<td>Female</td>
<td>73</td>
<td>80</td>
<td>153(76.5)</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>200(100)</td>
</tr>
<tr>
<td><strong>Age in years</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 45</td>
<td>8</td>
<td>13</td>
<td>21 (10.5)</td>
</tr>
<tr>
<td>45-54</td>
<td>28</td>
<td>23</td>
<td>51 (25.5)</td>
</tr>
<tr>
<td>55-64</td>
<td>38</td>
<td>22</td>
<td>60 (30.0)</td>
</tr>
<tr>
<td>&gt; 64</td>
<td>26</td>
<td>42</td>
<td>68 (34.0)</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>200 (100)</td>
</tr>
<tr>
<td><strong>Level of income</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; GH ¢ 250</td>
<td>56</td>
<td>89</td>
<td>145 (72.5)</td>
</tr>
<tr>
<td>GH ¢ 250 - GH ¢ 500</td>
<td>33</td>
<td>10</td>
<td>43 (21.5)</td>
</tr>
<tr>
<td>&gt; GHc 500</td>
<td>11</td>
<td>1</td>
<td>12 (6)</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>200</td>
</tr>
<tr>
<td><strong>Mode of Payment of Medical Bills</strong></td>
<td></td>
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<td></td>
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<tr>
<td>NHIS</td>
<td>93</td>
<td>94</td>
<td>187 (93.5)</td>
</tr>
<tr>
<td>Employer</td>
<td>4</td>
<td>5</td>
<td>9 (4.5)</td>
</tr>
<tr>
<td>Self</td>
<td>3</td>
<td>1</td>
<td>4 (2)</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>200</td>
</tr>
<tr>
<td><strong>Place of Residence</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>73</td>
<td>84</td>
<td>157(78.5)</td>
</tr>
<tr>
<td>Rural</td>
<td>27</td>
<td>16</td>
<td>43 (21.5)</td>
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<tr>
<td>Total</td>
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<td>100</td>
<td>200</td>
</tr>
<tr>
<td><strong>Employment status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>20</td>
<td>17</td>
<td>37 (18.5)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>64</td>
<td>57</td>
<td>121 (60.5)</td>
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<tr>
<td>Retired</td>
<td>16</td>
<td>26</td>
<td>42 (21)</td>
</tr>
</tbody>
</table>

Most of the patients both case and controls live in the urban settlement, 73% of cases, and 84% of controls as shown. A total of 121 (60.5%) of participants were unemployed, out of which 64 were cases and 57 were controls. 21% were retired, 18.5% were employed among which 20 (20%) were cases as against 17 (17%) controls.

4.3 Family history and family support for diabetes mellitus patients

Out of the 200 respondents, 96 (48%) has a family history of diabetes. These were 49 (51%) whose siblings, 35 (36.5%) parents, and 12 (12.5%) of cousins, aunties and uncles had diabetes. Some had no history of the disease and others were not aware of any history of diabetes. One hundred and forty eight (74%) had some form of support from their family members as against 34 (17%) who do not receive any family support.

4.4 Type of antidiabetes therapy

Table 3 is showing the type of therapy or the type of medication commonly prescribed. Most of the respondents (83.5%) were on combination therapy. There was no difference between the groups. Gliclazide & Metformin was the most used. Respondents on Insulin and Oral medication were the next most frequently prescribed combination therapy.

