THE MAGNITUDE AND PATTERN OF DIABETIC

RETINOPATHY AT CENTRAL HOSPITAL OF YAOUNDE,

CAMEROON

A study carried out in part fulfilment for the degree of Master of Medicine in Ophthalmology in

the University of Nairobi

Dr. Njikam Jude Eric, MD

2011

DECLARATION

This dissertation is my original work and has not been presented for a degree in any other University.

Signed...Date....17/10/2011

Dr. Njikam Jude Eric, MD

PPROVAL

his dissertation has been submitted for examination with our approval as University

upervisors.

igned..Date 15/11/2011

r. Kariuki Millicent Muthoni,

IB.ChB, M.Med. Ophthalmology (Nairobi), FEACO.

ecturer, Department of Ophthalmology

niversity of Nairobi.

igned..Date 21/10/2011

r. K.H. Martin Kollmann,

IB.ChB, MD, D.Trop.Med & Med. Parasitol., M.Med.Ophthalmology, MBA Health Care,
EACO,

enior Lecturer, Department of Ophthalmology,

niversity of Nairobi.

DEDICATION

To my closest friend, Nina, my lovely son Samuel for the continuous support, encouragement and patience given to me while undertaking my postgraduate studies and my parents, who put a relentless effort for my achievement and all my teachers.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

BCVA   Best corrected Visual acuity

BP   Blood Pressure

CHY   Central Hospital of Yaoundé.

CSME   Clinically Significant Macular Edema.

CURES   Chennai Urban Rural Epidemiological Study

DCCT   Diabetes Control and Complication Trial

DR   Diabetic Retinopathy

DRS   Diabetic Retinopathy Study

ETDRS   Early Treatment of Diabetic Retinopathy Study

FBS   Fasting Blood Sugar

HBA1c   Glycosylated haemoglobin

HRPDR   High Risk Proliferative Diabetic Retinopathy

IDDM   Insulin Dependent Diabetes mellitus

IFG   Impaired Fasting Glucose

IGT   Impaired Glucose Tolerance

NHRPDR   Non High Risk Proliferative Diabetic Retinopathy

NVD   Neovascularisation at the disc

NVE   Neovascularisation Elsewhere

OHA   Oral Hypoglycaemic Agents

PDR   Proliferative Diabetic Retinopathy

UKPDS   United Kingdom Prospective Diabetic Study

WHO   World Health Organization.

ABSTRACT

Objective: To determine the magnitude, pattern and associations of diabetic retinopathy in diabetic patients attending diabetic clinic of Central Hospital of Yaoundé, Cameroon (CHY).

Design: Cross-sectional hospital based study.

Subjects: A total of 371 patients ranging from 20-99 years of age attending the diabetic clinic during the month of September to mid October 2010 underwent detailed eye examination for diabetic retinopathy. The blood pressures, HbA1c, fasting blood sugar of all the patients were also measured and clinical grading of diabetic retinopathy was done by using the ETDRS guidelines.

Results: The prevalence of diabetic retinopathy was found to be 49.2%. Severe NPDR was found in 12(3.2%) patients, CSME was found in 30(8.1%) patients, PDR in 53(14.3%) patients and vision threatening diabetic retinopathy was found in 27.3% of patients. There was a statistically significant association between diabetic retinopathy and duration of diabetes, HbA1c, FBS, systemic blood pressure and nephropathy. Only 41 (11.1%) patients did not have an eye examination prior to this study. Among the study subjects only 50.1% had their HbA1c below 7%. There was no association of DR and sex of the patient.

Conclusion: The prevalence of diabetic retinopathy was high in this study and the number of diabetics who had an eye examination by an ophthalmologist prior to this study was 88.9%. Majority of the subjects had poor blood sugar control. explaining probably the high prevalence of diabetic retinopathy and therefore the need for a better referral system for early screening and management of DR in this population.

1.0 INTRODUCTION AND LITERATURE REVIEW

1.1 Diabetes Mellitus

In 1999, WHO defined diabetes mellitus as a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both. The effects of Diabetes mellitus include long-term damage, dysfunction and failure of various organs[1]. Thus, the metabolic abnormalities result from inadequate insulin action on target tissues, due to deficient insulin action, or a combination of both[2]

1.1.1 Epidemiology

Diabetes mellitus is one of the most common non-communicable diseases, and its epidemic proportion has placed it at the forefront of public health challenges facing the world. In estimating the total number of persons with diabetes mellitus, we cannot rely solely on reported numbers of diagnosed cases. It is estimated that about half of persons with diabetes are unaware of their disease and, even in industrialized countries, many individuals go undiagnosed. Although more recent data show that the proportion of undiagnosed cases has decreased in some areas, it is still at least about one quarter to one third of all persons with diabetes mellitus[1].

It is predicted that between 2000 and 2025, the size of the world's adult population will increase from almost 4 billion to 5.5 billion, mainly on account of a 60% increase in developing countries[1]. At the same time the number of adults with diabetes in the world is predicted to increase from 150 million in 2000 to 300 million in 2025, while in developing countries that

1

number will be more than double[1]. Thus, in 2025, more than 75% of the world's diabetic population will be living in developing countries.

These projections of the number of people with diabetes take into account the fact that there will be more people in the world (population growth) and that there will be more elderly people (population ageing). They also take into account trends in urbanization, physical inactivity, and obesity. In fact current trends in obesity suggest that these projections are conservative and that the increase in the prevalence of diabetes may be even greater[2].

In developing countries, it is the people in the middle, productive years of their lives that are particularly affected by diabetes. In these countries, three-quarters of all people with diabetes are under 65 years old and 25% of all adults with diabetes are younger than 44. In industrialized countries, more than half of all people with diabetes are older than 65, and only 8% of adults with diabetes are younger than 44[1].

The prevalence of diabetes in persons 35-64 years in sub-Saharan African in 2000 is estimated 3-5%[3]. The number of people estimated to have diabetes by WHO in Kenya in 2000 was 183,000 and this was projected to increase to 498,000 by year 2030[4]. In Cameroon, the number of people estimated to have diabetes by WHO in 2009 was 900,000 about 6% of total population of Cameroon[5].

1.1.2 Classification of diabetes mellitus

Several classifications exist for diabetes. The recommended classification includes both staging of diabetes mellitus based on clinical descriptive criteria and a complementary etiological classification. The etiological type named Type 1 or insulin dependant diabetes mellitus (IDDM)

encompasses less than 10% of diabetics, of which majority are primarily due to pancreatic islet beta–cell destruction and are prone to ketoacidosis. It includes those cases attributable to an autoimmune process, as well as those with beta–cell destruction and those for whom neither etiology nor pathogenesis is known (idiopathic)[1,2,6].

Type 2 includes the common form of diabetes (over 90%) which results from defect(s) in insulin secretion, almost always with a major contribution from insulin resistance. The class "Impaired Glucose Tolerance" is classified as a stage of impaired glucose regulation, since it can be observed in any hyperglycemic disorder, and is itself not diabetes. A clinical stage of "Impaired Fasting Glycaemia" has been introduced to classify individuals who have fasting glucose values above the normal range, but below those diagnostic of diabetes.

Gestational diabetes is a state of carbohydrate intolerance resulting in hyperglycemia of variable severity, with onset or first recognition during pregnancy. The definition applies irrespective of whether, or not insulin is used for treatment, or whether the condition persists after pregnancy[1,6].

1.1.3 Diagnostic criteria

There is abundance of data indicating that hyperglycaemia is harmful. However there are limitations in the data and the methodologies used to derive cut off points at which this level of harm is specifically increased and which clearly differentiates diabetes from non diabetes. It is thus difficult to accurately define normal glucose levels. Despite the limitations with the data from which the diagnostic criteria for diabetes are derived, the current WHO criteria distinguish a group with significantly increased premature mortality and increased risk of microvascular and cardiovascular complications (Appendix No I).

