# A SURVEY OF COMMON COMPLICATIONS ASSOCIATED WITH DIABETES MELLITUS IN THE DIABETIC CLINIC AT THE UNIVERSITY TEACHING HOSPITAL, LUSAKA, ZAMBIA

THESIS
M. MED
BAN
2001
C.1

**Lewis Banda BSc.Hb (UNZA) MB.ChB (UNZA)**

**A dissertation submitted in partial fulfilment of the requirements for the award of Masters of Medicine (Internal Medicine) degree of the University of Zambia**

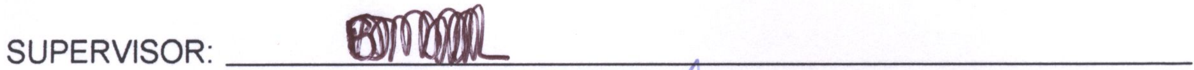
**University of Zambia**

**School of Medicine**

**September 2001**

# APPROVAL PAGE

The dissertation of Dr Lewis Banda is approved in partial fulfilment of the requirements for the award of the Masters Degree in Internal Medicine.



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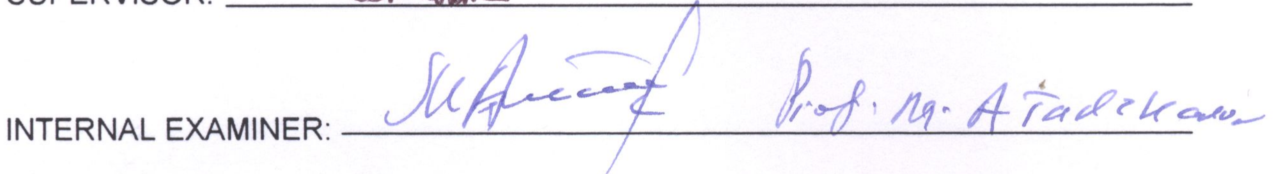

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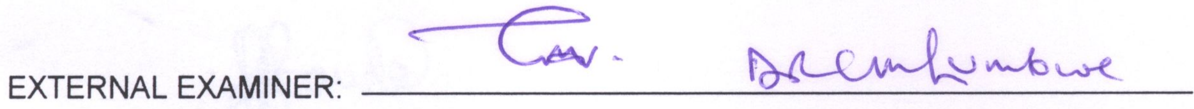

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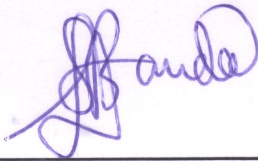

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# ABBREVIATIONS

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| UTH | - | University Teaching Hospital |
| IDDM (type 1) | - | Insulin Dependent Diabetic Mellitus |
| NIDDM (type 2) | - | Non-Insulin Dependent Diabetic Mellitus |
| UKPDS | - | United Kingdom prospective diabetic Study |
| DCCT | - | Diabetic Control and complications Trial |
| DR | - | Diabetic Retinopathy |
| DM | - | Diabetes Mellitus |
| BMI | - | Body Mass Index |
| HOT | - | Hypertension Optimal Treatment Trial |
| JVC VI | - | Joint National committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure |
| $HbA_{1C}$ | - | Glycosylated hemoglobin |
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| NKHS | - | Non-Ketotic Hyperosmolar syndrome |
| ACEI | - | Angiotensin converting enzyme inhibitor |
| CAD | - | Coronary artery disease |
| HDL | - | High density lipoprotein |
| LDL | - | Low density lipoprotein |
| VLDL | - | Very low density lipoprotein |
| AGEs | - | Advanced Glycosylated end products |
| WHO | - | World Health Organization |
| HIV | - | Human immunodeficiency virus |

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# ABSTRACT


**STUDY SETTING:**

The study was conducted at the University Teaching Hospital Lusaka Zambia in the diabetic clinic.

**OBJECTIVES:**

The study described common complications, their prevalence and factors associated with their development in diabetic mellitus patients.

**DESIGN AND METHODS:**

80 patients with diabetes mellitus were prospectively as well as retrospectively evaluated for complications over a period of 9 months. The evaluation included clinical and laboratory methods. The clinical methods entailed a detailed history, review of files and anthropometric measurements of weight and heights from which the body weight was expressed as body mass index (BMI). Ocular examination included the fundoscopy. A full physical examination was performed for possible diabetic complications. Laboratory methods included urinalysis, blood glucose, blood creatinine and other tests depending on the indication.

**RESULTS:**

This study has demonstrated that obesity is a major risk factor for the development of DM and that most of the patients attending the clinic are on diet and oral hypoglycaemic drugs. In this study only 31.3% had a family history of DM. Despite the regular attendance of the clinic by the majority of patients, complications were high and

included, hypoglycaemia (19.2%), DKA (9.2%), hypertension (74.4%), peripheral neuropathy (40%0), erectile dysfunction (58%), cataracts (38.8%), diabetic retinopathy (20%), diabetic foot complications (13.8%), strokes and clinical nephropathy (3.8%) each and 2.5% had coronary artery disease. 15% of the patient had tuberculosis.

**CONCLUSION:**

This survey revealed that the majority of the patients are middle aged obese non-insulin dependent diabetics (NIDDs) with a female preponderance.

Attendance and compliance is generally good in this study population but monitoring and control of diabetes is poor.

Complications are a common problem in this study population. TB is also a big problem among diabetic patients. Hyperlidemia and cardiovascular complications are still a small problem.

# CHAPTER 1

## 1.1 BACKGROUND INFORMATION AND REVIEW OF LITERATURE

Diabetes mellitus is a group of metabolic disorders characterised by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It is the commonest endocrine disorder and affects approximately 30 million people worldwide.

Diabetes mellitus is the commonest endocrine disorder seen at UTH. The incidence and prevalence of diabetes is not known in Zambia just like most African countries (1,2,3,). Worldwide, the prevalence varies from country to country. The criteria used in the diagnosis of diabetes could be the cause of these differences (4). In the western world it is said to be between 1-5%. In the United States, the prevalence is about 3.5% in the total population with type1 accounting for 10% and type 2 accounting for 90% (5). Prevalence of type 2 is age-dependent. Most cases of type1 diabetes are diagnosed before age 40, and most of type2 are diagnosed after age 40. The prevalence of type 2 diabetes rises dramatically in old age; the disease is found in 10.4% of Americans aged 65 or older.

Diabetes also shows ethnic preferences, with higher prevalence among African Americans, Mexican Americans, and native Americans as compared with non-Hispanic whites.
A better understanding of age and ethnic preferences of diabetes could be used to develop more effective public awareness programs for early recognition and diagnosis.

The pathophysiology of type 2 diabetes suggests a prolonged preclinical phase, including compensated insulin resistance with hyperinsulinemia, before overt hyperglycemia appears. Thus according to estimates based on oral glucose tolerance testing, half of all patients with type 2 diabetes are not diagnosed at present.

Worldwide the incidence and prevalence of diabetes is said to be on the increase. The increased prevalence in diabetes mellitus means that there will be an increase in the morbidity and mortality from diabetic complications.

Diabetes has acute metabolic emergencies and long-term complications.

The acute metabolic consequences include Diabetes Ketoacidosis, hyperosmolar non-ketotic syndrome and hypoglycaemia in those already on treatment. Poor compliance and insulin treatment are the main precipitating causes of acute decompensation. Glycation of tissue proteins and other macromolecules and excess production of polyol compounds from glucose are among the mechanisms thought to produce damage from chronic hyperglycaemia (43,44). The glycation of proteins and other macromolecules lead to the formation of the advanced glycosylated end products (AGES). In animal studies, lowering of AGE levels, even in the presence of poor glycaemic control, results in decreased incidence or the absence of retinopathy, nephropathy, neuropathy, and other macrovascular disease. These complications can be avoided by pre-treatment with aminoguanidine, which blocks formation of AGES (45). Studies of aminoguanidine use in patients with type 1 or 2 diabetes and nephropathy and in diabetic patients receiving dialysis are in progress (46). Improved patient education and better access to medical care may decrease development of hyperglycaemic and hypoglycaemic emergency (6).

The major complications encountered in diabetics include:

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SUPERVISOR: ____________________

INTERNAL EXAMINER: ____________________ Prof. Ng. A Tadikawa

EXTERNAL EXAMINER: ____________________ Dr Chembumbwe

CHAIRMAN, BOARD OF EXAMINERS: ____________________

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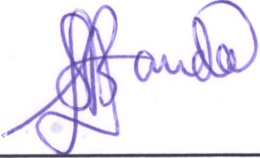

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| VLDL | - | Very low density lipoprotein |
| AGEs | - | Advanced Glycosylated end products |
| WHO | - | World Health Organization |
| HIV | - | Human immunodeficiency virus |

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The major complications encountered in diabetics include:

**1. Hypoglycaemia**

Hypoglycaemia is the major, most common, and most feared complication of insulin therapy. Although acute morbidity and mortality of severe hypoglycaemia are remarkably low compared with other causes of coma, there are worrying indications that recurrent severe hypoglycaemia can lead to progressive cognitive impairment (7)

**2. Diabetic Ketoacidosis (DKA)**

Diabetic ketoacidosis can occur when there is a complete lack of insulin, as with type 1 (insulin dependent) diabetes, or inadequate insulin levels, such as might occur during severe illness or stress in patient with either type I and type II (non-insulin dependent) diabetes (8). The incidence is 2 per 100 patient years with type 1 diabetes, and the disorder tends to be more common in younger than in older age-groups (9). Between 20% and 30% of cases occur in patients with newly diagnosed diabetes (10). In a patient with established type 1 diabetes and no precipitating illness or stress, ketoacidosis represents a failure of patient education (11).

**3. Non-ketotic hyperosmolar syndrome (NKHS)**

Non-ketotic hyperosmolar syndrome occurs predominantly in patients with type 2, in whom dehydration and severe hyperglycaemia may occur without development of ketoacidosis. NKHS may occur as sequel to severe stress and may follow stroke or an excessive carbohydrate intake. The pathogenesis of NKHS usually includes impaired renal excretion of glucose; thus, antecedent renal insufficiency or prerenal azotemia is common. Precipitating factors are similar to those for DKA (12).

Diabetes is usually irreversible and, although patient can have a reasonably normal life-style, its late complications results in reduced life expectancy and considerable uptake of health resources. Macrovascular disease leads to increased prevalence of coronary artery disease, peripheral vascular disease and strokes, while microvascular damage results in diabetic retinopathy and contributes to nephropathy (13).