The remaining 33 (16.5%) respondents on monotherapy were on metformin 18 (9%) followed by gliclazide 7 (3.5%), glibenclamide 5 (2.5%), and insulin 3 (1.5%).
Table 3: Distribution of the types of antidiabetic therapy in the study population

<table>
<thead>
<tr>
<th>Type of therapy</th>
<th>Cases</th>
<th>Controls</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monotherapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glibenclamide</td>
<td>2</td>
<td>3</td>
<td>5 (2.5)</td>
</tr>
<tr>
<td>Gliclazide only</td>
<td>2</td>
<td>5</td>
<td>7 (3.5)</td>
</tr>
<tr>
<td>Metformin only</td>
<td>9</td>
<td>9</td>
<td>18 (9)</td>
</tr>
<tr>
<td>Insulin only</td>
<td>0</td>
<td>3</td>
<td>3 (1.5)</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>20</td>
<td>33 (16.5)</td>
</tr>
<tr>
<td>Combination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gliclazide &amp; Metformin</td>
<td>36</td>
<td>50</td>
<td>86 (43)</td>
</tr>
<tr>
<td>Glibenclamide &amp; Metformin</td>
<td>19</td>
<td>19</td>
<td>38 (19)</td>
</tr>
<tr>
<td>Insulin with Oral medication</td>
<td>32</td>
<td>11</td>
<td>43 (21.5)</td>
</tr>
<tr>
<td>Total</td>
<td>87</td>
<td>80</td>
<td>167 (83.5)</td>
</tr>
</tbody>
</table>

4.5 Comorbidities

Figure 4 below shows the distribution of some comorbidity among the various age groups. Hypertension was the common comorbidity among the age groups, but it was highest among the older participants (86.2%) as compare to 6.2% for cardiac diseases. Hypertension had the same percentage as cardiac disease among participants who are less than 45 years old. Cardiac disease decreased as participants advanced in age. From 23.5% in <45 age group, it decreased to 6.2% in the >64 age group.
Figure 4: Comorbidity among type 2 diabetes mellitus patients

### 4.6 Univariate analysis of cases and controls

#### 4.6.1 Risk Factor Analysis

As depicted by table 4 below, seventy four percent of cases were less than 65 years old, as compared to 58% of controls. Majority of both cases and controls were in the 45-64 age group; sixty six percent of the cases as against 45% controls. This age group showed to be significantly associated with poor glycaemic control (OR 2.17, 95% CI 1.17, 3.99, p=0.0013).

Twenty seven percent of the cases were males as compared to 20% of controls. The male sex appeared to be associated with poor glycaemic control. This was not significantly associated
(OR 1.48, 95% CI 0.765, 2.861, p=0.24). Seventy five percent of cases had low educational level and 25% had high education level. Among the controls, 82% had low educational level as compared to 18% who had high educational level. Educational level however was not significantly associated with glycaemic control (OR 0.85, 95% CI 0.440, 1.629, p= 0.62).

Majority of patients lived in the urban areas for both cases and controls. However, the place of residence where not significantly associated with poor glycaemic control (OR 1.94, 95% CI 0.971, 3.884, p=0.061).

Table 4: General characteristic as risk factor for glycaemic control.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (%)</th>
<th>Controls (%)</th>
<th>Odds ratio (OR)</th>
<th>Confidence interval (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age in years</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;45</td>
<td>6</td>
<td>12</td>
<td>0.759</td>
<td>0.254,2.267</td>
<td>0.662</td>
</tr>
<tr>
<td>45-64</td>
<td>67</td>
<td>47</td>
<td>2.165</td>
<td>1.173,3.994</td>
<td>0.013</td>
</tr>
<tr>
<td>≥65</td>
<td>27</td>
<td>41</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>27</td>
<td>20</td>
<td>1.479</td>
<td>0.765,2.861</td>
<td>0.244</td>
</tr>
<tr>
<td>Female</td>
<td>73</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>75</td>
<td>82</td>
<td>0.846</td>
<td>0.440,1.629</td>
<td>0.617</td>
</tr>
<tr>
<td>High</td>
<td>25</td>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Residence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>27</td>
<td>16</td>
<td>1.942</td>
<td>0.971,3.884</td>
<td>0.061</td>
</tr>
<tr>
<td>Urban</td>
<td>73</td>
<td>84</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From table 5 below shows, majority of participants had less than Gh₵ 250 as their monthly income. This was significantly associated with poor glycaemic control (OR 0.06, 95% CI 0.008, 0.505, p=0.009). Family support has a strong effect on glycaemic control. Insurance, disease duration and family history of DM showed no significant association.
Table 5: Risk factors for glycaemic control