3

The diagnosis of diabetes in an asymptomatic subject should never be made on the basis of a single abnormal blood glucose value. For the asymptomatic person at least one additional plasma/ blood glucose test result with a value in the diabetic range is essential (either fasting or from oral glucose tolerance test).

1.1.4 Treatment

The backbone of diabetes management is proper diet and regular exercise. which have to be individualized[6]. Both could be the only management needed for controlling blood glucose in gestational diabetes, IGT and type 2 diabetes in its early phase. Patient with type 2 diabetes may require oral hypoglycaemic agent and/or insulin. while type 1 patients need insulin therapy to survive. The treatment plan for diabetes may include diabetes education. meal planning and nutritional recommendations, exercise. oral anti-diabetic agents. insulin and the management of associated conditions and complications[6, 7 8].

1.1.5 Diabetic eye disease

Diabetes mellitus has been associated with lesions in the eye such as conjunctival micro aneurysms, reduced corneal sensation and tear production. thickened corneal stroma. cataract. transient lenticular myopia during hyperglycaemia, iris neovascularisation (rubeosis iridis). thickened basement membrane at the pigment epithelium of the pars plicata. arteriosclerosis of the choroids. obliterated lumen of the choriocapillaris at the macula. vitreous detachment and haemorrhage. diabetic retinopathy, drusen, glaucoma. optic disc neovascularisation. ischemic optic neuropathy and central retinal vein occlusion[9]

1.2   Diabetic Retinopathy

Diabetic retinopathy (DR) can be defined as damage to the micro-vascular system of the retina due to prolonged hyperglycaemia. It occurs both in type 1 and type 2 diabetes mellitus. Diabetic retinopathy exhibit features of both micro-vascular and leakage.

1.2.1   Epidemiology

Diabetic retinopathy is a leading cause of visual loss in diabetic patients and the most frequent cause of visual loss among working age persons in developed countries. WHO has estimated that Diabetic retinopathy is responsible for 4.8% of the 37 million cases of blindness through out the world[10].

It develops nearly in all persons with type1 diabetes and in more than 77% of those with type 2 diabetes who survive over 20 years with the disease. DR account for 5-10% blindness in the intermediate economies[10]. PDR affects 5-10% of the diabetic population[11]. Type 1 diabetics are at a particular risk with an incident of about 60% after 30 years. Protective factors for PDR include carotid occlusive disease, posterior vitreous separation, high myopia and optic atrophy[11].

In the Wisconsin epidemiologic study of diabetic retinopathy, 13% of the study population who had had diabetes for less than 5 years and 90% of those who had had it for 10-15 years, had some degree of diabetic retinopathy.[12]

Of those with an onset of 30 years or more (presumed type 2), 20% who were taking insulin and 24% who were not, had some degree of diabetic retinopathy when the duration of diabetes mellitus was less than 5 years[13]. When the duration of diabetes mellitus was 15-20 years, 84%

taking insulin and 53% not taking insulin had some degree of diabetic retinopathy.[14] Sixty percent of people who have had insulin dependent diabetes mellitus for 20 years or more will have proliferative diabetic retinopathy.[15] While more than 12% of those who have had the condition for 30 years or more, will be blind.[16]

Each year in the United States, over 33,000 new cases of diabetic macula oedema, 86,000 cases of proliferative diabetic retinopathy and 12,000-14,000 new cases of blindness occur.[16, 17]

Several studies conducted in Africa have demonstrated that diabetic retinopathy is a major cause of blindness among diabetics as illustrated by selected references below: Nabatanzi et al found the prevalence of DR to be 35.2% in Uganda[18] and Seyoum et al found a prevalence of 37.8% in Ethiopia[19]. Kariuki et al found the prevalence of DR to be 49.8% in patients attending a diabetic eye clinic at KNH, Kenya. 82% of the patients had no previous eye examination and 48.6% of DR patients needed some form of treatment. 32.5% had potentially blinding DR whereas 19.8% had blinding conditions[20]. Githeko et al found the prevalence of DR to be 18.3% whereas 49% of the patients had blinding conditions in his study at rural hospitals in central Kenya[21]. Nkumbe et al found that 30.4% of newly diagnosed diabetics had DR whereas 12.5% had blinding conditions at KNH[22]. Gichuhi et al found that most patients with POAG or ocular hypertension did not show any DR[23]. Wachira et al in his study on diabetic retinopathy in pregnancy in 2006 found that there was no statistically difference in prevalence levels of diabetic retinopathy between pregnant and non pregnant women in the study population[24]

1.2.2 Classification of diabetic retinopathy

Different classifications for diabetic retinopathy exist depending on the purpose. However diabetic retinopathy can be classified into early stage, Non Proliferative Diabetic Retinopathy

(NPDR) and a more advanced stage Proliferative Diabetic Retinopathy (PDR). NPDR is further classified into mild, moderate, severe and very severe NPDR. PDR may be early, high risk or advanced. Macular edema can be present at any level of diabetic retinopathy. The retinal microvascular changes that occur in NPDR are limited to the confines of the retina and do not occur beyond the internal limiting membrane (ILM).

Characteristic findings in NPDR include: microaneurysms, dot and blot hemorrhages, retinal edema, hard exudates, dilatation and beading of retinal veins, intraretinal microvascular abnormalities (IRMA), nerve fiber layer infarcts, arteriolar abnormalities and areas of capillary non perfusion.

NPDR can affect vision through two mechanisms:

1) Intraretinal capillary closure resulting in macula ischemia.

2) Increased retinal capillary permeability resulting in macula edema.

Macula edema is the more common cause of visual loss in diabetic patients. It may manifest as focal or diffuse retinal thickening with or without exudates.

Severe NPDR is defined by the ETDRS by the 4:2:1 rule.

- Diffuse intra retinal hemorrhages and microaneurysms in 4 quadrants.

- Venous beading in 2 quadrants.

- Intraretinal microvascular abnormalities (IRMA) in 1 quadrant.

Proliferative Diabetic Retinopathy (PDR) is characterized by neovascularisation on the optic nerve head (NVD=new vessels at the disc) or along the course of the major vessels (NVE=new vessels elsewhere). Extraretinal fibrovascular proliferation extends beyond the internal limiting membrane and may lead to vitreous or pre-retinal haemorrhage or may contract to cause tractional retinal detachment.

The Diabetic Retinopathy Study (DRS) and Early Treatment Diabetic Retinopathy Study (ETDRS) classified DR into 9 stages: (Appendix No II).

1.2.3 Risk factors

The following risk factors are associated with diabetic retinopathy[11]: duration of diabetes, glycaemic control, blood pressure, contraception and pregnancy, insulin, serum lipids, ethnicity, age at diagnosis, age, nutritional factors, cigarette smoking, alcohol, obesity, and physical activity.

Duration of diabetes is the most important risk factor. In patients diagnosed with diabetes before the age of 30 years, the incident of DR after 10 years is 50% and after 30 years 90%. DR rarely develops within 5 years of the onset of diabetes or before puberty, but 5% of type 2 diabetics have DR at presentation.

Poor methabolic control complements duration as a major risk factor and is greatly relevant to the development and progression of DR[11].

Hypertension if poorly controlled has been associated with worsening of DR and the development of proliferative diabetic retinopathy (PDR) in both type 1 and type 2 diabetics[11].

Nephropathy if severe is associated with worsening of DR. Conversely, treatment of renal disease (e.g. renal transplantation) may be associated with improvement of retinopathy and better response to photocoagulation[11].

Pregnancy is the occasionally associated with rapid progression of DR. Predicting factors include poor control of diabetes, too rapid tightening of control during the early stages of pregnancy and the development of pre-eclampsia and fluid imbalance[11].

Other risk factors that have been shown to be associated with Diabetic retinopathy include: elevated serum lipids, alcohol, anaemia and obesity.

1.2.4 Screening for diabetic retinopathy

Since early diabetic disease is asymptomatic, screening is imperative. The abnormalities that characterize diabetic retinopathy occur in a predictable progression with minor variations in the order of their appearance. The detection of their presence and the extend of involvement of the retina require a careful examination of the retina, preferably with the dilation of the pupils. Generally the larger the pupil, the better the view.