The common long-term complications of diabetes in most series include the following:

**4. Hypertension**

Hypertension is common among patients with type 2 diabetes. This is associated with hyperinsulinaemia and obesity. Prevalence rates vary between 40% and 75% (14). Non diabetic populations that have blood pressure of 120/80mmHg or below have been found to have the lowest cardiovascular mortality rates, and intuitively this level might seem optimal of diabetic patients. However, this apparent logic is counter balanced by concerns that in elderly diabetic patients who may have critical vascular stenoses, mortality rates may increase if their hypertension is treated too vigorously (the J point hypothesis) (15).

Evidence favours the therapeutically adventurous group that overtreats, as their patients tend to have fewer cardiovascular complications. In the diabetic cohort of 1,501 patients in the Hot trial, the incidence of major cardiovascular events (stroke and heart attack) was decrease by 50% when diastolic blood pressure was lowered from 90 to 80mmHg (16). Similarly, in UKPDS 38 (17), macrovascular risk (myocardial infarction, stroke, and amputation) was reduced by 34% when an attained average blood pressure of 144/42mmHg with tight control was compared with that of 154/87mmHg with less tight control.

Both the American Diabetes Association and the JNC VI recommend a blood pressure of less than 130/85mmHg as a goal in diabetic patients, but their consensus antedate the three trials described. The optimal blood pressure in diabetic patients is still uncertain but is probably close to 120/80mmHg and that the position of the J point is defined individually by the patients tolerance or intolerance to anti-hypertensive medication (17).

Hypertension is implicated in accelerating the microangiopathy of diabetes, particularly the retinopathy and nephropathy. In a study at UTH, hypertension was found to be 26.9%(1).

**5. Retinopathy**

This has the potential to cause visual loss and seems to be related to the duration of diabetes, type of treatment and degree of control as was shown by the study in Nigeria (18).

Overall the prevalence of retinopathy in diabetic patients is about 25%. In NIDDs the prevalence of diabetes is 20% and in IDDs it is about 40% (19). In patients diagnosed as having diabetes prior to the age of 30, the incidence of DR is 50% after 10 years and 90% after 30 years. It is extremely rare for DR to develop within 5 years of the onset of diabetes, but about 5% of NIDDs have background DR at presentation.

Although good control will not prevent DR, it may retard its development by a few years. Conversely, poorly controlled patients may develop DR sooner than well-controlled patients (20). The growing conviction that complications of diabetes are linked to poor metabolic control has led to aggressive efforts to normalise metabolism by either continuous subcutaneous infusion with insulin pumps or by multiple injections. The DCCT was needed to prove conclusively

that better glucose control mitigates the ravages of diabetes in type 1 disease in which the incidence of microvascular disease was significantly reduced in an intensively treated group when compared with a conventionally treated group (glycosylated haemoglobin [$HbA_{1C}$], 7% versus 9%). Type 2 diabetes is 10 times more prevalent than type 1, results of two prospective trials, the Kumamoto study (20) and UKPDS 33 (21), which addressed the benefits of glycemic control in type 2 diabetes, will have an enormous impact.

In the Kumamoto trial, the intensive therapy group had an average $HbA_{1C}$ level of 7% compared with 9% in the conventionally treated group. This translated into less retinopathy (7.7% versus 32%), less nephropathy (7.7% versus 28%) and improvement in nerve conduction for the intensively treated group.

In the UNKPDS 33, average $HbA_{1C}$ levels were 7% in the intensively treated group and 7.9% in the conventional group. The 0.9% reduction was associated with 12% fewer diabetes-related end points in the intensively treated group (P=0.029). Diabetes-related end points were principally defined as sudden death, death from hyperglycaemia or hypoglycaemia, myocardial infarction, angina, heart failure, stroke, renal failure, amputation, and eye complications. Most of this benefit (25% risk reduction) was found with microvascular end points (P=0099). The risk of myocardial infarction was reduced 16% in the intensively treated group, but this was of borderline significance (P=0.52)

Although it is convenient to separate microvascular disease (retinopathy, nephropathy, neuropathy) from macrovascular disease (myocardial infarction, stroke, amputation), this distinction is artificial. This is because peripheral neuropathy may lead to amputation of a denervated foot, and similarly, a denervated heart from automatic neuropathy may result in sudden death. Again, diabetic nephropathy is associated with hypertension, dyslipidaemia, and hyperhomocysteinemia, all potent risk factors for macrovascular disease (15)

## 6. Nephropathy

Diabetic nephropathy develops in close to 40% of patients with type 1diabetes and 5% to 40% of patients with type 2 diabetes. Genetics plays an important role: patients who have one or two deletions of the angiotensin-converting enzyme (ACE) gene, a defect in the sodium proton pump, or a family history of hypertension are at increased risk of progression to diabetic nephropathy (22). However, in such patients, nephropathy does not occur until type 1 diabetes develops; the worse and more prolonged the hyperglycaemia, the greater the risk of diabetic nephropathy (23)

Development of Nephropathy: A proposed Mechanism for development of nephropathy in patients with type 1 diabetes is elevation of growth hormone levels due to poor glycaemic control, resulting in hyperperfusion of glomerulus and glomerular hypertension. Mesangial cells in the glomerulus respond to glomerular hypertension by producing growth factors, especially tumour necrosis factor-a, which results in increased glomerular permeability, proliferation of glomerular epithelial cells and excessive production of basement membrane and collagen tissue. These effects eventually lead to glomerular scarring and renal failure (24). Patients who are prone to excessive production of collagen (e.g. those in whom keloid forms) are at greatest risk, which may explain the higher prevalence of diabetic nephropathy in African American (25).

Pathogenesis in patients with type 2 diabetes differs in that Mesangial lymphokine production is associated not only with hyperglycaemia but also with increased resistance and generalised vascular diseases. Thus albuminuria may occur even before hyperglycaemia develops.

In the early stages of diabetes nephropathy, there are no clinical signs or

symptoms of renal disease. Glomerular changes can be identified only by renal biopsy, which is impractical, or by the presence of microalbuminuria (Albumin excretion rate, >30 but ¾ 300mg/day). Therefore, all diabetic patients should be tested annually for microalbuminuria (26).

Microalbuminuria often occurs before a rise in blood pressure is detected. If microalbuminuria is found, causes other than diabetes (e.g. urinary infection, Ketosis, use of dihydropyridine calcium blockers) should be ruled out. Microalbuminuria should be confirmed on at least two separate occasions one or two months apart. Tight glycaemic control, keeping glycosylated haemoglobin $A_{1C}$ ($HbA_{1C}$) levels within or just above the non-diabetic range, undoubtedly is beneficial.

When type 1 diabetes has been present for 10 to 15 postpubertal years, Macroalbuminuria (albumin excretion rate, >300mg/day) may appear as the first clinical indication of diabetic nephropathy. Invariably, oedema or hypertension accompanies Macroalbuminuria. Untreated, albuminuria worsens, causing a classic nephrotic syndrome; however, as the number of surviving glomeruli that have the ability to filter albumin decrease, the nephrotic syndrome resolves.

After Macroalbuminuria appears, without treatment, renal function declines at the rate of about 1mL/min per month, or 10% per year; thus end-stage renal disease occur after about 7 years. Treatment can prolong the active life of the kidney, but once Macroalbuminuria occurs, end-stage renal disease is inevitable.

Inadequate treatment of hypertension, use of radiocontrast materials or potentially nephrotoxic drugs, overuse of diuretics, and urinary tract infection may hasten progression to end-stage renal disease and should be carefully avoided (27)

Onset of diabetic nephropathy may be avoided with good glycaemic control, such as that used in the DCCT (average $HbA_{1C}$ level, 7.1%) (28). In the primary-prevention group, who had type 1 diabetes for less than 5 years and had no retinopathy, patients who received intensive versus standard insulin therapy had a 34% decrease in the frequency of microalbuminuria and no significant decrease in the frequency of Macroalbuminuria. In the secondary-prevention group, who already had mild retinopathy, patients who received intensive therapy had a significant decrease in both microalbuminuria (43%) and Macroalbuminuria (56%).

In patient with type 2 diabetes, a recent interventional study (29), showed a decrease in the frequency of development of both microalbuminuria 57% and macroalbuminuria (70%) with intensive insulin therapy. Diabetes nephropathy may have already developed since type 2 diabetes is diagnosed, on the average, 8 years after onset.This delay in diagnosis may be alleviated somewhat by recent recommendations that the fasting blood sugar used as a criterion for diagnosis be lowered to 7 mmol/L (120mg/dL) and that everyone over the age 45 (or younger if at high risk) be screened for diabetes every 3 years with use of a fasting glucose test. Microalbuminuria and even macroalbuminuria can be present at the time of diagnosis in type 2 diabetes patients. The most likely cause of this phenomenon is insulin resistance (30), but delayed diagnosis and the presence of macrovascular disease and hypertension could also explain the finding of proteinuria in patients with recently identified type 2 diabetes. Only 50 % to 70% of NIDDs and nephropathy have retinopathy, compared with 100% of IDDs, indicating the presence of other risk factors for nephropathy in patient with type 2 diabetes.
Aggressive control of hypertension in diabetic patients without microalbuminuria has been shown to help avoid nephropathy (31). ACE inhibitors should be part of the regimen, and a low-protein diet may also be helpful.

In Africa, studies on nephropathy are scarce. M.S. Abudulla found 46% of study patients had renal involvement, 45.7% of whom had hypertension and 7% had features of Kimmelstiel-wilson syndrome (32).

### 7. Peripheral neuropathy

Peripheral neuropathy is associated with the risk of foot ulcers, amputations and charcot joints. This is associated with poor glycaemic control and poor education. Peripheral neuropathy was found to be 10% in a study by Mulaisho (in 1977–79) (1) and was 14.7% in a study by Ju Egere and Nonwankwa in 1972 at UTH and Kitwe Central Hospital respectively. (1, 33).

### 8. Diabetic foot complication

Diabetic foot ulcers are very common, with an annual incidence of 2% to 3% and prevalence of 4% to 10% (34). The figures indicate that a large percentage of foot ulcers are chronic. Half of all amputation in the United States occur in patients with diabetes. In a study at UTH, it was found that the commonest indication for amputation was trauma 31.5% and road traffic accidents were the major contributors. The second commonest was dry gangrene in 22% followed by tumours and diabetes in 9% in each case (personal communication)

Needing a first amputation is a poor prognostic sign in diabetic patients; 28% to 51% of these patients require a second amputation within 5 years. The presence of peripheral diabetic complications leading to amputation is also associated with systemic complications. The 5-year mortality rate after lower extremity amputation ranges from 39% to 68% (34).