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (%)</th>
<th>Controls (%)</th>
<th>Odds ratio (OR)</th>
<th>Confidence interval (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Income Gh₵&lt;250</strong></td>
<td>89</td>
<td>56</td>
<td>0.063</td>
<td>0.008,0.505</td>
<td>0.009</td>
</tr>
<tr>
<td>250-500</td>
<td>10</td>
<td>33</td>
<td>0.330</td>
<td>0.038,2.902</td>
<td>0.318</td>
</tr>
<tr>
<td>&gt;500</td>
<td>1</td>
<td>11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Insurance</strong> Yes</td>
<td>99</td>
<td>98</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family support</strong> Yes</td>
<td>74</td>
<td>74</td>
<td>3.50</td>
<td>1.10,1.13</td>
<td>0.034</td>
</tr>
<tr>
<td>No</td>
<td>22</td>
<td>12</td>
<td>6.40</td>
<td>1.70,23.9</td>
<td>0.006</td>
</tr>
<tr>
<td>N/A</td>
<td>4</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family history of DM</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>50</td>
<td>46</td>
<td>1.495</td>
<td>0.856,2.611</td>
<td>0.158</td>
</tr>
<tr>
<td>No</td>
<td>27</td>
<td>30</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Don’t know</td>
<td>23</td>
<td>24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Disease duration</strong> &lt;5</td>
<td>47</td>
<td>55</td>
<td>0.726</td>
<td>0.416,1.265</td>
<td>0.258</td>
</tr>
<tr>
<td>&gt;5</td>
<td>53</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Type of therapy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monotherapy</td>
<td>13</td>
<td>21</td>
<td>0.562</td>
<td>0.264,1.197</td>
<td>0.135</td>
</tr>
<tr>
<td>Combination</td>
<td>87</td>
<td>79</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Smoking</strong> Yes</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>99</td>
<td>99</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Alcohol intake</strong> Yes</td>
<td>2</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>98</td>
<td>99</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Eight seven percent of cases were on combination therapy as compared to 79% of controls. Type of therapy was not significantly associated with glycaemic control (OR 0.56, 95% CI 0.264, 1.197, p=0.14). Only one percent of patients had a history of smoking as compared to 1.5% patients who take in alcohol.
As table 6 depicts, 30 patients had high total cholesterol (\(\geq 5.2\) mmol/l). Total cholesterol (TC) statistically was not associated with glycaemic control (OR 1.5, 95% CI 0.97, 3.03 \(p=0.06\)). Forty eight cases had HDLP of less than 1.15mmol/l, as against 75 controls. HDLP was significantly associated with glycaemic control (OR 0.3, 95% CI0.17, 0.56, \(p<0.001\)). LDLP and TG were not significantly associated with glycaemic control as shown by their respective \(p\) values.

Among the 200 participants, 128 (64%) were obese with 62 cases and 66 controls. The type of BMI has no significant effect on glycaemia. About Sixty eighty percent of the patients had abdominal obesity, majority of them were cases (52%). Majority of patients who had normal waist circumference were among the control group. Systolic blood pressure of greater or equal to 140mmHg formed 54.5%. There was no much difference in terms of percentage among cases and controls. Diastolic blood pressure showed some significant effect on glycaemic control (OR 1.89, 95% CI 1.054, 3.396 \(p=0.033\))
Table 6: Comorbidities as risk factors for glycaemic control