The minimum sensitivity for any screening method to be effective, if it is to be repeated at the recommended interval is 60%. Screening for diabetic retinopathy needs to be community-based in addition to clinic based services and can include a range of examination modalities[11].

1.2.5 Pathogenesis of diabetic retinopathy

Diabetic retinopathy is a microangiopathy affecting the retinal precapillary arterioles, capillaries and venules with features of both microvascular occlusion and leakage.[26] [27] several factors have

9

been implicated in the mechanism(s) for diabetic retinopathy. These include aldose reductase induction, myo-inositol depletion, non enzymatic glycation and free radical damage. The major tissues affected by diabetes are retina, kidneys and nerves- which are all freely permeable to glucose.[28] Growth factors may also influence the progression of diabetic complications by altering the innate glucose regulatory mechanism.

Due to factors that are not yet fully understood, there is reduction in the number of pericytes which are usually wrapped around capillary endothelial cells and are thought to be responsible for the structural integrity of the vessel wall. The reduction in pericytes leads to localized weaknesses in the vessel wall causing saccular pouches of capillary wall distention clinically seen as microaneurysms. It also leads to breakdown of the inner blood-retinal barrier causing plasma constituents to leak into the retina.

Increased vascular permeability and microaneurysms lead to haemorrhage and retinal edema, which may either be diffuse or localized. Chronic localized retinal edema leads to the deposition of hard exudates at the junction of healthy and edematous retina. The hard exudates are composed of lipoproteins and lipid filled macrophages, typically surrounding leaking microvascular lesions, forming a circinate pattern. They may sometimes get absorbed spontaneously into the surrounding healthy capillaries or may get enlarged due to chronic extravasation.

Microvascular occlusion is thought to be due to several factors including: thickening of the capillary basement membrane, capillary endothelial cell damage and proliferation, changes in the red cells leading to defective oxygen transport and increased stickiness and aggregation of platelets. Capillary non perfusion leads to retinal hypoxia which in turn causes retinal ischemia.

Initially, the non perfused area is located in the mid retinal periphery. Retinal hypoxia leads to formation of arteriovenous shunts associated with significant capillary occlusion ("dropout") which run from venules to arterioles referred to as intra-retinal micro-vascular abnormalities (IRMA) and neovascularisation, thought to be formed by a vasoformative substance, elaborated by the hypoxic retinal tissue in an attempt to revascularise hypoxic areas of the retina. This substance promotes neovascularisation.

1.2.6 Treatment of Diabetic Retinopathy

Evidence based treatment reported from several studies indicate that early treatment can reduce the risk for severe visual loss and blindness from PDR by more than 90%[29]

. 1-Early changes of DR do not require treatment.

2-Focal photocoagulation:

This has been shown to be useful in treatment of clinically significant macular oedema (CSME).Lesions amenable to treatment include:

- focal leak greater than 500μ from the center of the macula causing retinal

    thickening or hard exudates.

- focal leaks 300μ-500μ from the centre of the fovea,

- areas of diffuse leakage within the macula area demonstrable on fluorescein
    angiography and avascular areas within the macula area.

Focal photocoagulation has been shown to decrease the risk of moderate visual loss and increase the chance of visual improvement in the eye with macular oedema

3-Pan retinal photocoagulation:

It is not recommended for mild and moderate NPDR if the patient can be carefully followed up[30] [31] [32]. It is indicated in:

- High risk proliferative retinopathy.

- Neovascularisation of the iris.

- Traction retinal detachment and

- Non high risk proliferative retinopathy with attenuating circumstances such as imminent cataract surgery.

4-Vitrectomy:

The goals of this treatment are:

- (i) to remove vitreous opacity and / or fibrovascular proliferation.

- (ii) to allow completion of panretinal laser photocoagulation or direct ciliary body

laser photocoagulation.

- (iii) to relieve retinal traction, tractional displacement or ectopia, traction detachment

by removal or dissection of epiretinal membranes, in cases of rhegmatogenous retinal detachment.

- (iv) to achieve reattachment of retina by closure of breaks and placement of internal

    tamponade.

- (v) to remove posterior hyaloid face in some cases of diffuse macular oedema with

    posterior hyaloid face thickening.

The group comprise some cases of vitreous/ subhyaloid haemorrhage, haemorrhagic ghost cell glaucoma, retinal detachment, severe widespread fibrovascular proliferation, and macular detachment. In vitreous haemorrhage, the surgery is advised at 6 months for

NIDDM if haemorrhage persists. Ealier intervention is however recommended for IDDM[33].

The Diabetes Control and Complications Trial (1983-1993) conclusively demonstrated that intensive control of blood glucose as reflected in measurements of glycosylated haemoglobin reduced he risk for progression of diabetic retinopathy.

2.0 RATIONALE

Diabetic retinopathy is an important public health problem and a significant cause of visual loss and blindness. The magnitude of diabetic retinopathy is expected to rise dramatically in Cameroon.

The magnitude of visual complications of diabetes mellitus in Central Hospital of Yaounde is not known, and this study will establish the nature and magnitude of diabetic retinopathy in order to provide a basis for specific intervention of diabetic retinopathy in Central Hospital of Yaounde.

The need to invest in treatment facilities for DR in the country has not been quantified. (E.g. equipment for laser photocoagulation. fluorescein angiography. vitrectomy. etc...).

3.0 OBJECTIVES

3.1 General objective

To determine the magnitude, pattern and associations of diabetic retinopathy in diabetic patients attending diabetic clinic of Central Hospital of Yaounde, Cameroon (CHY).

3.2 Specific objectives

1. To determine the **prevalence** of Diabetic retinopathy in Diabetic patients attending HCY diabetic clinic

2. To determine the **pattern** of Diabetic Retinopathy by standardised grading using the ETDRS guidelines.

3. To determine the **association** between Diabetic Retinopathy and the following risk factors:

    (i) Duration of diabetes.

    (ii) Glycaemic control.

    (iii) Blood pressure.

4.0   RESEARCH METHODS and MATERIALS

4.1   Study design

This study will be a hospital based cross sectional study in Central Hospital of Yaounde, Cameroon.

4.2   Study population

All diabetic patients with informed consent, attending the diabetes clinics during the study period.

4.3   Study period

The study will be carried out between August and September 2010 over a period of 6 weeks.

4.4   Study setting

Central Hospital of Yaounde is a government Hospital. Located in the centre of Yaounde. It is the second largest hospital in Yaounde with total bed capacity of about 650, and a total catchments area of about 3.5 million. It has a diabetic clinic which runs every day, where averages of 45-65 patients are seen each day.

4.5   Sample size

We will see all the consecutive patients till we reach our sample size (371patients).

16

4.6   Sampling method

In each diabetes clinic day, about 45-65 patients are seen in the diabetic clinic. To cover the sample size in the study period, the researcher has to see at least 60 patients each clinic day for 6 weeks. We will see all the consecutive patients till the sample size is reached. The daily registry will be used and every other patient will be picked from the daily registry for the study.

4.7   Inclusion criteria

All diagnosed diabetic patients aged 12 years and older, attending diabetic clinic during the study period. Diabetic retinopathy rarely develops within 5 years of onset of disease or before puberty.

Informed consent must be given by the patient.

4.8   Exclusion criteria

1. Opaque ocular media not allowing adequate visualization of the fundus for grading of diabetic retinopathy.

2. Diabetic children aged less than 12 years.

3. Those who declined to give consent.

4.9   Materials

1. Structured questionnaire will be used for data collection

2. Tropicamide 1% eye drops

3. Landolt ring, Slit lamp.

4. Indirect binocular ophthalmoscope. 20D and 90D Volk loupes.

5. Retinoscope.

6. Digital BP machine.

4.10 Data Collection Procedure

Patients will be recruited from the diabetic clinic when they come for their visit to the Ophthalmologist. Using the systematic sampling, every other patient will be selected using the daily registry system on the booking order list. Informed written consent will be obtained. Demographic data will be entered on the questionnaire and blood sample for Hba1C will then be obtained. The patient's blood pressure will be measured in sitting position after 5-10 minutes of rest, using an automatic wrist BP machine (Phillips). Hypertension will be defined as systolic BP of $\geq$140mmHg, and a Diastolic Bp of $\geq$90 mmHg[35].