Sensory neuropathy, ischaemia, and infection are the predominant pathogenic factors in development of foot ulcers in diabetic patients (35). Peripheral sensory neuropathy is found in 80% of such cases. Ulceration develops because patients lack protective sensation to warn them of injury to the foot. As a result, puncture wounds may go unnoticed, foreign bodies may remain in subcutaneous tissue, or poorly fitting shoes may continue to be worn until pressure necrosis develops.

The most common location for neuropathic ulcers is over bony prominences (e.g.metatarsal heads). Repetitive and excessive pressure leads to formation of a callus, which eventually separates from the underlying dermis forming an ulcer. Disruption of the protective skin barrier leads to infection with skin organisms and the warm, moist environment within a shoe accelerates spread of the infection. In addition, ischaemia caused by peripheral vascular disease retards wound healing. Patients with diabetes also commonly have morphologic changes in foot structure (e.g. bony dislocation and collapse of the arch resulting from charcot's neuropathic arthropathy). This continues the cycle of abnormal weight bearing, excessive pressure, and ulceration.

Hyperglycaemia also effects host immunologic defences. Granulocyte adherence, chemotaxis, phagocytosis, and bactericidal function are impaired with hyperglycaemia and improved with better glucose control (36).

**9. Cardiovascular Complications**

Cardiovascular Complications (e.g. coronary artery disease [CAD], stroke, peripheral vascular disease, cardiomyopathy, and congestive heart failure) are leading cause of morbidity and mortality related to Diabetes mellitus. Complications of atherosclerosis are responsible for about 80% of deaths in

diabetic patients and 75% of hospitalisation for diabetic complications (37). However, this has been found true in European and North American series and seen rarely in series done in Africa (1,33)

Atherosclerosis is due to abnormalities in lipoprotein metabolism. Even before developing hyperglycaemia, persons with impaired glucose tolerance have an elevated risk of macrovascular disease. Fifty percent of patients have evidence of cardiovascular disease at the time of diagnosis of type 2 diabetes.

Diabetes can be considered a vascular disease because it causes both microvascular complications (e.g.nephropathy, retinopathy) and macrovascular complications. Tight glycaemic control has been shown to prevent long-term microvascular complications in patients with type 1 diabetes in the DCCT (28) and those with type 2 diabetes in the UKPDS (17). However glycaemic control did not reduce the incidence of cardiovascular events in either study. This result in the DCCT may have been due, in part, to the young age of the subjects and the short duration of the study.

The American Heart Association now recognises diabetes as a major cardiovascular risk factor (4). Other major cardiovascular risk factors include cigarette smoking, dyslipidemia, and hypertension, which act as independent contributors to cardiovascular disease in diabetic patients.

**10. Dyslipidemia**

Patients with type 2 diabetes have an atherogenic dyslipidemia characterised by three lipoprotein abnormalities; elevated levels of very-low-density Lipoprotein (VLDL) triglycerides; small, dense low-density lipoprotein (LDL) cholesterol

particles; and decreased high-density (HDL) cholesterol. Evidence suggests that all elements of diabetic dyslipidemia are independently atherogenic. Because of frequent changes in glycaemic control of diabetic patients and their effect on lipoprotein levels, the American Diabetic Association has recommended that fasting levels of LDL, HDL, total cholesterol, and triglycerides be measured every year in adult patients. If values fall in lower risk levels, assessment may be repeated every 2 years.

There is increasing evidence supporting a role for VLDL triglycerides in macrovascular disease in persons with diabetes. Results of the Paris prospective study showed that a plasma triglyceride level 125mg/dl was the only factor significantly associated CAD deaths in patients with impaired glucose tolerance or diabetes (39). Studies of triglyceride-lowering therapy in patients with type 2 diabetes are limited to subgroup analysis. In a subgroup analysis of the Helsinki Heart Study (40), for example a non-significant trend toward reduction of myocardial infarction and cardiovascular disease was noted with Gemfibrozil therapy.

The effect of reduced HDL levels on the accelerated cardiovascular disease in diabetes has not been clearly isolated, because raising HDL cholesterol levels has proved difficult without the aid of physical activity, weight loss, and nicotinic acid therapy (which may increase insulin resistance and hyperglycaemia. The veterans Affairs High-density Lipoprotein cholesterol Intervention Trial found a 22% reduction in relative risk for CAD, death, and nonfatal myocardial infarction in men receiving gemifibrozil therapy that resulted in a 6% increase in HDL cholesterol levels. There is yet no subgroup analysis of the diabetic patients.

Several studies have shown that diabetes causes specific modifications in LDL cholesterol that increase atherogenic potential, including glycation and small,

denser LDL cholesterol particles (37). Although lipid intervention studies have largely excluded patients with diabetes, two secondary prevention trials clearly showed that lowering LDL cholesterol levels reduces the risk of major cardiac events to an even greater degree in diabetic patients than non-diabetic patients. In a subgroup analysis of the Scandinavian Simvastatin Survival Study, patients with type 2 diabetes who received Simvastatin therapy had a 37% reduction in the risk for any cardiovascular event after a 35% reduction in LDL cholesterol levels ($P<0.01$) (41). The other study that showed that treatment with pravatastin sodium reduced the risk of cardiac events in diabetic patients by 25% ($P\leq0.001$) was Long-Term Intervention with pravastatin in Ischaemic Disease Study (42). Both studies used post hoc subgroup analysis to identify a significant sample of diabetic patients.

## 1.2 TREATMENT OF DIABETES

The goals of treatment of diabetes are to control symptoms and to prevent acute and long-term complications. Several studies have shown that maintaining near-normal glucose levels helps prevent long-term complications and improves quality of life for people with diabetes mellitus (21,23,28). Diabetes mellitus is treated by the following measures:

### a. DIET

All patients with diabetes require diet therapy. Good glycemic control is unlikely to be achieved with insulin or oral therapy when diet is neglected, especially when the patient is also overweight. In older patients diet alone should be tried in the first instance, and dietary knowledge and compliance should always be reassessed with care before proceeding to the next step. This is of particular importance in obese patients who fail to lose weight. Regular exercise helps to control weight and reduce cardiovascular risk.

The diet for a diabetic patient is in principle no different from the diet considered healthy for the population as a whole. Calories should be tailored to the needs of the diabetic patient. The total amount of carbohydrates in diet should provide 50-55% of the total calories, with fat 30-35% and protein 15%. An overweight patient is started on a reducing diet of 1000-1600 kcal (4000-6000 J). A lean patient is put on an isocaloric diet. Patients who are underweight because of the untreated diabetes require energy supplementation. Patients on insulin or oral agents should be advised to eat the same amount at the same time each day. Patients on insulin require snacks between meals and at bedtime to buffer the effect of injected insulin. Alcohol is not forbidden, but its energy content should be taken into account.

## b. ORAL HYPOGLYCAEMIC AGENTS

Diet and lifestyle changes are the key to successful treatment of NIDDM. If satisfactory metabolic control of diabetes is not established by these methods after several weeks, then tablets may be needed in addition. There are currently 5 classes of oral hypoglycaemic agents. These include the following:

### i. SULPHONYLUREAS

Their principal action is to promote insulin secretion in response to glucose and other secretagogues. They were in addition believed to increase insulin sensitivity in peripheral tissues, but this view is now largely discounted. Sulphonylureas are therefore ineffective in patients without a functional beta-cell mass and should not be avoided in young ketotic patients, who require early insulin therapy, and are contraindicated in pregnancy. Insulin should be substituted during surgery or severe intercurrent illness. Hypoglycaemia is the most common and dangerous side effect.

The first-generation: Tolbutamide, chlorpropamide and tolazamide are rarely used today because they depend on renal excretion and tax the kidney.

The second-generation: Glyburide, glipizideand glimepiride – are more potent and don't require the renal excretion.

Sulphonylureas are metabolized in the liver except chlorpropamide, which is excreted partially unchanged in the urine.

An average-weight adult who has no lipid abnormalities is an ideal candidate for sulphonylurea therapy early in the coarse of diabetes. Therapy starts with low, single daily doses that are gradually increased until patient reaches his target glucose level. About 20% of patients who take Sulphonylureas don't respond to therapy at all (primary failure). Another 10% have a positive initial response but glucose control diminishes over time (secondary failure).

**ii. MEGLITINIDE**

Another insulin secretagogue, meglitinide has many of the same actions and adverse effects as the Sulphonylureas. Designed to treat postprandial hyperglycaemia, it increases insulin release but more rapidly than sulphonyureas. The only meglitinide currently available is repaglinide (prandin). Its effect is glucose-dependent and decreases when the patient's glucose level decreases. Metabolized in the liver, so the risk for accumulation is minimal.

**iii. BIGUANIDES**

Metformin is currently the only biguanide on the market. It works by reducing hepatic glucose production, enhancing tissue response to insulin, and improving glucose transport into the cells. Used alone, it doesn't cause hypoglycaemia because it doesn't stimulate insulin secretion.

Metformin typically doesn't promote weight gain and may improve lipid abnormalities. The dosage is typically started low and gradually increased until patient's blood glucose and HbA1c values reach target levels.

Metformin is primarily excreted through the kidneys, so document your patient's baseline serum creatinine level before starting therapy. If renal function is impaired, the drug could accumulate in his blood stream and cause lactic acidosis. Baseline liver function tests are recommended too because lactate metabolism occur in the liver. If the patient has a condition such as heart failure, which puts the patient at risk for acute renal dysfunction or tissue hypoperfusion, Metformin should be discontinued and replaced by another drug.

**iv. ALPHA-GLUCOSIDASE INHIBITORS**

Acarbose and miglitol are the available drugs in this class. Metabolized by intestinal bacteria and digestive enzyme, the limit the absorption of carbohydrates

from the small intestines. They're most appropriate for a patient who has normal fasting blood glucose levels and significantly elevated postprandial readings; they have no direct effect on fasting-blood glucose levels. The best way to gauge the effectiveness of alpha-glucosidase inhibitor therapy is to monitor the patient's 2-hour postprandial blood glucose levels. There is no risk of hypoglycaemia when on arcabose or miglitol unless the patient is also taking a sulphonylurea or insulin.

**v. THIAZOLIDINEDIONES**

The two currently available thiazolidinediones, rosiglitazone (Avandia) and pioglitazone (Actos), combat Type 2 diabetes by increasing sensitivity at insulin receptor sites on the cells. They're most appropriate for adults who produce insulin but can't use it because the insulin receptor sites are inadequate or ineffective. These drugs don't cause hypoglycaemia and they may have positive effects on lipid levels and blood pressure.