<table>
<thead>
<tr>
<th>Comorbidities/ Variables</th>
<th>Cases (%)</th>
<th>Controls (%)</th>
<th>Odds ratio (OR)</th>
<th>Confidence interval (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;5.2</td>
<td>35</td>
<td>44</td>
<td>1.50</td>
<td>0.97,3.03</td>
<td>0.063</td>
</tr>
<tr>
<td>≥5.2</td>
<td>65</td>
<td>56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDLP (mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1.15</td>
<td>48</td>
<td>75</td>
<td>0.31</td>
<td>0.17,0.56</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>≥1.15</td>
<td>52</td>
<td>25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDLP (mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;2.6</td>
<td>37</td>
<td>42</td>
<td>1.23</td>
<td>0.70,2.18</td>
<td>0.467</td>
</tr>
<tr>
<td>≥2.6</td>
<td>63</td>
<td>58</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG (mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1.7</td>
<td>66</td>
<td>67</td>
<td>1.05</td>
<td>0.58,1.88</td>
<td>0.881</td>
</tr>
<tr>
<td>≥1.7</td>
<td>34</td>
<td>33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal (&lt;24.9)</td>
<td>38</td>
<td>34</td>
<td>1.044</td>
<td>0.587,1.857</td>
<td>0.883</td>
</tr>
<tr>
<td>Obesity (≥25)</td>
<td>62</td>
<td>66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WC (cm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>29</td>
<td>34</td>
<td>0.474</td>
<td>0.235,0.474</td>
<td>0.037*</td>
</tr>
<tr>
<td>Abd. obesity</td>
<td>71</td>
<td>66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;140</td>
<td>45</td>
<td>46</td>
<td>0.928</td>
<td>0.527,1.634</td>
<td>0.796</td>
</tr>
<tr>
<td>≥140</td>
<td>55</td>
<td>54</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;90</td>
<td>53</td>
<td>70</td>
<td>1.89</td>
<td>1.054,3.396</td>
<td>0.033*</td>
</tr>
<tr>
<td>≥90</td>
<td>47</td>
<td>30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** TG- triglycerides; BMI –Body Mass Index; TC- Total Cholesterol; HDLP- High Density lipoprotein; LDLP- Low Density Lipoprotein; WC- Waist Circumference;
SBP- Systolic Blood Pressure; DBP- Diastolic Blood Pressure; Abd. Obesity- Abdominal Obesity.

4.7 Multivariate analysis

After the Univariate analysis, a multivariate analysis was constructed as shown in table 7 below for the risk factors that showed statistical significant association with glycaemic control. These variables were; age of patient, income levels, DBP, HDLP-cholesterol, family support and WC. Out of these six variables, HDLP and income levels were found to be significant (p values, 0.015, and 0.011 respectively).

Table 7. Multivariate analysis of significant variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>S.E</th>
<th>OR</th>
<th>CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>0.517</td>
<td>0.337</td>
<td>1.677</td>
<td>0.866,3.247</td>
<td>0.125</td>
</tr>
<tr>
<td>INCOME</td>
<td>-2.698</td>
<td>1.067</td>
<td>0.067</td>
<td>0.008,0.545</td>
<td>0.011*</td>
</tr>
<tr>
<td>FAM. SUPT</td>
<td>0.38</td>
<td>1.1</td>
<td>1.5</td>
<td>0.17,12.7</td>
<td>0.729</td>
</tr>
<tr>
<td>DBP</td>
<td>0.355</td>
<td>0.480</td>
<td>1.426</td>
<td>0.556,3.656</td>
<td>0.460</td>
</tr>
<tr>
<td>HDLP</td>
<td>-1.227</td>
<td>1.5</td>
<td>0.293</td>
<td>0.109,0.787</td>
<td>0.015*</td>
</tr>
<tr>
<td>WC</td>
<td>-1.320</td>
<td>0.717</td>
<td>0.267</td>
<td>0.065,1.089</td>
<td>0.066</td>
</tr>
</tbody>
</table>

*Variables showing significant p values. FAM. SUPT; family support.
CHAPTER FIVE

5.0 DISCUSSION

This present study was undertaken to determine the possible factors affecting the glycaemic control among type 2 diabetes mellitus and the proportion of poor of glycaemic control. The excluded were the newly diagnosed, type 1, and gestational diabetes mellitus patients.