The best corrected visual acuity will be assessed for each eye using Landolt ring at 6 metres, for all patients. Objective and Subjective refractions will be done for those with vision of less than 6/6 by an ophthalmologist or optometrist. Anterior segment examination using a slit lamp (HAAG Streit Bern 90032747, Swiss Made) before dilated fundus examination. The pupils will be then dilated using 1% tropicamide eye drops and posterior segment examined using a binocular indirect ophthalmoscope (HEINE EN50®, Germany) and 20D Volk loupe. after which stereoscopic binocular examination of the fundus using a slit lamp and a 90D Volk loupe. Fundus photography will be done to all patients and cross checking of findings will be done by an ophthalmologist. The diabetic retinopathy will be graded according to the ETDRS classification (Appendix No II). After examination, the findings will be explained (see appendix

18

for questionnaire) to the patient and those requiring medical treatment, Laser or retinal surgery will be treated.

4.11 Data analysis and presentation

After cross checking the questionnaires for any missing entries, data will be coded and entered to SPSS version 16 for analysis. The results of the study will be presented in forms of tables, histograms, and pie charts.

4.12 Ethical considerations

Ethical approval will be obtained from the ethics committee of Central Hospital of Yaoundé and written informed consent will be obtained from the patients or next of kin prior to data collection. The effects of drugs, for example, the temporary effect of tropicamide eye drops on accommodation and any inherent danger of driving will be explained to the patients. All eye drops used will be registered in Cameroon. Confidentiality of patients' records will be strictly observed, and only the researcher will have access to the data. Those requiring treatment will be treated or referred accordingly.

5.0 RESULTS

This study involved interviews and clinical examination of 371 diabetic patients out of which 54.7% were females and the overall mean age was 59.2 (10.9) years. Majority of the patients were in the age groups of 50-59 years (39.4%) and 60-69 years (39.4%).

Table 1: Demographic characteristics (n=371)

Variable	Frequency (%)
Sex	
Male	168 (45.3)
Female	203 (54.7)

Figure 1: Distribution of patients by age, (n=371)
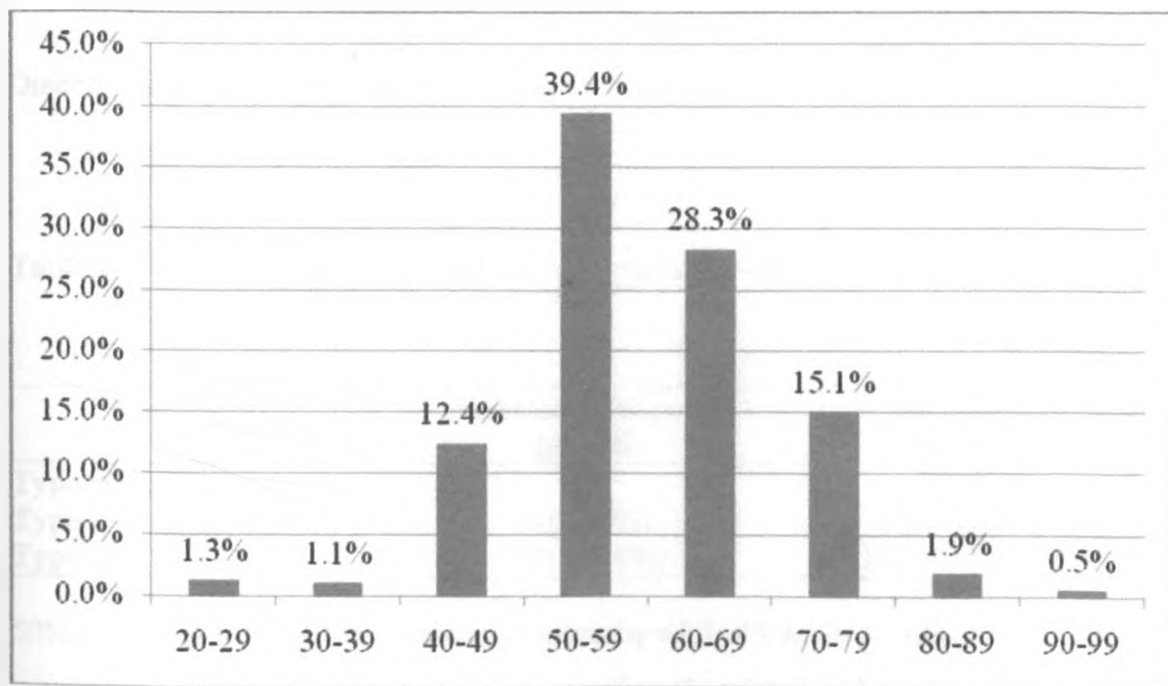
The mean age was found to be 59.2 (SD=10.9) years.

20

Figure 2: Prevalence of Diabetic retinopathy (n=371)
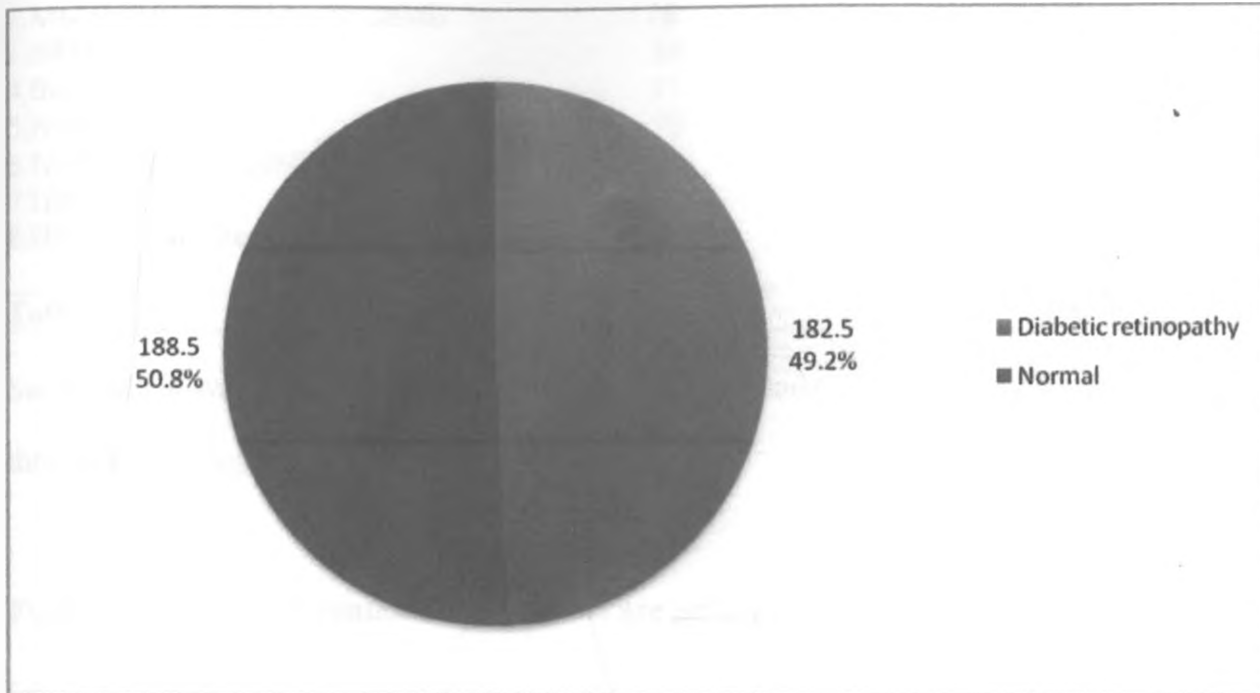
Diabetic retinopathy was identified among 49.2% of the patients.