Thiazolidinediones are primarily metabolized in the liver, so the patient should undergo liver function testing before starting therapy.

**vi. COMBINATION THERAPY**

Once oral agents become ineffective, substituting another drug rarely wins. Combining therapies, however, can be highly effective. Combinations may include oral agents from two or more classes or an oral agent with bedtime dose of NPH insulin. Sulphonylureas are frequently combined with Metformin, an alpha-glucosidase inhibitor, or insulin.

**c. INSULIN**

Insulin is found in every vertebrate, and the central-part of the molecule shows few species differences. Small differences in the amino acid sequence may alter

the antigenicity of the molecule. Beef insulin differs from human insulin by three amino acids and readily induces antibody formation, whereas pork insulin, which differs by only one amino acid, is relatively non-immunogenic. Animal insulin has now largely been replaced by biosynthetic human insulin, which is produced by DNA coding of cultured yeast or bacterial cells to produce proinsulin, with subsequent enzymatic cleavage to insulin. This technology has permitted development of insulin analogues, in which the structure of the insulin molecule is modified in such a way as to modify its pharmacokinetics without altering its biological effect. Human insulin is now standard in most of the west, but animal insulin are still used widely in developing countries. Different insulin preparations are available depending on the onset of activity. There are the fast acting groups that include soluble, intermediate, and long acting formulations such as ultra-lente. Complications of insulin therapy include weight gain, hypoglycaemia, lipodystrophies and allergy.

## 1.3 JUSTIFICATION FOR THE STUDY

The University Teaching Hospital in Lusaka has major financial constraints, which restrict the clinical service it can provide. This has resulted in patients with diabetes mellitus in the diabetic clinic not being monitored for most of the long-term complications. Patients are admitted to the wards with hyperglycemic emergencies and hypoglycemia and factors leading to this acute metabolic decompensation have not been addressed. These complications lead to increased morbidity and mortality among diabetic patients. There have been studies done which show improved outcome with better glycaemic control, better control of hypertension, proteinuria and hyperlipidaemia. These include the Diabetic Control and complication Trial of 1993 (DCCT) and the United Kingdom prospective Diabetes Study of 1998 (UKPDS).

Therefore, there is need to know the nature and extent of these complications in the diabetic clinic so that guidelines in the investigations and treatment of these complications can be formulated.

## 1.4 OBJECTIVES OF THE STUDY

a. GENERAL OBJECTIVE

To describe the common complications associated with diabetes mellitus in the diabetic clinic at UTH, Lusaka.

b. SPECIFIC OBJECTIVES

i. To describe common complications and their prevalence among diabetic patients at UTH diabetic clinic
ii. To identify factors associated with the development of complications
iii. To obtain data that would be used to make recommendations to the diabetic clinic
iv. To describe the standard of care of the diabetic patient in the clinic.

# CHAPTER 2

## 2.1 RESEARCH METHODOLOGY

### STUDY SITE

The study was conducted prospectively at the UTH diabetic clinic from January 2001 to September 2001. The sources of the patients were the adult filter clinic, medical wards, surgical wards, gynaecology and obstetric wards and referral patients from outside UTH.

### STUDY DESIGN

This was a cross-sectional descriptive study and involved review of patients' records, detailed history from patient, physical examinations and investigations.

### SAMPLING PROCEDURE

Eighty patients were prospectively recruited from the diabetic clinic during the usual clinic days.

### INCLUSION CRITERIA

Adults aged 15 years and older.

Attendance at the diabetic clinic and had to have the UTH medical record.

Patients had to meet the criteria of diagnosing DM i.e. fasting blood sugar greater or equal to 7mmol/L or random blood sugar greater than 10mmol/L.

A written or verbal consent to participate in the study.

Should have attended the diabetic clinic on two or more occasions.

Should have at least one or more complications of diabetes mellitus



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**Exclusion Criteria**

Patients less than 15 years old.

Patients with no record in diabetes clinic.

Diabetes mellitus of less than one year duration.

Patients with fasting blood glucose <5.5 or random blood glucose <7.8 mmol/L.

## CLINICAL METHODS

All patients in the study underwent a thorough clinical evaluation. This included a medical history and physical examination. Medical history included reviewing past medical history i.e. medical records of the patients.

Physical examination included the following:

1.Anthropometric measurements: Height and weight, from which the body mass index BMI was calculated using the formula:

BMI = weight (kg)/height in metres)$^2$

2.Blood pressure: measured in sitting and standing position. Hypertension was diagnosed with BP equal-to or greater-than 140/90 mmHg on three occasions or those already receiving treatment.
3.Peripheral pulses were palpated i.e. Dorsalis pedis, posterior tibial and popliteal
4.The cardiovascular and respiratory systems were examined
5.The central nervous system was examined and included the state of the skin, vibration sense with a tuning fork, ankle jerks, knee jerk, touch with an orange stick and pinprick.

6.Fundoscopy: was done after dilating the pupils with cyclopentolate and in difficult cases were referred to the ophthalmologist.

7. Auscultation of the carotid and femoral arteries for bruits.

## 2.2 LABORATORY METHODS

Fasting blood sugar was done on all patients.

Urinalysis was done for clinical nephropathy. Clinical nephropathy was defined as proteinuria on at least two occasions at 1-2 months apart using conventional dipsticks. Early morning (first void) urine samples were used to avoid exercise-induced proteinuria and to avoid increase in proteins following a meal.

Other tests included the following:

(1) Urine for microscopy, culture and sensitivity.

(2) Lipid profiles (Total cholesterol, HDL, VLDL and LDL).

(3) ECG

(4) Echocardiography

(5) X-ray (for the chest and lower limbs); according to the indication and availability of the test.

## DATA COLLECTION AND ANALYSIS

All the data was recorded on the data collection sheets, which was then pooled and analysed with the advice of medical statistician using the EPI-INFO programme.

# CHAPTER 3

## RESULTS

**Table 1. Sex distribution of the patients studied**

| SEX | FREQUENCY | PRECENT |
|---|---|---|
| MALE | 35 | 43.8% |
| FEMALE | 45 | 56.3% |
| TOTAL | 80 | 100% |

There were more females than males 56.3% to 43.8%.

**Table 2. Age distribution**

| AGE | FREQUENCY | PERCENT |
|---|---|---|
| 15 - 24 | 5 | 6.3% |
| 20 – 34 | 6 | 7.5% |
| 35 – 44 | 8 | 10.0% |
| 45 – 54 | 17 | 21.3% |
| 55 – 64 | 24 | 30.0% |
| 65 – 74 | 17 | 21.3% |
| 75 – 84 | 3 | 3.8% |
| TOTAL | 80 | 100% |

The age distribution was wide between 15 years and 77 years with a mean age of 53.4 years. The majority of the patients fell in the age group between 55 to 64 years (30%). The median was 56.5 years, the standard deviation was 14.6 and the mode was 57 years.

**Table 3. Body mass index**

| Body mass index | frequency | Percent |
|---|---|---|
| 16.73 – 18.4 | 5 | 7.5% |
| 18.5 – 24.9 | 29 | 43.3% |
| 25.0 – 29.9 | 14 | 20.9% |
| 30.0 – 39.9 | 18 | 26.9% |
| >40 | 1 | 1.5% |
| Total | 67 | 100% |

A total of 67 patients had their body mass index measured. 7.5% of the patients were below the acceptable range of the BMI. 43.3% were within acceptable range, 20.9% were overweight, 26.9% were obese and one patient was found to be morbidly obese representing 1.5% of the patients.

**Table 4. Diabetic treatment history**

| Treatment | Frequency | Percentage |
|---|---|---|
| Diet alone | 4 | 5.1% |
| Diet and hypoglycaemic | 44 | 55.7% |
| Diet and insulin | 31 | 39.2% |
| Total | 79 | 100% |

There were a total of 79 patients whose treatment was clear with the majority being those on diet and hypoglycaemic drugs (55.7%), followed by those on insulin (39.2%), and those on diet alone were only 4 representing 5.1%.

**Table 5. (a) Marital status**

| Marital status | Frequency | Percentage |
|---|---|---|
| Single | 7 | 8.8% |
| Married | 54 | 67.5% |
| Widowed | 16 | 20.0% |
| Divorced | 3 | 3.8% |
| Total | 80 | 100% |

Most of the patients were married (67.55%), 20.0% were widowed, 8.8% were single and 3 patients (3.8%) were divorced.

**(b) Cross tabulation of marital status with sex**

| Marital status | Male | Female | Total |
|---|---|---|---|
| Single | 2 | 5 | 7 |
| Married | 31 | 23 | 54 |
| Widowed | 1 | 15 | 16 |
| Divorced | 1 | 2 | 3 |
| Total | 35 | 45 | 80 |

54 patients were married and the majority being males. 16 patients were widowed and the majority were females, 7 patients were single and 3 patients were divorced (2 females and 1 male).

**Table 6. Duration of diabetes**

| Duration | Frequency | Percentage | Cummulative Total |
|---|---|---|---|
| 1- 9 years | 47 | 59.5% | 59.5% |
| 10-19 years | 22 | 27.8% | 87.3% |
| 20-29 years | 6 | 7.6% | 94.9% |
| 30-39 years | 4 | 5.1% | 100% |
| Total | 79 | 100% | |

Most of the patients had duration of diabetes ranging between 1-9 years (59.5%); 27.8% between 10-19 years, 7.6% between 20-29 years and 4 patients between 30-39 years (5.1%). The duration of diabetes for the patients in the survey ranged between 1-35 years.

**Table 7. Cross tabulation of age and duration of diabetes**

| Age | 1-9 years | 10-19 years | 20-29 years | 30-39 years | Total |
|---|---|---|---|---|---|
| 15-24 years | 4 | 1 | 0 | 0 | 5 |
| 25-34 years | 2 | 3 | 0 | 1 | 6 |
| 35-44 years | 6 | 2 | 0 | 0 | 8 |
| 45-54 years | 13 | 4 | 0 | 0 | 17 |
| 55-64 years | 14 | 7 | 2 | 0 | 23 |
| 65-74 years | 5 | 5 | 4 | 3 | 17 |
| 75-84 years | 3 | 0 | 0 | 0 | 4 |
| Total | 47 | 22 | 6 | 4 | 79 |

The age groups between 45-54 years, 55-64 years and age group between 65-74 years had the majority of patients with the duration of diabetes of between 1-9 years. Also there are the age groups with longer duration of diabetes that is duration between 10-19 years, 20-29 years and 30-39 years for age group between 65-74 years.