5.1 Proportion of poor glycaemic control

The proportion of poor glycaemic control among type 2 diabetes mellitus was 36%, this finding was different from a study by Titty (2010) done in Tamale Teaching hospital, with a sample size of 240, reported the prevalence of glycaemic control to be 60%. The Titty study concentrated on newly diagnosed patients over a period of one year. Similarly, other studies showed higher prevalence, Longo-Mbenza et al, (2008) and Noor et al, 2012 with prevalence of 68% and 89.5% respectively. The reason for the low proportion of poor glycaemic control in Atibie could be due to the regular clinic day counselling, and the fact that the hospital has diabetic association whose members meet once very month outside the clinic days, where both open discussion for all members concerning every aspect of their lives are done, and a close door consultation which is handled by the doctors and nurse for patients who need special assistance. This study focused on known diabetic patients who are regular at the attending the diabetic clinic and the study period were over few weeks.
5.2 Socio-demographic characteristics and poor glycaemic control

Being 76.5% of the respondents, females formed the majority. This percentage is similar to the 69% Benoit et al, (2005) found in the Project Dulce. In the study by Farmer et al (2011) males were the majority of respondents.

There was no difference between males and females for poor glycaemic control in this study at Atibie hospital. In the study by Longo-Mbenza et al (2008), the females were however more likely to have better glycaemic control.

Older respondents had poorer glycaemic control, these findings is different from other studies; Nichols et al (2000) and Benoit et al, (2012) found poorer metabolic control among the younger age group. The older respondents in our environment need more attention to be able to achieve and maintain good glycaemic control.

The relation between age and glycaemic control varies in different studies. As found by Longo-Mbenza et al (2008), there was no significant association between age and poor glycaemic control, but Farmer et al (2012) found a strong association with increasing age (CI -0.58,-0.18, p< 0.001).

As income level increases, the risk of having a poor glycaemic control reduces. Therefore income level was significantly associated with poor glycaemic control. Harris et al (1999) found no association between poor glycaemic control and financial status. Similarly, studies by Blaum et al (1997) and Haffner et al (1989) did not find an association between glycaemic control and financial status. Our study however, is different in many ways. It was done in sub-Saharan region with a study population which have a low socioeconomic status, and majority of patients have no or low educational level.
The effect of insurance status of respondents on poor glycaemic control collaborates previous study. Harris et al (1989) did not find any association between poor glycaemic control and insurance status. A total of 61.4% of participants with low income level had poor glycaemic control, even though more than 93% of participants’ hospital bills were paid by the NHIS, some of the prescriptions and HbA1c test were not covered by the NHIS, patients may have difficulty in procuring such medications and may also be reluctant in going to check for their HbA1c level. Majority of respondents were living in an urban area, however, there was no difference between rural and urban dwellers for poor glycaemic control. Family support has a very strong effect on how a patient’s diabetes controlled, whereas patients with some form of family support are about three times more likely to have a good glycaemic control, patients with no family support are six times likely to have poor glycaemic control as compare to patients with support. Most family supports may come in the form of encouragement, financial support, monitoring patients’ diet and making sure that they are taking their medications.

5.3 Hypertension and poor glycaemic control

There have been several studies on the prevalence of hypertension in type 2 diabetes both in developed and developing countries. Studies that concentrated in the sub-Sahara region include; Longo-Mbenza et al (2008) which found the prevalence of hypertension to be 73.3% in Democratic Republic of Congo, 70.1% in Cameroon by Choukem et al (2007), and about 50% in Kenya by Otieno et al (2005). This current study found the prevalence of hypertension among type 2 diabetic patients as 60.5%, majority was among the cases.
5.4 BMI, waist circumference and glycaemic control

BMI had no significant association with glycaemic control. Similarly study by Noor et al (2012) found that correlation between BMI and HbA1c was negative, weak and not significant.