Table 2: Type of diabetes and diabetic retinopathy (n=371)

	Diabetic retinopathy (n=185)	Normal (n=186)	P value
Type of diabetes			
Type 1	6 (50.0%)	6 (50.0%)	0.992
Type 2	179 (49.9%)	180 (50.1%)	

50% (6patients) of type I had diabetic retinopathy while 49.9(179 patients) of the type II had retinopathy. Diabetic retinopathy was not significantly related to the type of diabetes in this population (p=0.992).

21

Table 3: Classification of diabetic retinopathy in the most affected eye (n=371)

Diabetic retinopathy	Frequency	Percent
0.Normal	186	50.1%
1.Minimal NPDR	72	19.4%
2.Macula edema that is not CSME	18	4.9%
3.NPDR with CSME	30	8.1%
4.Severe NPDR	12	3.2%
5.NHRPDR	23	6.2%
6.NHRPDR with CSME	20	5.4%
7.HRPDR	8	2.2%
8.HRPDR not amenable to photocoagulation	2	0.5%
Total	**371**	**100%**

Severe NPDR was found in 12 (3.2%), PDR in 53(14.3%) and CSME in 30 (8.1%) and vision threatening retinopathy in 101(27.3%).

Figure 3: Modality of treatment the patients are getting (n=371)
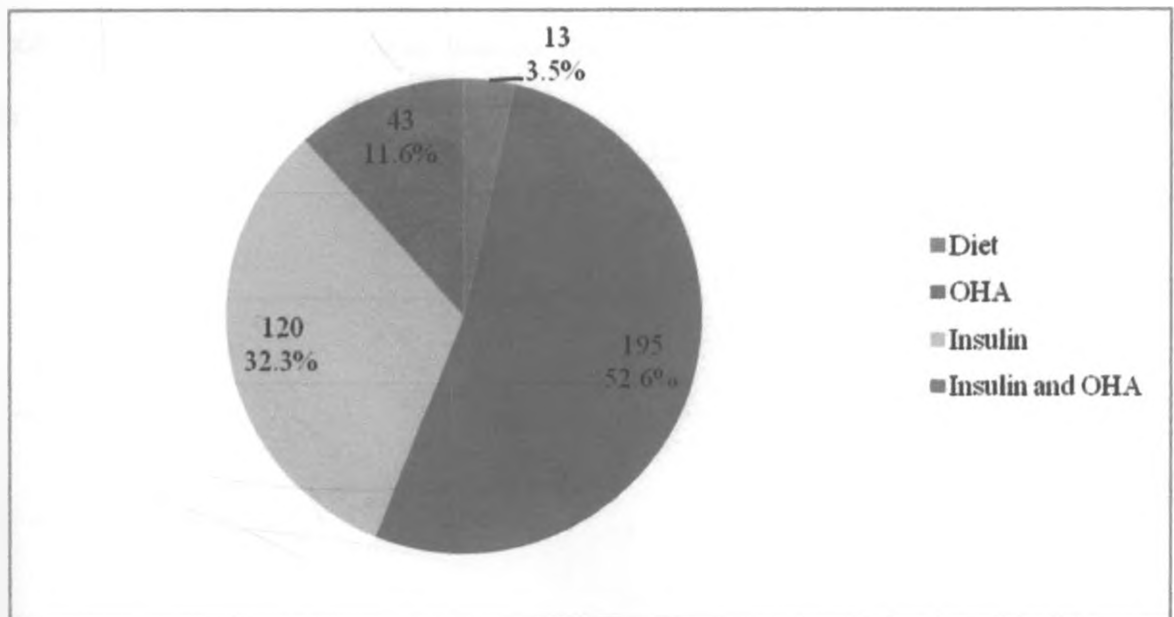
All the patients were on diabetic diet.13 patients (3.5%) were on diabetic diet only.195 (52.6%) were also on oral hypoglycemic agents (OHA), 120 (32.3%) were on insulin and 43 (11.6%) had been on both OHA and insulin between the time of diagnosis and time of examination.

Figure 4: Mode of treatment and diabetic retinopathy (n=371)
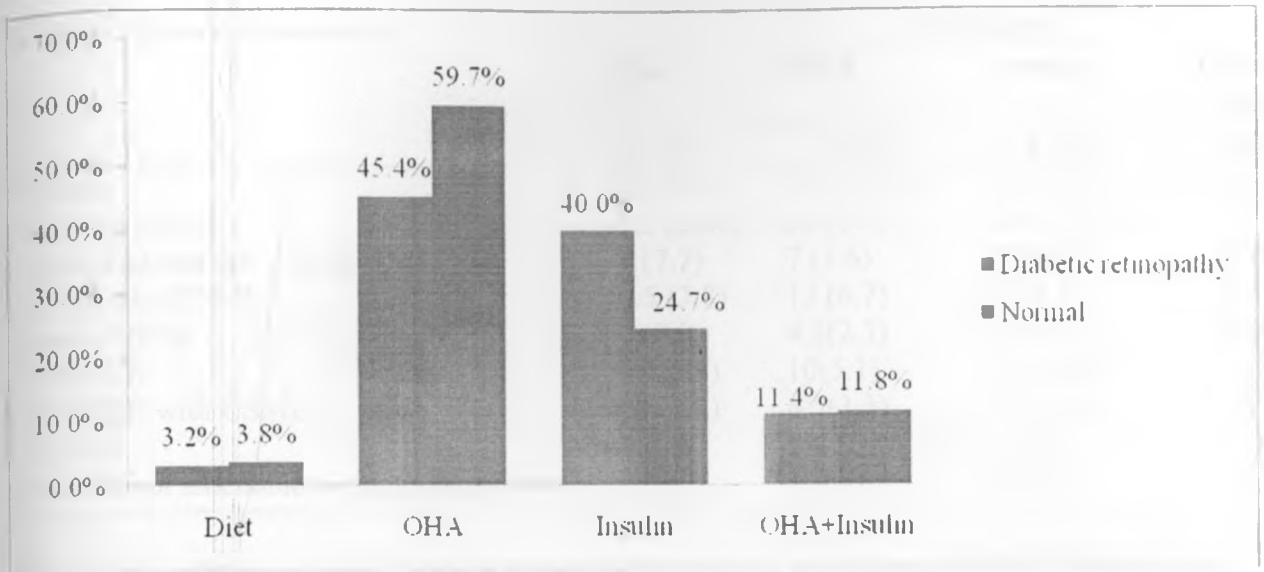
Diabetic retinopathy was present in 40.0% (74 patients) on insulin, 45.4% (84 patients) of those

on OHA, 3.2% (6 patients) of those on diet only and 11.4% (21 patients) of the patients who had

been on both insulin and OHA. The mean duration of insulin treatment was 7.5 years, that of

OHA was 7 years and that of diet was also 7.0 years.

Table 4: Mode of treatment and grade of diabetic retinopathy (n=371)

Grade of diabetic retinopathy	treatment			
	Diet	OHA	Insulin	Insulin and OHA
	n (%)	n (%)	n (%)	n (%)
0.Normal	7 (53.8)	112 (57.4)	47(39.2)	22 (52.3)
1.Minimal NPDR	3.5 (26.9)	38 (19.5)	29.5 (24.6)	11 (25.6)
2.Macula edema that is not CSME	1 (7.7)	7 (3.6)	8 (6.7)	0 (0.0)
3.NPDR with CSME	0.5 (3.8)	13 (6.7)	8 (6.7)	5.5(12.8)
4.Severe NPDR	0 (0.0)	4.5(2.3)	7.5(6.3)	0 (0.0)
5.NHRPDR	0.5(3.8)	10(5.1)	12(10.0)	1(2.3)
6.NHRPDR with CSME	0.5(3.8)	6.5(3.3)	7.5(6.3)	2(4.7)
7.HRPDR	0 (0.0)	2(1.0)	1(0.8)	1(2.3)
8.HRPDR not amenable to photocoagulation	0 (0.0)	1(0.5)	0(0.0)	1(1.2)

Diabetic retinopathy was significantly related to the mode of treatment ($p<0.001$).Patients who

had been on both insulin and OHA had the more severe grades of diabetic retinopathy.