**Table 8. Family history of diabetes**

| Family history | frequency | Percent |
|---|---|---|
| Yes | 25 | 31.3% |
| No | 55 | 68.8% |
| Total | 80 | 100% |

There was a positive family history of diabetes in 31.3% and in 68.8% the history was negative.

**Table 9. History of classical symptoms**

(a) Polyuria/Nocturia

| Polyuria/Nocturia | Frequency | Percent |
|---|---|---|
| Present | 43 | 54.4% |
| Absent | 36 | 45.6% |
| Total | 79 | 100% |

(b) Polydipsia

| Polydipsia | Frequency | Percent |
|---|---|---|
| Present | 39 | 49.4% |
| Absent | 40 | 50.6% |
| Total | 79 | 100% |

(c) Polyphagia

| Polyphagia | Frequency | Percent |
|---|---|---|
| Present | 30 | 38.0% |
| Absent | 49 | 62.0% |
| Total | 79 | 100% |

The classical symptoms of polyuria/nocturia, polydipsia and polyphagia were 54.4%, 49.4% and 38.0% respectively.

**Table 10. Attendance to diabetic clinic**

| Attendance | Frequency | Percent |
|---|---|---|
| Good | 67 | 85.9% |
| Fair | 10 | 12.8% |
| Poor | 1 | 1.3% |
| Total | 78 | 100.0% |

The majority of the patients attended diabetic clinic three times in nine months representing 85.9% while 12.8% attended twice and one patient attended only once. Two other patients had come for the first time as referrals.

**Table 11. History of self-monitoring**

| Self-monitoring | Frequency | Percent |
|---|---|---|
| Positive | 9 | 11.3% |
| Negative | 71 | 88.8% |
| Total | 80 | 100.0% |

The majority of the patients did not monitor their diabetic control (88.8%) and only 11.3% had some form of self-monitoring.

**Table 12. Type of self-monitoring**

| Type | Frequency | Percent |
|---|---|---|
| Blood | 3 | 33.3% |
| Urine | 6 | 66.7% |
| Total | 9 | 100.05 |

The most common method used for self-monitoring was urine (66.7%) and the use of blood for self-monitoring was only 33.3%.

**Table 13. History of smoking**

| Smoking history | Frequency | Percent |
|---|---|---|
| Positive | 5 | 6.25% |
| Negative | 75 | 93.75% |
| Total | 80 | 100.0% |

The number of patients who were found to be smokers were 5 representing 6.25%. 93.75% were non-smokers. All of the patients found to be smokers were male.

**Table 14. History of hypoglycaemia**

| History of hypoglycaemia | Frequency | Percent |
|---|---|---|
| Positive | 15 | 19.2% |
| Negative | 63 | 80.2% |
| Total | 78 | 100.0% |

19.2% of the patients had a positive history of hypoglycaemia.

**Table 15. Cross tabulation of age and hypoglycaemia**

| Age group | Positive history | Negative history | Total |
|---|---|---|---|
| 15-24 years | 3 | 2 | 5 |
| 25-34 years | 3 | 3 | 6 |
| 35-44 years | 1 | 7 | 8 |
| 45-54 years | 4 | 13 | 17 |
| 55-64 years | 1 | 21 | 22 |
| 65-74 years | 3 | 14 | 17 |
| 75-85 years | 0 | 3 | 3 |
| Total | 15 | 63 | 78 |

There were four patients with hypoglycaemia in the age group 45-54 years, 3 patients in the age groups 15-24, 25-34 and 65-74 years, 1 patient each in the age groups 35-44 and 55-64 years and none had hypoglycaemia of the 3 patients in the age group 75-85 years. The p value was 0.039.

**Table 16. Type of treatment with history of hypoglycaemia**

| Type of treatment | Positive | Negative | Total |
|---|---|---|---|
| Diet alone | 0 | 4 | 4 |
| Diet and hypo | 2 | 41 | 43 |
| Diet and insulin | 13 | 18 | 31 |
| Total | 15 | 63 | 78 |

None of the 4 patients who were on diet alone had hypoglycaemia. 2 patients of the 43 patients who were on diet and oral hypoglycaemic drugs had hypoglycaemia. The majority of patients who had hypoglycaemia where among those who on insulin were 13 out of 18 patients had hypoglycaemia. The p value = 0.00019.
( hypo = hypoglycaemic drugs).

**Table 17. Patients with Diabetic Ketoacidosis**

| History of DKA | Frequency | Percent |
|---|---|---|
| Positive | 7 | 9.2% |
| Negative | 69 | 90.8% |
| Total | 76 | 100.0% |

Only 7 patients of the 76 patients had diabetic ketoacidosis representing 9.2%.

**Table 18. Cross tabulation of age group and diabetic ketoacidosis**

| Age group | Positive | Negative | Total |
|---|---|---|---|
| 15-24 years | 3 | 1 | 4 |
| 25-34 years | 1 | 5 | 6 |
| 35-44 years | 1 | 6 | 7 |
| 45-54 years | 2 | 15 | 17 |
| 55-64 years | 0 | 22 | 22 |
| 65-74 years | 0 | 17 | 17 |
| 75-84 years | 0 | 3 | 3 |
| Total | 7 | 69 | 76 |

Diabetic ketoacidosis was highest in the age group 15-24 years were 3 out of the patients had DKA. 1 patient had DKA in the age groups 25-34 and 35-44 years out of 5 and 6 respectively. 2 patients out of 15 had DKA in the age group 45-54 years. There were no cases of DKA in the age groups 55-64, 65-74 and 75-84 years. The p value = 0.00025.

**Table 19. Cross tabulation of type of treatment and DKA**

DKA

| Type of treatment | Positive | Negative | Total |
|---|---|---|---|
| Diet alone | 0 | 4 | 4 |
| Diet + hypo | 1 | 42 | 43 |
| Insulin | 6 | 23 | 29 |
| Total | 7 | 69 | 76 |

The majority of patients who developed DKA were those who were
On treatment with insulin (6 patients out of 23), 1 patient out of 42 patients who were on oral hypoglycaemic drugs had DKA and none of the patients on diet alone had DKA. P value = 0.025.

**Table 20. Cross tabulation of duration of diabetes and DKA**

| | DKA | | |
|---|---|---|---|
| Duration | Positive | Negative | Total |
| 1-9 years | 4 | 42 | 46 |
| 10-19 years | 3 | 17 | 20 |
| 20-29 years | 0 | 6 | 6 |
| 30-39 years | 0 | 4 | 4 |
| Total | 7 | 69 | 76 |

DKA occurred in the patients with duration of diabetes of 1-9 and 10-19 years. There were no cases of DKA in the patients with duration of diabetes of 20-29 and 30-39 years.

**Table 21. Patients found to have hypertension**

| Hypertension present | Frequency | Percent |
|---|---|---|
| Positive | 58 | 74.4% |
| Negative | 20 | 25.6% |
| Total | 78 | 100.0% |

Hypertension was present in 74.4% of the diabetic patients in the study group.

**Table 22. Table of duration of diabetes and hypertension**

| | Hypertension | | |
|---|---|---|---|
| Duration | Positive | Negative | Total |
| 1-9 years | 33 | 14 | 47 |
| 10-19 years | 16 | 5 | 21 |
| 20-29 years | 5 | 1 | 6 |
| 30-39 years | 4 | 0 | 4 |
| Total | 58 | 20 | 78 |

33 out of 47 patients in the group with duration of diabetes of 1-9 years were hypertensive, 16 out of 21 in the group with duration 10-19 years, 5 out 6 in the group 20-29 years duration and all the 4 patients were hypertensive in the 30-39 years duration.

**Table 23. Outcome of eye examination**

| Eye findings | Frequency | Percent |
|---|---|---|
| Cataract | 31 | 38.8% |
| Normal | 33 | 41.3% |
| Retinopathy | 16 | 20.0% |
| Total | 80 | 100.0% |

38.8% of study patients had cataracts, 20.0% had retinopathy and 41.3% had normal findings.

**Table 24. Patients with hypertension and retinopathy**

| Hypertension | Present | Total |
|---|---|---|
| Retinopathy | 16 | 16 |
| Total | 16 | 16 |

All the patients with retinopathy were found to be hypertensive.

**Table 25. Age groups with diabetic eye disease**

| Age group | Cataract | Normal | Retinopathy | Total |
|---|---|---|---|---|
| 15-24 years | 2 | 3 | 0 | 5 |
| 25-34 years | 1 | 4 | 1 | 6 |
| 35-44 years | 2 | 4 | 2 | 8 |
| 45-54 years | 7 | 8 | 2 | 17 |
| 55-64 years | 8 | 11 | 5 | 24 |
| 65-74 years | 8 | 3 | 6 | 17 |
| 75-84 years | 3 | 0 | 0 | 3 |
| Total | 31 | 33 | 16 | 80 |

The presence of either cataract or retinopathy appears to increase age.

**Table 26. Duration of diabetes with eye disease**

| Duration | Cataract | Normal | Retinopathy | Total |
|---|---|---|---|---|
| 1-9 years | 18 | 24 | 5 | 47 |
| 10-19 years | 7 | 7 | 8 | 22 |
| 20-29 years | 3 | 1 | 2 | 6 |
| 30-39 years | 3 | 0 | 1 | 4 |
| Total | 31 | 32 | 16 | 79 |

The presence of either cataract or retinopathy increases with the duration of diabetes. The prevalence of either cataract or retinopathy increases in a given duration of diabetes.

**Table 27. Type of treatment with eye disease**

| Treatment | Cataract | Normal | Retinopathy | Total |
|---|---|---|---|---|
| Diet alone | 2 | 2 | 0 | 4 |
| Diet + hypos | 18 | 17 | 9 | 44 |
| Insulin | 11 | 13 | 7 | 31 |
| Total | 31 | 32 | 7 | 79 |

Of the patients cataract 2 were on diet alone, 18 on diet and oral hypoglycaemic drugs and 11 were on insulin. Those with retinopathy 9 were on diet and oral hypoglycaemic drugs, 7 were on insulin and none were on diet alone.

**Table 28. Male patients with history of erectile dysfunction (ED)**

| History of ED | Frequency | Percent |
|---|---|---|
| Positive | 18 | 58.1% |
| Negative | 13 | 41.9% |
| Total | 31 | 100.0% |

58.1% of the 31 male patients had history of erectile dysfunction.

**Table 29. Male patients with ED and hypertension**

| | Presence of hypertension | | |
|---|---|---|---|
| History of Ed | present | Absent | Total |
| Positive | 14 | 4 | 18 |
| Absent | 7 | 5 | 12 |
| Total | 21 | 9 | 30 |

14 patients with erectile dysfunction had hypertension while 4 with erectile dysfunction did not have hypertension.