Majority of patients had abdominal obesity; but there was no much difference among cases and controls. Even though intra-abdominal adiposity plays an important development of metabolic syndrome and increased central obesity may offset a controlled glycaemia, this study found no significant association between abdominal obesity and poor glycaemic control.

Several studies had different findings; Noor et al (2012) found an increasing central obesity using Waist circumference was significantly associated with uncontrolled HbA1c. (Yoshida et al, 2001, Okosun et al, 2002) argued that physiologic factors are important in diabetes control where they have showed the association between glycaemic control and waist circumference.

5.5 Biochemical measurement and glycaemic control

There were no significant association between high total cholesterol, LDLP, TG and poor glycaemic control. HDLP was significantly associated with glycaemic control. These findings contradicted that of Longo-Mbenza et al (2008) in Kinshasa, DRC, except for TG which was not significant, and low HDLP which was paradoxically, significantly associated with lower HbA1c. Benoit et al (2005) also reported that, total cholesterol was associated with glycaemic control. Since high total cholesterol has high risk effect on the development of cardiovascular diseases, it requires regular screening for effective management.
5.6 Management of Type 2 diabetes mellitus

The findings from this study found the proportion of individuals with short duration of disease were more than half of the study population, majority of whom had good glycaemic control. For majority of respondents who had a long disease duration respondents glycaemic control was poor. This shows more support should be given to this category of patients. They need support to achieve and maintain adequate glycaemic control.

For this study, the disease duration was however not significantly associated with glycaemic control. Other studies however found duration of disease to significantly affect glycaemic control (Benoit et al 2005, Longo-Mbenza et al 2008).

The result of this study reveals that, more than 80% of the respondents are on combination therapy and greater proportion of respondents on combination therapy did not attain a good glycaemic control. Patients receiving a combination therapy made up of insulin and oral medication were least likely to attain good glycaemic control, whilst more than half of patients on Gliclazide and metformin are more likely to attain good glycaemic control. Weight gain during treatment with insulin and other agents prevent the achievement of good glycaemic targets and probably limit the success of treatment. Insulin-related weight gain has been variously attributed to the anabolic effects of high-dose insulin, increase appetite, and the reduction of glycosuria with a resultant retention of calories.

Although there exist different type of treatment and differences in the proportion of individual patients attaining a good glycaemic control, type of treatment was not significantly associated glycaemic control.
5.7 Study limitations

Aspects of the questionnaire depended on the memory of participants which may have resulted in recall bias and cannot be validated by objective measurements.

This study focused on type 2 diabetic patients who have had the disease more than one year, hence does not apply to patients with newly diagnosed and other types of diabetic.

Conclusion on the association between the some independent variables and poor glycaemic control may be limited since there is no information on which appeared first.

Because single standardized measuring instruments was used, validation could not be done.
CHAPTER SIX

6.0 CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusion

This study was conducted at the Atibie Government Hospital in Atibie, Kwahu South District in May 2012. It set out to determine the factors that affect glycaemic control among type 2 diabetes mellitus, by identifying the proportion of poor glycaemic control and the risk factors for poor glycaemic control.

The proportion of poor glycaemic control in the month of May 2012, in Atibie Government Hospital was 36%. Poor glycaemic control was more common among the males, but this was insignificant.

There was also no significant association between poor glycaemic control and family history of diabetes, and educational level, place of residence, systolic blood pressure, BMI, total cholesterol, LDLP, and triglycerides. Patients’ less than 45 years, abdominal obesity, and diastolic blood pressure were not associated with poor glycaemic control. People who were above 45 years, who had an income less than 250 was statistically significant factors that affected poor glycaemic control, therefore poverty plays a major role in controlling diabetes.