Table 5: Sex of patient and diabetic retinopathy (n=371)

	Diabetic retinopathy (n=185)	Normal (n=186)	P value
Sex			
Male	91 (54.2%)	77 (45.8%)	0.132
Female	94 (46.3%)	109 (53.7%)	

Diabetic retinopathy was present in 54.2% of the male patients and 46.3% of the female patients.

There was no significant relationship between the sex of patient and diabetic retinopathy

($p>0.001$).

Table 6: Duration in year of diabetes and diabetic retinopathy (n=371)

	Diabetic retinopathy (n=185)	Normal (n=186)	P value
Mean duration of diabetes	13.0	6.9	<0.001

There was a significant relationship between the duration of diabetes mellitus and development of diabetic retinopathy (p<0.001).

Table 7: Duration of diabetes and Grade of diabetic retinopathy (n=371)

Grade of diabetic retinopathy	Mean duration in years
0.Normal	6.9
1.Minimal NPDR	11.0
2.Macula edema that is not CSME	10.5
3.NPDR with CSME	13.4
4.Severe NPDR	18.5
5.NHRPDR	16.6
6.NHRPDR with CSME	16.0
7.HRPDR	15.4
8.HRPDR not amenable to photocoagulation	18.0

The mean duration of diabetes was 9.9 years(SD=7.7) among all the patients and the patients diagnosed with retinopathy were found to have had diabetes for a significantly longer duration (13 years) compared to the normal group whose duration of diabetes was 6.9 years, P<0.001.

Table 8: Grading of VA (BCVA) diabetic retinopathy by WHO classification (n=742)

Visual acuity	Diabetic retinopathy (n=365)	Normal (n=377)	P value
6/6 - 6/18	258 (70.7%)	352 (93.4%)	<0.001
<6/18 - 6/60	80 (22%)	22 (5.8%)	
<6/60 - 3/60	2 (0.5%)	0 (0.0%)	
<3/60	25 (6.8%)	3 (0.8%)	

Eyes with diabetic retinopathy were found to have poorer visual acuity than those without diabetic retinopathy and it was statistically significant (p<0.001). 107 eyes (29.3%) had visual loss while 82 eyes (22.5%) having visual impairment and 28 (7.6%) being blind.

Table 9: WHO grading of VA (BCVA) and grade of diabetic retinopathy (n=742)

Grade of diabetic retinopathy	Visual acuity			
	6/6 - 6/18 n (%)	<6/18 - 6/60 n (%)	<6/60 - 3/60 n (%)	<3/60 n (%)
0.Normal	352 (93.4)	22 (5.8)	0 (0.0)	3 (0.8)
1.Minimal NPDR	143 (89.4)	17 (10.6)	0 (0.0)	0 (0.0)
2.Macula edema that is not CSME	21 (61.8)	10 (29.4)	0 (0.0)	3 (8.8)
3.NPDR with CSME	27 (48.2)	24 (42.8)	0 (0.0)	5 (9.0)
4.Severe NPDR	20 (83.3)	3 (12.5)	0 (0.0)	1 (4.2)
5.NHRPDR	27 (57.4)	12 (25.5)	0 (0.0)	8 (17.0)
6.NHRPDR with CSME	14 (42.4)	15 (45.5)	0 (0.0)	4 (12.1)
7.HRPDR	0 (0.0)	5 (62.5)	1 (12.5)	2 (25.0)
8.HRPDR not amenable to photocoagulation	0 (0.0)	0 (0.0)	1 (33.3)	2 (66.7)

The severity of diabetic retinopathy was co-related with visual outcome. 25 eyes were blind from diabetic retinopathy and 3 eyes with no diabetic retinopathy were blind from glaucoma.

Table 10: Diabetic retinopathy and HbA1c levels

	Diabetic retinopathy (n=185)	Normal (n=186)	P value
HbA1c	9.4	6.7	<0.001

A total of 371 patients (742 eyes) had they HbA1c levels measured. The mean level for patients

with retinopathy was higher (9.4%) than those of patients without retinopathy (6.8%) and it was

statistically significant (p<0.001).

Table 11: HbA1c levels and grading of diabetic retinopathy (n=371)

Grade of diabetic retinopathy	Mean HbA1c
0.Normal	6.8
1.Minimal NPDR	8.8
2.Macula edema that is not CSME	9.9
3.NPDR with CSME	9.6
4.Severe NPDR	10.0
5.NHRPDR	10.2
6.NHRPDR with CSME	10.0
7.HRPDR	9.1
8.HRPDR not amenable to photocoagulation	11.0

Diabetic retinopathy was significantly related to the level of HbA1c (p<0.001). Severity of

retinopathy increased with increase level of HbA1c.

Table 12: Fasting blood sugar and diabetic retinopathy

	Diabetic retinopathy (n=185)	Normal (n=186)	P value
Fasting blood sugar	10.6	9.4	0.014

A total of 371 patients (742 eyes) had their FBS levels measured. The mean level for patients with retinopathy was higher (10.6 mmol/l) than those of patients without retinopathy (9.4 mmol/l) and it was statistically significant (p=0.001).

Table 13: Fasting blood sugar levels and grading of diabetic retinopathy (n=371)

Grade of diabetic retinopathy	Mean FBS
0.Normal	9.4
1.Minimal NPDR	10.5
2.Macula edema that is not CSME	10.2
3.NPDR with CSME	12.0
4.Severe NPDR	10.9
5.NHRPDR	10.2
6.NHRPDR with CSME	9.6
7.HRPDR	11.7
8.HRPDR not amenable to photocoagulation	12.3

There was a general increase in level of FBS and severity of retinopathy but FBS levels did not correlate very well with the severity of diabetic retinopathy.

Figure 5: Correlation between the mean HbA1c, mean FBS levels and severity of diabetic retinopathy
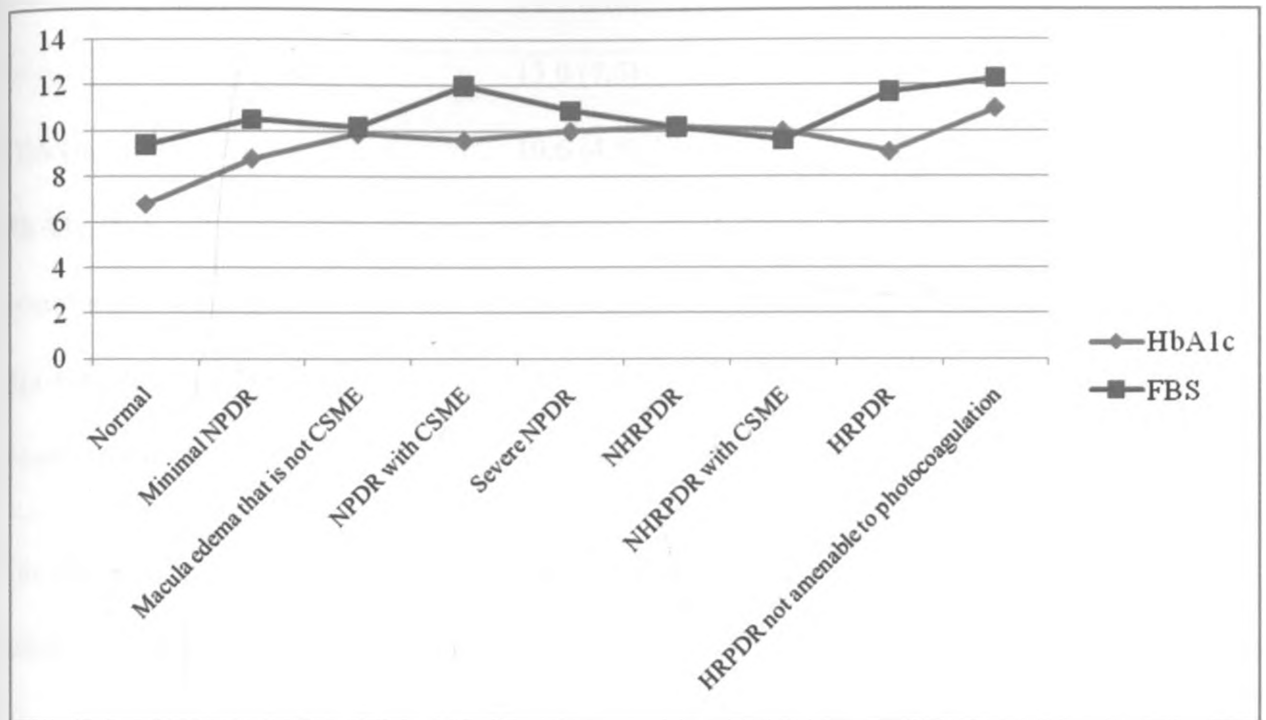
The severity of retinopathy correlated better with HbA1c levels as compared to FBS levels.