**Table 30. Patients with peripheral neuropathy symptoms**

| Numbness/painful extremities(feet + hands) | Frequency | Percent |
|---|---|---|
| Present | 32 | 40.0% |
| Absent | 48 | 60.0% |
| Total | 80 | 100.0% |

40% of the patients in this study group had features of peripheral neuropathy.

**Table 31. Comparing symptoms and pinprick examination of the feet**

| Pinprick | Symptoms +ve | Symptoms -ve | Total |
|---|---|---|---|
| Normal | 15 | 44 | 59 |
| Reduced | 17 | 2 | 19 |
| Total | 32 | 46 | 78 |

Of the 32 patients with symptoms 17 had reduced sensation to the pinprick and 15 the sensation was normal.
(+ve/-ve = presence or absence respectively).

**Table 32. Comparing symptoms and touch**

| Touch | Symptoms +ve | Symptoms -ve | Total |
|---|---|---|---|
| Normal | 13 | 42 | 55 |
| Reduced | 19 | 4 | 23 |
| Total | 32 | 46 | 78 |

19 patients of the 32 with symptoms had reduced sensation to touch while 13 had normal sensation but had symptoms.

**Table 33. Comparing ankle jerks with symptoms of peripheral neuropathy**

| Ankle jerks | Symptoms +ve | Symptoms -ve | Total |
|---|---|---|---|
| Increased | 0 | 2 | 2 |
| Normal | 3 | 32 | 35 |
| Reduced/Absent | 29 | 12 | 41 |
| Total | 32 | 46 | 78 |

29 patients with symptoms of peripheral neuropathy had either reduced or absent ankle jerks. 3 patients had symptoms but the ankle jerks were normal.

**Table 34. Diabetic foot**

| Diabetic foot | Frequency | Percent |
|---|---|---|
| Absent | 69 | 86.3% |
| Present | 11 | 13.8% |
| Total | 80 | 100.0% |

13.8% of the patients were found to have diabetic foot of the 80 patients in this series.

**Table 35. Duration of diabetes and diabetic foot diabetes**

| Duration of diabetes | Frequency diabetic foot | Total |
|---|---|---|
| 1-9 years | 3 | 3 |
| 10-19 years | 5 | 5 |
| 20-29 years | 1 | 1 |
| 30-39 years | 2 | 2 |
| Total | 11 | 11 |

The majority of patients with diabetic foot were found in the group of patients with duration of diabetes 10-19 years followed by 3 in 1-9 years, 2 in the 30-39 years and 1 in the 20-29 years. Most important diabetic foot complications were found in all the duration of diabetes.

**Table 36. Comparing symptoms of peripheral neuropathy and diabetic foot**

| Foot examination | Symptoms present | Total |
|---|---|---|
| Diabetic foot | 11 | 11 |
| Total | 11 | 11 |

All the patients with diabetic foot had symptoms of peripheral neuropathy.

**Table 37. Comparing the state of the skin of the feet and symptoms of peripheral neuropathy**

| State of skin | Symptom +ve | Symptoms -ve | Total |
|---|---|---|---|
| Callus | 2 | 0 | 2 |
| Darkened, hair loss | 1 | 0 | 1 |
| Hair loss | 7 | 0 | 7 |
| Hair loss, callus | 1 | 0 | 0 |
| Normal | 22 | 46 | 68 |
| total | 33 | 46 | 79 |

11 patients had skin changes with symptoms of peripheral neuropathy while 22 patients had normal skin but were symptomatic.

**Table 38. Patients with heart failure**

| Features of failure | Frequency | Percent |
|---|---|---|
| Present | 8 | 10.0% |
| Absent | 72 | 90.0% |
| Total | 80 | 100.0% |

10% of the patients had heart failure.

**Table 39. Patients with strokes**

| Features of stroke | Frequency | Percent |
|---|---|---|
| Present | 3 | 3.8% |
| Absent | 77 | 96.3% |
| Total | 80 | 100.0% |

3.8% were found to have strokes. 2 patients had right-sided strokes and 1 had a left-sided stroke with a left carotid bruit. 2 other patients were found to have ocular cranial nerve palsies (1 had third cranial nerve palsy and the other had the seventh cranial nerve palsy) representing 2.5% patients.

**Table 40.Cross tabulation of duration of diabetes and heart failure**

| Duration | Frequency | Total |
|---|---|---|
| 1-9 years | 3 | 3 |
| 10-19 years | 3 | 3 |
| 20-29 years | 1 | 1 |
| 30-39 years | 1 | 1 |
| Total | 8 | 8 |

The patients with failure were found at every period of diabetic duration but commonly in the patients with duration 1-9 and 10-19 years. None of these patients were smokers.

**Table 41. Urinalysis result (proteinuria)**

| Presence of protein | Frequency | Percent |
|---|---|---|
| 1+ Protein | 3 | 3.8% |
| 3+ Protein | 1 | 1.3% |
| Negative | 74 | 93.7% |
| Trace | 1 | 1.3% |
| Total | 79 | 100.0% |

93.7% of the urine was negative for protein and 6.3% was positive for protein. Only 3 patients were found to have clinical nephropathy (3.8%) and these patients elevated urea and creatinine.

**Table 42. Patients with tuberculosis**

| Presence of TB | Frequency | Percent |
|---|---|---|
| Positive | 12 | 15.0% |
| Negative | 68 | 85.0% |
| Total | 100 | 100.0% |

15% of the patients were found to be on TB treatment, diagnosed or gave history of TB treatment

**Table 43. Total cholesterol of the 5 patients with lab results**

| | |
|---|---|
| 0.43 mmol/L | Normal |
| 15.6 mmol/L | Greatly raised |
| 3.35 mmol/L | Normal |
| 6.14 mmol/L | Raised |
| 6.63 mmol/L | Raised |

3 patients out of 5 had increased cholesterol and 2 of these patients had angina (2.5%) with ECG changes.

# CHAPTER 4

## DISCUSSION AND CONCLUSION

The study looked at the common complications associated with diabetes mellitus in the diabetic clinic at the University Teaching Hospital, Lusaka.

There were 80 patients in the study, of which the majority 45 (56.3%) were female patients and 35 (43.8%) were male patients (See table 1). These results are similar to the findings by other investigators who also found a female preponderance in their study (35). This could be as a result of more females seeking medical attention than males or diabetes mellitus is more common in females.

There was a wide range in terms of age distribution. The youngest was 15 years and the oldest was 77 years in this study group. The mean age was 53.4 years. The majority of the patients were in the age group 55-64 years (30%). This may be due to the fact that the majority of the patients are the non-insulin dependent diabetic (NIDDs)-See table 2.

67 patients had their body mass index (BMI) measured. In the Western countries minimum mortality is seen in people who are 90-110% of ideal body weight as calculated from the Metropolitan Life Insurance statistics, corresponding to BMI of 19-24 for females and 20-25 for males. People with a BMI> 25 are generally said to carry too much fat. Studies have demonstrated increasing risk of premature death with increasing obesity, although other short studies with shorter follow up

have not always confirmed Hippocrates` observation that ` the very fat are apt to die young` (35). 7.5% were below the accepted range for BMI, 43.3% were within the accepted range and 49.3% were above the accepted range (20.9% were over weight, 26.9% were obese and 1.5% were morbidly obese. The patients below the accepted range needed to be on diet to increase weight and needed to be on insulin but the advice was to lose weight and most were on oral hypoglycaemic drugs. The 49.3% who were obese needed to be on Metformin (15, 21), which has been shown to be a drug of choice, and also to be on diet and exercise (15). All the obese patients were on Glibenclamide and insulin, which may encourage weight gain. There was little dietary advice and advice to exercise was non-existent. See table 3.

55.7% of the patients were on diet and oral hypoglycaemic drugs, 39.2% were on insulin and 5.1% were on diet alone. See table 4.

Majority of the patients were married (67.5%), 20% were widowed, 8.8% were single and 3.8% were divorced. The majority of married patients were male and the majority of the widowed patients were female. The married patients have someone to look after them in cases of emergencies such as hypoglycaemia and also in chronic complications such as blindness due to cataracts or retinopathy. Those who were widowed mostly elderly female patients who have difficulties learning about their condition and also had very little support from their families and were looking after very young grandchildren. See table 5 (a) and (b).

The duration of diabetes for the patients in the study ranged between 1-35 years. The majority of the patients had duration of diabetes between 1-9 years (59.5%). The age group 65-74 years had the majority of patients with longer duration of diabetes. This shows clearly that the incidence of diabetes increases with age and that the majority of the patients have non-insulin dependent diabetes (5). See table 6.

There was a positive family history of diabetes in 31.3%. This is important in that the knowledge can be used to screen the other family members for diabetes. The majority of the patients in this study have type 2 diabetes supported by the obesity and the type of treatment. In fact, it is estimated that onset of type 2 diabetes precedes diagnosis by 10 to 12 years (29, 30). The major risk factors for type 2 diabetes are (1) family history of diabetes (i.e., parents or siblings with diabetes). (2) Obesity (greater than or equal 20% over desired body weight or body mass index > 27kg/m2). (3) Race or ethnicity, with high risk of diabetes (e.g. African American, Hispanic American, Native American) (30). (4) Age equal to or greater than 45 years. (5) Previously identified impaired fasting glucose or impaired glucose tolerance. (6) Hypertension (Greater than or equal to 140/90 mmHg. (7) Hyperlipidemia (HDL cholesterol level < 35 mg/dl [0.90 mmol/L] or triglyceride level > 250 mg/dl [2.82 mmol/L], or both. (8) History of gestational diabetes or delivery of a baby over 4.1 kg. See table8.

The classical symptoms of diabetes were commonly polyuria and nocturia (54.4%), polydipsia (49.4%) and polyphagia (38%). This reflects the fact that diabetic control was poor in this study. Most of the patients had very elevated random or fasting blood sugars. Diabetic control in our clinic is done by fasting or random blood sugars and glycosylated haemoglobin is not routinely done. See table 9.

Attendance to diabetic clinic was good (85.9%) and poor in only 1.3%. This points to other reasons other than compliance to the poor control and presence of the complications. The likely contributing factors are shortages of medication, laboratory facilities and poor education (6). See table 10.