Psychological and biological factors, self-care skills, knowledge of disease and, diet, exercise, other comorbid diseases were not explained by this study. This study provides a useful assessment of the factors affecting diabetes management. It does not provide answers to why patients are not optimally controlled but does provide a starting point from which to investigate and address the obstacles that prevent patients with diabetes from reaching their metabolic targets.
6.2 Recommendations

TO THE DIABETIC CLINIC ATIBIE GOVERNMENT HOSPITAL

- The height of patients to be taken at first clinic attendance and recorded.

- The waist circumference should be taken regularly as it is done for weight measurement.

- Since patients with type 2 diabetes mellitus are already at high risk for cardiovascular diseases, regular lipid profile should be done at short intervals for all patients.

TO MINISTRY OF HEALTH AND GHANA HEALTH SERVICE

- There should be nationwide research to identify the factor affecting the poor glycaemic control in Ghana. This study can be used as starting point.

- Diabetic patients with low financial status should be identified and included in the national livelihood enhancement programme.

- HbA1c assay equipment and reagents should be provided to the district hospitals and the test should be covered by the NHIS so to make it available and affordable to facilitate the management of DM.
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APPENDIX I

Diabetes diagnostic criteria (WHO/IDF, 2006).

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<th>Condition</th>
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<th>Fasting glucose</th>
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<td></td>
<td>mmol/l(mg/dl)</td>
<td>mmol/l(mg/dl)</td>
<td>%</td>
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<td>Normal</td>
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<td>&lt;6.1(&lt;110)</td>
<td>&lt;6.0</td>
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<tr>
<td>Impaired fasting glycaemia</td>
<td>&lt;7.8(&lt;140)</td>
<td>≥6.1(≥140) &amp; &lt;7.0(&lt;126)</td>
<td>6.0-6.4</td>
</tr>
<tr>
<td>Impaired glucose tolerance</td>
<td>≥7.8(≥140)</td>
<td>&lt;7.0(&lt;126)</td>
<td>6.0-6.4</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>≥11.1(≥200)</td>
<td>≥7.0 (≥126)</td>
<td>≥6.5</td>
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APPENDIX II

Subject number: .................................................................

CONSENT TO PARTICIPATE IN A RESEARCH PROJECT

TITLE OF PROJECT: FACTORS THAT AFFECT GLYCAEMIC CONTROL AMONG TYPE 2 DIABETES MELLITUS PATIENTS IN KWAHU SOUTH DISTRICT, EASTERN REGION, GHANA.

Before agreeing to participate in this research study, it is important that you read the following explanation of this study or have it read to you. This statement describes the purpose, procedures, benefits, risks, discomforts, and precautions of the program. Also described are the alternative procedures available to you, as well as your right to withdraw from the study at any time.

Explanation of Procedures

You are being invited to participate in a research project; factors affecting the control of diabetes among type 2 diabetes mellitus patients in kwahu south District. The approach of the research is through the use of a questionnaire, physical measurements and blood specimen collection. You will complete the questionnaire that will require answers on personal profile and risk factors affecting diabetic control (i.e. duration of disease, smoking status.). Afterwards, physical examination will be done and blood specimen will be collected only once for each participant. The collection of data is expected to be completed in four weeks.

Risks and Discomforts
By participating in this research, you are likely to experience some form of discomfort or pain. This includes the discomfort of questioning, physical examination, and the pain of blood specimen collection. The team will try and decrease your chances of these risks from occurring, but if an untoward event happens, they will provide you with free medical care.

Benefits
There are no direct benefits by participating in this project. However, this research is expected to provide data on diabetic control for stakeholders on policy making.

Confidentiality
All information gathered from the study will remain confidential. Your identity as a participant will not be disclosed to any unauthorized persons; only the researchers, Ghana Health Service and School of Public Health will have access to the research materials, which will be kept in a locked drawer. Any references to your identity that would compromise your anonymity will be removed or disguised prior to the preparation of the research reports and publications.