Table 14: Association of diabetic retinopathy with selected variables

Variable mean (SD)	DR status		p-value
	Yes	No	
Mean age (years)	59.3 (9.8)	59.1 (12.0)	0.879
Duration of DM (years)	13.0 (7.5)	6.9 (6.6)	<0.001
FBS (mmol/l)	10.6 (4.9)	9.4 (5.0)	0.014
HbA1c (%)	9.4 (2.5)	6.7 (2.6)	<0.001
Systolic BP (mmHg)	152.3 (27.5)	141.8 (22.1)	<0.001
Diastolic BP (mmHg)	85.1 (13.8)	82.3 (14.0)	0.057
Nephropathy	21 (11.4)	0.0(0.00)	0.006

The above table shows that there is statistically significant association between diabetic retinopathy and duration of diabetes, HbA1c, systemic BP.

Table15: Previous eye examination by an ophthalmologist (n=371)

	Number of Patients	Percent
Yes	330	88.9%
No	41	11.1%

Among 371 patients examined. 330 (88.9%) had previous eyes examination done by an ophthalmologist whereas 41(11.1%) did not benefit from the previous eyes examination.

6.0 DISCUSSION

Diabetic retinopathy is increasingly becoming a major cause of blindness in the world in the age group of 20-60 years[36] and the incidence of diabetes mellitus and its complications are rising in developing countries[37]. Measures to reduce visual disabilities and improve quality of life of those affected have become important, and so is the need for information on diabetic retinopathy in these countries.

The magnitude of diabetic retinopathy in Cameroon has not been described. This study is therefore the first to describe the prevalence, pattern and associations of diabetic retinopathy in patients attending diabetic clinic in the capital Yaoundé and also will be helpful in providing baseline data for planning and organizing a diabetic clinic in the eye unit.

Three hundred and seventy one (371) patients were examined out of which 203 (54.7%) were female while 168 (45.3%) were male giving a sex ratio of Male: Female of about 1:1.2.This finding is probably due to the fact that women are usually more affected by diabetes than men; but there was no association between sex of the patient and occurrence of diabetic retinopathy.

The prevalence of diabetic retinopathy in diabetic patients attending the medical clinic at the CHY was found to be 49.2%. The black African diabetic has higher prevalence of diabetic retinopathy as illustrated by different studies done in Africa; Kariuki et al[20] in KNH in 1999 found this prevalence to be 49.8 % and Guadie et al in 2009 in Jimma Hospital in Ethiopia found 41.4% ; these two prevalence are relatively similar to the prevalence of our study. However in rural Kenya, a study done by Githeko et al among diabetic attending peripheral health institutions found a prevalence of 18.3% which is much lower than our study[21] but similar to another study done in India where the prevalence of diabetic retinopathy in an urban population older than 40 years was 18%[38].

31

The high prevalence of diabetic retinopathy in our study could partly be explained by the fact that CHY is a referral center where those with long term disease and complications tend to be followed. Another factor which could explain partly the high prevalence of diabetic retinopathy is the fact that 49.2% of patient had their HbA1c and FBS poorly controlled.

Severe non proliferative diabetic retinopathy (NPDR) was found in12 (3.2%) of 371 diabetic patients while proliferative diabetic retinopathy (PDR) was found in 53 (14.3%) and clinically significant macula edema in 30 (8.1%) patients. Vision threatening retinopathy (defined as presence of PDR or macula edema) was present in101 (27.3%) patients and had had laser photocoagulation done in CHY; the prevalence of vision threatening retinopathy is similar to that was found in Luganville, Vanuatu (22.1%) by Tasanee et al[39] but higher than the study done in United States among adults above 40 years with DR which was 8.2%[40].

The prevalence of PDR found in this study (14.3%) is similar to that was found in Kenya (12.1%) by Kariuki[20] et al in 1999 but higher than what was found in Ethiopia (1.7%) by Seyoum[19] et al in 2001. These higher rates could be partly explained by the fact that most diabetic patients had poor sugar control and elevated HbA1c.

Of the 49.8% (185) with diabetic retinopathy, 46.3% (94) were females and 54.2% (91) were males, which was not statistically significant (p-value=0.132). This finding is consistent with other studies which have reported no association between diabetic retinopathy and sex.[20, 41, 42]

Diabetic retinopathy was found associated with higher mean age of 59.2 years where those with no diabetic retinopathy had a mean age of 50.1 years. This finding is similar to the study done in Oman by Dandona et al[43] who found that for duration of diabetes mellitus less than 10 years the

prevalence was 7%, and between 10-14 years it was 26% and beyond 15years it was 63% However this finding could be associated with longer duration of diabetes in the older population.

Among type I diabetes, 50% (6patients) had diabetic retinopathy while 49.9% (179patients) of type II had diabetic retinopathy (in this study, type I diabetes was defined as any patient whose age was ≤ 40 years and type II diabetes as anyone >40 years at the time of diagnosis). Type I diabetes has been associated with more frequent and more severe ocular complications.[11] However in our study, patients with type II had high prevalence of diabetic retinopathy (97.7%) compared to those with type I diabetes (3.3%). This may partly be explained by the relatively shorter duration of the disease in type I patients with a mean duration of 9.9 years (SD=9.3) compared to those with type II diabetes who had mean duration of 10.2 years (SD=7.7) (p-value=0.909). The Wisconsin Epidemiology Study of Diabetic Retinopathy (WESDR)[12] similarly found that type I diabetic patients who had had diabetes for < 5 years duration had a prevalence of 13% compared to 20-24% in those with type II diabetes who had had the disease for the same duration. Type I diabetic patients in this study were fewer than would be expected going by the type I: II ratio of 1-2:10 reported in the literature. This may be partly explained by the fact that the morbidity and mortality of type I is relatively higher leading to the low numbers of type I patients seen.

Duration of diabetes is the most important predictor for development of diabetic retinopathy. In our study, patients with diabetic retinopathy had a statistically significant higher mean duration of diabetes 13 years compared to 6.9 years in those with no diabetic retinopathy (p-value<0.001).

A study done in Southern India shown similar association between diabetic retinopathy and duration of diabetes, in this study the prevalence of diabetic retinopathy was 7% in individuals with a short duration of diabetes (<10 years), but among those whose duration of diabetes was 10-14 years, the prevalence of diabetic retinopathy was 26% and 63% in those with 15 years or more duration. [44]

It has been observed that long term glycaemic control plays an important role in delaying the onset and lowering down the progression of diabetic retinopathy. This protective effect has been investigated in type I[16, 45] and type II diabetic patients.[46]

In our study a total of 371 patients (742 eyes) had they HbA1c levels measured. The mean level of HbA1c for patients with diabetic retinopathy was higher (9.4%) as compared to 6.8% for patients who had no diabetic retinopathy (p-value<0.001). The highest level of HbA1c (11.0%) was associated with the most severe case of retinopathy (grade 9). The prevalence of retinopathy increased with increase in the mean HbA1c values even after controlling for duration of diabetes (p-value<0.001).