Only 11.3% of the study patients had some form of self-monitoring for their diabetes. 66.7% of those with self-monitoring used urinalysis and 33.3% was

blood glucose monitoring. Urinalysis is not the best method of monitoring mainly for physiological reasons. For most patients, the renal threshold for excretion of glucose is 180 mg/dl (10 mmol/L). Hence, glucose may remain undetectable in urine until levels exceed 10 mmol/L in plasma. Glycosylated haemoglobin is considered an important monitoring tool for diabetes but not available in the University Teaching Hospital (UTH). See table 11 and 12.

6.25% of the study patients were smokers. All the smokers were male. Diabetes is recognised as a major cardiovascular risk factor (15, 39, 40, 41, 42, 43, 44). The other risk factors, cigarette smoking, dyslipidemia, hypertension, act as independent contributors to cardiovascular disease in diabetic patients. In the study group the risk is very small. This might account for the difference in the pattern of morbidity caused by the above mentioned co-morbid states in respect of ischaemic heart disease (34, 35). See table 13.

19.2% of the patients had a history of hypoglycaemia. The majority of the patients were in the age group 45-54 years (p value = 0.039). The highest episodes of hypoglycaemia were found among patients on insulin treatment. The p value = 0.00019 which is less than 0.05. This is statistically very significant. 2 patients on diet and hypoglycaemic drugs had hypoglycaemia. Hypoglycaemia occurred at all periods of duration of diabetes from 1-35 years but commonly among those with duration from 1-19 years. The risk factor to developing hypoglycaemia was omission of the meal after receiving insulin. The other risk factors include alcohol intake, exercise and not adjusting the dose of insulin, adjusting dose upwards without consulting medical team and attempted self harm can not be ruled-out. Since hypoglycaemia was common among the patients with duration of diabetes

of 1-19 years, the newly diagnosed patients should be educated on symptoms of hypoglycaemia. All the patients were not carrying sugar or any sugary food on them at the time of the episode. See table 14,15 and16.

Diabetic ketoacidosis (DKA) occurred in 9.2% of the study patients. DKA occurred in the patients with duration from 1-54 years. The highest cases of DKA were in the age group 15-24 years (p value = 0.00025 which was very significant). The majority of patients were on insulin treatment (p value = 0.025 which was significant). 1 patient out of 42 on diet and hypoglycaemic drugs developed DKA. All patients who developed DKA were in the duration of diabetes of between 1-19 years. Mostly the precipitants were omission of insulin treatment due to shortages and infections. Patients with duration of diabetes of 20-39 years did not develop DKA. This could be due to the fact that they were mainly Type 2 and usually oral hypoglycaemic drugs are available and rarely get overwhelming infections (8, 9, 10 and11). This shows that DKA is common among younger patients than in older age-groups (9). The incidence DKA is also very high in this study at 9.2% the incidence is quoted to be 2 per 100 patients per year (8). This may represent failure of education in those without stress or precipitating infection (10). See table 17, 18,19 and 20.

74.4% of the patients had hypertension in this study. This compare well with the figures quoted in most of the studies of 40-75% (14). Hypertension was found among all duration of diabetes from 1-39 years. The prevalence of 74.4% contrasts significantly with what was found by C. Mulaisho in 1973 in the clinic in which hypertension was only 26.92%. The majority of patients were found among those with duration of diabetes of 1-9 years. The hypertension was mostly not well controlled and patients had other associated complications such as

clinical nephropathy, strokes, coronary artery disease, hyperlipidemia and erectile dysfunction and retinopathy. The number of patients with hypertension was highest in the older age groups. The disparity in prevalence in two studies done in the same clinic can be due to the differences in the definition of hypertension. See table 21 and 22.

The major Eye complications were cataract and retinopathy. In this study 38.8% had Cataract and 20% had Diabetic retinopathy (DR). The prevalence of either cataract or retinopathy increased with advancement in age and duration of diabetes. Cataract develops in all age groups but is more common in older patients. Development of cataract or retinopathy is independent of the type of treatment the patient has been receiving because the conditions were found in all the treatment types. All the patients with diabetic retinopathy had hypertension. All had background retinopathy with one patient with macula involvement and poor vision. From the literature review the over prevalence of retinopathy is 25%. Those with NIDDM the prevalence is 20% and 40% in those with IDDM (19). See table 23, 24, 25, 26 and27.

Erectile dysfunction (ED) was common among the male patients. 58% had complaints of impotence. The majority of the patients had hypertension. There could be a role by the drugs used in treating hypertension in causing ED. See table 28 and 29.

Peripheral neuropathy was 40% in this study and was sensory in nature with complaints of numbness and painful/burning feet. These patients also had reduced ankle jerks. Peripheral neuropathy was found in 10% by Mulaisho in the same clinic in 1973 (1). The difference in the prevalence in the studies would be attributed to poor control of the diabetes in our patients due to shortages in

medication and laboratory supplies as a result of the economic decline Zambia has undergone over the years. See tables 30, 31, 32 and 33.

Diabetic foot complication was 13.8%. This figure is higher than that in the literature review (36), in which the prevalence is 4-10%. Diabetic foot complication is not affected by duration of diabetes and is commonly associated with peripheral neuropathy and infection. The patients were poorly educated on foot care. Diabetic control was poor in this study from the raised blood sugars in the majority of the patients. Peripheral vascular disease was rare in the study. Only one patient was found to have features of peripheral vascular disease. Associated features of diabetic foot complication included callus formation, darkening of the skin and loss of hair. See tables 34, 35, 36, and 30.

Heart failure was found in 10% of the study patients. Heart failure develops at any duration of diabetes and can be a presenting complication. Most patients with heart failure had hypertension. See table 38.

3.8% of the patients in the study had strokes. All the patients had associated hypertension. One patient had features of artherosclerosis with left-sided Carotid bruit. None of these patients were smokers. See table 39 and 40.

Clinical nephropathy was found in 3.8%. 93.7% of the samples were negative for protein and 6.3% were positive. Ordinary dipsticks for protein are insensitive to pick nephropathy because urine protein has to be detected (> 150mg/L). A lot of cases with microalbuminuria could have been missed in this study. All the patients with clinical nephropathy had hypertension.

Tuberculosis (TB) was found in 15% patients in the study. The annual incidence of TB in Zambia has risen from 100/100,000 between 1975-1985 to 350/100,000

in1994 (Ministry of Health statistics; latest figures are estimated to closer to 450/100,000). Recent research has shown a high prevalence of human immunodeficiency virus (HIV) and TB and that it is one the commonest cause of admission in adults and children at the University Teaching Hospital, UTH, Lusaka, Zambia (Elliot A, Luo N, et al, 1990). See table 40. Diabetes causes immunosuppresion (38) and this can explain the high levels TB in this study being from a country with high incidence. There is need to know the co-morbid HIV status in the diabetic patients at UTH. Other infections common in the study were fungal skin infection and urinary tract infection. Only 1 patient with TB had results for HIV testing. This patient was found positive with a low CD4 count of 74 but was not on treatment for HIV.

2.5% of the patients had angina (2 patients). 3 patients of the 5 patients who had lipid profiles had raised cholesterol levels. This is in contrast with the studies done by Seftel and McCance AJ who did not find any cases of ishaemic heart disease in the black Africans diabetic patients (34, 35). They attributed the absence of coronary artery disease despite the frequency of hypertension and diabetes mellitus to the absence of other conventional risk factors, particularly high serum cholesterol, in the black African. The presence of high cholesterol levels in this study may reflect a change in diet and people becoming more sedentary as they adopt the Western way of living. This is also reflected in the majority of our patients being obese (Table 3).

The patients in this study were poorly controlled as evidenced by the frequency of symptoms of diabetes and the very high fasting and random blood sugars the patients had. Most of the patients were poorly evaluated for the complications. Many did not have the eyes checked for cataract and retinopathy. There was no yearly fundoscopy and did not have their feet checked for complications. Laboratory services during the study were dodged with shortages and breakdown of equipment.

There was a serious shortage of insulin and oral hypoglycaemic drugs. The only oral hypoglycaemic drug given to patients at UTH is Glibenclamide. Metformin could only be bought in the private pharmacies and majority of the patients could not afford. Owing to the shortage patients resorted to rationing the drugs by either reducing the doses or reducing the frequency, e.g. giving oneself soluble insulin once a day.

All the patients in this study were black Africans. Majority were Zambians, 4 were from Democratic Republic of Congo and 1 from Kenya.

This study was done to describe the common complications of diabetes mellitus and to determine the prevalence of these complications. The study identified factors associated with these complications and described the standard of care of the diabetic patients in the diabetic clinic at the University Teaching Hospital, Lusaka, Zambia.

The patients in this study were poorly controlled as evidenced by presence of classical symptoms of diabetes (polyuria/nocturia was 54.4%, polydipsia was 49.4% and polyphagia was 38%) and also the high fasting and random blood sugar results.

Majority of the patients in this study did not have access to self-monitoring facilities. This can explain the increased frequency of hypoglycaemia and DKA in this study and the generally uncontrolled diabetes in these patients. This also can explain the high frequency of peripheral neuropathy including autonomic involvement in the male patients with ED and the increased frequency of infections (fungal skin infection, diabetic foot complication and TB cases). Hyperglycaemia is known to predispose to infection (38). Only 11.3% had some form of self-monitoring.

The unavailability of resources to treat patients in resource poor Nations who have infectious and non-infectious diseases despite the proven scientific efficacy

of anti-infectives and other therapeutics, raises several major ethical issues. The right of all human beings to enjoy living standards conducive to good health is enshrined in Article 25 of the United Nations Universal Declaration of Human Rights, established just over 50 years ago. This states that "everyone has the right to a standard of living adequate for the health and well-being of themselves and of their family, including food, clothing, housing, and medical care and necessary social services, and the right to security in the event of unemployment, sickness, disability, widowhood, old age, or other lack of livelihood in circumstances beyond their control. "

In 1978, the World Health Organization and the United Nations Children's Fund sponsored an International Conference on Primary Health Care at Alma-Ata in the former Soviet Union. At that conference a statement, the 'Alma-Ata Declaration', was formulated and opened with the reaffirmation that health is a fundamental human right and that the attainment of the highest possible level of health is an important world-wide social goal. The Alma-Ata Declaration states that the gross inequality in the health of people, particularly between the developed and developing countries, is politically, socially and economically unacceptable and it calls for a new international economic order to address this inequality. Following this Declaration, the World Health Organization set the goal of achieving "Health for All by the Year 2000", an objective that has failed.