Withdrawal from Project
Participation in this study is voluntary; refusal to participate will involve no penalty. You are free to withdraw consent and discontinue participation in this project at any time without prejudice from the research team.

Costs and/or Payments to Subject for Participation in Research
There will be no costs for participating in the research. Also, you will not be paid to participate in this research project.
Any questions concerning the research project and/or in the case of injury due to the project, participants can call Dr John Tengey of the School of Public Heath (0209214464) or Dr Francis Addai of Atibie Government Hospital (0243251273).

Questions regarding any rights issues as a person in this research project should be directed to the chairpersons of the Ethical Review Committees of the Ghana Health Service, Prof. Binka (0208131031)

**Consent to participate in Research**

I, ...............................................................................................................

☐ Confirm that I have read the written information (or have had the information read to me) for the study **Factors Affecting the Control of Diabetes Among Type 2 Diabetes Mellitus Patients in Kwahu South District, Eastern Region, Ghana** and that the study procedures have been explained to me by study staff during the consent process for this study.

☐ Confirm that I have had the opportunity to ask questions about this study and I am satisfied with the answers and explanations that have been provided.

☐ Understand that I grant access to data to authorised persons described in the information sheet.

☐ Have been given time and opportunity to consider taking part in this study.

Tick as appropriate (this decision will not affect your ability to enter the study):
I consent to participate in the above research study.

Signature of Subject: .................................
Date.............................

Signature of Interviewer: .................................
Date.............................

Name of
Impartial Witness: .................................

Signature of
Impartial Witness: .................................
Date.............................
APPENDIX III

Factors that Affect Glycaemic Control Among Type 2 Diabetes Mellitus Patients in Kwahu South District, Eastern Region, Ghana.

Subject No………….. Date……………..

Personal Profile

1. Name…………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………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15. If yes, do you receive any support from them? Yes ☐ No ☐

Medical History

16. How long have you been diagnosed with diabetes?

<table>
<thead>
<tr>
<th>1). 3-5yrs</th>
<th>2). 6-10yrs</th>
<th>3). 11-15yrs</th>
<th>4). 16-20yrs</th>
<th>5). &gt;20yrs</th>
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17. Do you currently use any form of diabetic medication? Yes ☐ No ☐

If yes which of these do you use? Gliclazide only ☐ Metformin only ☐
Glibenclamide only ☐ Insulin only ☐ Glibenclamide and Metformin ☐
Gliclazide and Metformin ☐ Insulin with oral medication ☐

Other specify.................................................................

18. If yes how long have you been using it? ..............................................

  Less than 3 months ☐ Between 3 months and 1 year ☐
  Between 1 year and 5 years ☐ More than 5 years ☐

19. Are you using any herbal medication?

  Yes ☐ No ☐

  If yes how long? ..............................................................

Co-morbidity

20. Do you have any of these diseases? Hypertension ☐ Cardiac Disease ☐
If others, state type ..............................................................................................................

When was these diseases diagnosed? ..................................................................................

Are you on any medication for any of these diseases?..........................................................

Duration of treatment....................

**Smoking status**

21. Do you smoke tobacco? Yes ☐ No ☐

    If yes, how long have you been smoking? ...............................................................

    How many cigarettes on average do you smoke per week? .......................

22. Have you smoked before? Yes ☐ No ☐

23. If yes how long did you smoke? .................................................................................

24. Do you use any other tobacco-related product? (snuff) Yes ☐ No ☐

    If yes, how long have you been using it? ...............................................................

    How many tobacco-related products on average do you smoke per week? .............

**Alcohol consumption**

25. Do you take alcohol? Yes ☐ No ☐

    If yes, how long have you been using it? ...............................................................

    How often do you take alcohol on average per week? .................................
# FACTORS THAT AFFECT GLYCAEMIC CONTROL AMONG TYPE 2 DIABETES MELLITUS PATIENTS IN KWAHU SOUTH DISTRICT, EASTERN REGION, GHANA

## CASE REPORT FORM

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