Some of the major hurdles associated with the unavailability of essential medicines in resource poor Nations include:

1. General poverty in developing countries

2. Poor prescribing habits including "polypharmacy" due to limited diagnostic facilities

3. Lack of National drug policies in many developing countries

4. Limited research aimed at determining the most appropriate drugs and therapeutics in most developing countries

It should be realised that financial access to essential drugs does not necessarily mean correct use and therefore some of the erratic drug supply seen in most resource Countries could be attributed to poor prescribing habits as a result of limited or absent diagnostic facilities. If the supply of essential drugs and therapeutics are to be improved, the capacity for these Countries to diagnose common ailments should be improved and manpower training and retraining should be tied to programs aimed at improving drug supply.

This study clearly demonstrates that for a disease that has recognized therapeutics, many patients at the University Teaching hospital develop complications that would otherwise be prevented with tight control and monitoring of their disease.

## STUDY LIMITATIONS

The limitations of this study included the following:

The sample size of 80 patients was rather small for one to make generalisations to he population of diabetic patients in the diabetic clinic at the UTH. The sample was small because of the short time in which to do the study and to submit the dissertation to the University Of Zambia. I was also affected by the nurses' and doctors strike, which lasted for over a month during which recruitment was suspended because of the closure of the clinic. There were serious financial constraints because my sponsors Central Board of Health did not release the money. The lack of laboratory services during the study period also contributed to this small sample size because the recruitment of the study patients had to be suspended at times.

This was a hospital-based study and so it has an inherent bias in that the patients seen are those mostly with complications and so the figures of complications will tend to be high especially that UTH is a tertiary hospital.

There was also inadequate laboratory service. Diabetic glycaemic control was based on fasting and random blood sugar as glycosylated haemoglobin is not done at UTH. Glycated haemoglobin of 7% and less has been shown to improve outcome for many of the complications in this study and reflects control in last 3 months. Diabetic nephropathy also could not be determined by use of ordinary urinalysis dipsticks and creatinine. The ordinary dipstick becomes positive when proteinuria > 150mg/L and creatinine begins to rise when there is significant fall in glomerular filtration rate (GFR) with antecedent microalbuminuria which is proteinuria > 30 but ¾ 300mg/L. From this it can be stated that a lot of patients with microalbuminuria were missed in this study.

The lipid profiles (Total cholesterol, High density lipoproteins (HDL), Low density lipoproteins (LDL) and tryglycerides) should have been done on all the patients but lack laboratory supplies and lack of funding hampered the study.

I would have loved to do HIV testing in the study patients to determine the prevalence of HIV. This can confound the figures of the patients with TB and peripheral neuropathy but time was short and there was no money to buy the kits or to pay to have the tests done.

The other problem was the lack of measuring scale to weigh the patients in the diabetic clinic. The figures of the BMI would have been affected in this study.

Record keeping is also a problem in UTH and the files of the patients were lost and these could not be recruited in the study and a number of patients were lost to follow up. This contributed to the small sample size.

The major drawback in this study was the lack of finance and it proved to be a drain on my personal finance and most of the tests could therefore not be done.

## RECOMMENDATIONS

There is need to train medical personnel to look after diabetic patients. Those to be trained should include doctors, nurses, nutritionists and laboratory technologists. There is need in the meantime to employ a consultant with knowledge of endocrinology who can train postgraduate students who currently run diabetic clinic. There is also need to have experts from other hospitals to come and teach at UTH.

Most of the complications are as a result of poor education given to the patients. A better system of educating the patients should be implemented in the clinic. This should involve diabetic nurse educator, diabetic nutritionist and the doctor. Preferably there is need to augment what is taught to the patients with posters in the clinic and printed handouts.

There is need to have a scale in the clinic and bed for weighing those that cannot stand. Weight is important in the choice of oral hypoglycaemic drugs and in determining the diet and exercise regime of the patient.

There is need to improve record keeping in the clinic, which can be used for future research. In this vein a computerised system is preferable. There is also need to design a different card for diabetic patients with such features as type of treatment, duration of diabetes, complications, eye examination results and when next due, level of control, urinalysis results, lipid profiles, results of feet examination, etc.

There is need to strengthen the laboratory services offered at UTH. The management should introduce glycosylated haemoglobin and radioimmunoassay for microalbuminuria. Lipid profiles should be readily done on all the patients especially the obese and hypertensive patients.

All adults who have the highest risk of developing diabetes such as those with hypertension, obesity, mothers of children who weigh > 4.1kg, relatives of diabetic patients and those older than 40 years should be encouraged to have fasting blood sugars tested for diabetes. This will prevent patients coming with serious complications.

The pharmacy should consider diversifying the range of oral hypoglycaemic drugs from just glibenclamide to include metformin and the newer drugs. This will avoid obesity as a consequence, which is a problem already in our diabetic population. Increasing the range of drugs will improve the standard of care. There is need also to stock drug for use in hypertension of a wide range including angiotensin converting enzyme inhibitors (ACE-I) which have been shown in studies to slow the rate of developing nephropathy, to improve outcome in heart failure and after myocardial infarction. These medications currently are erratically supplied by our pharmacy but they should stocked at all time to reduce morbidity and mortality by diabetes. The drugs are expensive for average Zambians and most of them fell to buy from private pharmacies.

There is need for further research in the diabetic clinic to determine prevalence of co-morbid HIV. This will help in determining the prevalence of peripheral neuropathy, diabetic nephropathy and diabetic eye complications. HIV can cause similar complications. HIV treatment with protease inhibitors can lead to dysglycaemias including diabetes and also is a risk to hyperlipidemia. Further research is needed in determining the prevalence of nephropathy in our diabetic population by using radioimmunassays for proteinuria and also the prevalence of hyperlipidemia. Zambia is a country with a high prevalence of HIV and TB. The prevalence of TB in the study might thus reflect on the prevalence of HIV in the diabetic patients in the diabetic clinic. HIV in patients with diabetes will pose new challenges in the management of the patients in future.

Data from this study suggests that the majority of our patients are non-insulin dependent. Most of the patients were middle aged and were obese and had hypertension. All these are risk factors to cardiovascular diseases such as strokes, heart failure, coronary artery disease and peripheral vascular disease. There is need therefore to determine the prevalence of both type-1 and type –2 diabetes. This can only be done by checking for anti-islet antibodies in the blood of type-1 diabetes patients. This epidemiological study will help in the management of major types of diabetes in future. As the data has indicated that hyperlipidemia might be a problem in the diabetic population, access to electrocardiography, lipid profiles, cardiac enzymes, computer tomogram (CT-SCAN), coronary angiography and drugs to lower the hyperlipidemia, may in future create a strain on the already constrained hospital. This is particularly more alarming because world-wide the prevalence of diabetes especially non-insulin dependent type is on the increase. As people become more affluent in Zambia and more westernised, the levels of the `western diseases` will be expected to increase.

The results of this study reveal that diabetic eye disease is prevalent. Both cataracts and retinopathy are causes of preventable blindness. The co-operation between the diabetic clinic and the eye clinic in terms of diagnosis and management of these complications should be emphasised. There is need to have laser treatment at UTH. Cataract extractions will increase as most of the patients are middle aged and diabetes accelerates the development of cataract in this population. There should be clinics for diabetic patients in the eye clinic were doctors from diabetic clinic will be taught on eye care especially that these doctors are postgraduate students.

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**APPENDICES**

**DATA COLLECTION FORM**

**A. IDENTITY**

- Study Number.................................... Date:.............................
- File Number:....................................
- Age:.............................................
- Marital Status:..................................
- Address.......................................... Residential Area:.............
- Duration of Diabetes..........................
- History of Diabetes in close relative: Yes/No

**B. CLINICAL FEATURE (TICK)**

| | |
|---|---|
| - Polyuria/Nocturia | Yes/No |
| - Polydipsia | Yes/No |
| - Polyphagia | Yes/No |
| - Poor Vision | Yes/No |
| - Diarrhea | Yes/No |
| - Vomiting | Yes/No |
| - Painful/numbness/hands/feet | Yes/No |
| - Sores on feet | Yes/No |
| - Swelling of feet | Yes/No |
| - Weakness/weight loss | Yes/No |
| - Skin rashes | Yes/No |
| - Impotence | Yes/No |

**C. TREATMENT HISTORY**:

1. DIET ALONE
2. DIEAT + ORAL HYPOGLYCEMICS
3. INSULIN
4. OTHER DRUGS

(Specify):..........................................................................................

COMPLIANCE/AVAILABILITY/GOOD/FAIR/POOR

HISTORY OF SMOKING: YES/NO

**D. ATTENDANCE TO DIABETES CLINIC:** Good/Bad/N/A

**E. SELF MONITORING:** Yes/No TYPE:...............

**F. PAST MEDICAL ADMISSION:** PRECIPITANT:

For (i) Diabetes Ketoacidosis Yes/No .........................

(ii) Hyperrosmolar non ketotic Yes/No ............................

(iii) Stabilization Yes/No ............................

(iv) Hypoglycemia Yes/No ............................

(v) TB Treatment Yes/No ............................

**G. PHYSICAL EXAMINATION**

(i) Weight .................................

(ii) Height .................................

(iii) Body mass index .................................

(iv) Skin .................................

(v) Eyes (Fundoscopy) .................................

(vi) Respiratory system .................................

(vii) CVS: (i) SVP ........................

(ii) Heart sounds ..............

(iii) SP 1. Sitting..........................

2. Standing.......................

(iv) Peripheral pulses

a. Dorsalis Pedis (RT) (LT)

b. Pasterior Tibial (RT) (LT)

c. Popliteal (RT) (LT)

(viii) (i) State of skin.................................................

(ii) Vibration sense..............................................

(iii) Ankle RT.............................. LT....................

(iv) Knee jerks RT........................ LT....................

(v) Pin prick.......................................................

(vi) Touch...........................................................

## H. INVESTIGATIONS:

1. URINALYSIS: $1^{st}$ early morning sample for protein......................(1)

   ....................(2)

   .....................(3)

2. U/E + Creatinine/fasting blood sugar (where appropriate............... ....

## CONCENT FORM FOR INCLUSION IN THE STUDY

I am doing a research on complications of "sugar disease" which is also known as diabetes. Diabetes is an illness beset with a lot of complications. These affect the eyes, heart and blood vessels leading to raised blood pressure, the nerves and may affect the kidneys. These are the complications, which will be looked for in this study. In the study, I will request you to be examined following an interview and you will be required to give urine for analysis and also blood for estimation of your sugar levels and to check how your kidneys are functioning if you wish to participate in this study from your own free will, you will be required to sign for inclusion in the study. This study will give us a better way of managing patients in future.

I........................................ (Full name) hereby give consent for inclusion in the study I have understood the details of the study as explained to me by the investigator.

**Signature**..............................................................

**Date**....................................................................