The DCCT has confirmed that measuring glycohemoglobin in blood is an excellent tool for the long term control of the glycaemic state; because the percentage of glycohemoglobin in human blood depends on the concentration of glucose, the duration of glucose exposure to the hemoglobin and the turnover of erythrocytes; so that the higher the glucose concentration and the longer the exposure time, the highest the percentage of HbA1c. [47]

The fasting blood sugar has been measured also in all our study subjects (371); although measurement of glycaemic control is best demonstrated by measuring the glycosylated hemoglobin (HbA1c), in this study measurement of fasting blood sugar showed that the mean level for patients with retinopathy was higher (10.6 mmol/l) than those of patients without

34

retinopathy (9.4 mmol/l) and it was statistically significant (p=0.001) as has been previously shown in other studies. [32]However, the fasting blood sugar levels did not correlate as well as the HbA1c levels to the severity of diabetic retinopathy. FBS varies with time of last meal, the type of meal and time of last treatment, which are not standardized.

In our study the mean systolic and diastolic blood pressures in the study population were 147.0 mmHg and 83.7mmHg respectively. Patient with diabetic retinopathy had higher mean systolic (152.3mmHg) and diastolic (85.1mmHg) as compared to those with no diabetic retinopathy who had mean systolic (141.8mmHg) and diastolic (82.3mmHg) and it was statistically significant (p-value<0.001).These finding correlate with other studies which have shown that higher systolic and diastolic blood pressure are associated with diabetic retinopathy. [12, 41]

Nephropathy was found associated significantly with diabetic retinopathy. Among 185 patients with diabetic retinopathy, 21 (11.4%) patients had nephropathy diagnosed by renal specialist. The chances of diagnosing retinopathy among patients increased with the presence of nephropathy OR 3.3 (1.4-7.9), P=0.006. This has also been described in other study done in India by J Prakash, M Lodha, SK Singh, Rubina Vohra, R Raja, Usha who found the severity of Diabetic Retinopathy associated with Nephropathy. [48]

In our study the mode of treatment of was associated with presence or absence of diabetic retinopathy. Diabetic retinopathy was found in 45.4% (84patients) on OHA (mean duration: 7 years), followed by 40.0% (74patients) of those on insulin (mean duration: 7.5 years) and by 11.4% (21patients) of those on both insulin and diet only (mean duration: 7 years) and 3.2% (6patients) was on diet only. It was found that more type II diabetics were put on insulin; that patients on insulin were having higher HbA1c and FBS than those on OHA. Patients on insulin

had poor glycaemic control; this may be explained by the fact that these patients had failed to be controlled on OHA and were put on insulin as last resort. Patients whose mode of treatment was OHA, had higher prevalence of diabetic retinopathy (45.5%) and were followed by those on insulin only (40%) and was significant (p-value<0.001). This could be a reflection of the severity of disease and hence the need for combination therapy. This association between diabetic retinopathy and mode of treatment has also been previously described in other studies.[20]

Severity of diabetic co-related with the visual outcome with 66.7% of grade 9 patients being blind and 33.3% having visual impairment; and 75% of grade 8 patients having visual impairment and 25% being blind. Patients with CSME (grade 3 and 6) had high prevalence of visual loss (grade 3 was 50% and grade 6 was 57.6%). This supports earlier observations that vision loss is a common complication in diabetics with diabetic retinopathy. Previous study done by Kariuki et al in KNH came to the similar findings.[20]

Among all examined study subjects, 330 (88.9%) have previous eye examination by an ophthalmologist and only 41 (11.1%) did not. This may partly be explained by the fact that CHY is a referral center where all the diabetic patients benefit from eye examination by the ophthalmologist at the first visit.

Study limitations

- Disease duration was self-reported. This introduces errors due to recall failure. Further, type II disease may be present for variable durations before becoming manifest and duration of disease from time of diagnosis is not necessarily the duration from onset resulting in an underestimate.

- Cost constrictions of HbA1c did not allow going beyond 371 patients.

7.0 CONCLUSIONS

1. The prevalence of diabetic retinopathy was high (49.2 %.) while visually threatening diabetic retinopathy was found to be 27.3%.

2. Longer duration of diabetes mellitus, poor glycaemic control, hypertension and nephropathy were significantly associated with diabetic retinopathy.

3. The majority of patients (88.9%) had previous eye examination before this study.

4. Patients with diabetic retinopathy were more likely to have poor vision compared to those without diabetic retinopathy.

5. Majority of patients were poorly controlled (mean FBS10.6 mmol/l and mean HbA1c 9.4%) and were not on dietary management for control of their diabetes.

8.0 RECOMMENDATIONS

1. There should be a close collaboration between the medical diabetic clinic and the ophthalmic unit in order to establish a good screening system for early detection of diabetic retinopathy.

2. There should be a deliberate effort to emphasize the fact that blindness due to diabetic retinopathy is potentially preventable in the majority of the patients.

3. There is need for treatment facilities for DR (equipment and personnel to perform laser photocoagulation, fluorescein angiography and victrectomy) to be extended in other parts of the country.

4. Regular measurement and reduction of cost of HbA1c for an excellent long term control of glycaemic state should be introduced in the medical units.

5. Encourage and investigate the role of the private sector and mass media in combating this epidemic.

6. This study was done in an urban setting, it is thus necessary to have a similar study in the rural setting to determine the magnitude of this problem in such a population.

9.0 ACKNOWLEDGEMENTS

10.0 REFERENCES

1. Khatib, OMN. Guidelines for the prevention, management and care of diabetes

   mellitus. EMRO Technical Publication series. WHO. 20006:**32**: 13-14

2. IDF. Diabetes atlas. 2nd ed. Brussels, International Diabetes Federation.2003.

3. WHO/IDF. *Diabetes action now*: an initiative of the World Health Organization and the

   International Diabetes Federation. 2004:6.

4. WHO. *Facts and figures on diabetes*. WHO African region. 2008 Geneva: 2.

5. http://www.worlddiabetesfoundation.org.

6. Clinical practice recommendations 2005. *Diabetes care*. 2005; **28 (suppl. 1)**.

7. WHO. Diet, nutrition and the prevention of chronic diseases: Report of a joint

   WHO/FAO expert consultation. Geneva, World Health Organization.2003 (WHO

   Technical Report series, No. 916).

8. WHO. Report on the regional consultation on diabetes prevention and control; Teheran

   Islamic republic of Iran, 2-5 February 2003. Cairo Egypt. World Health Organization

   regional office for Eastern Mediterranean, 2003 (WHO-EM/NCD/035/E/L).

9. Kunimoto D.Y et al. *The Wills eye manual*; office and emergency room diagnosis

   treatment of eye disease. 4th ed. 2004. Diabetes Mellitus **13.2**: 324-328.

10. Resnikoff S et al. Global data on visual impairment in the year 2002. *Bulletin of the

    World Health Organization*. 2004.**82**: 844-851.

11. Kanski JJ. Diabetic retinopathy. *Clinical ophthalmology*. Butterworth Heinemann 5th eds. 2003: 439-455.

12. Klein R, Klein BE, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiological Study of Diabetic Retinopathy III. Prevalence and Risk of diabetic retinopathy when age at diagnosis is 30 or more years. *Arch Ophthalmol* 1984: 102: 527-532.

13. Dandona L, Dandona R, Naduvilath TJ, McCarty CA, Rao GN. Population based assessment of diabetic retinopathy in an urban population in Southern India. *Br J Ophthal* 1999; 83: 937-940.

14. Klein R, Moss SE, Linton KL. The Beaver Dam eye study. Retinopathy in adults with newly discovered and previously diagnosed diabetes mellitus. *Ophthalmology* 1991: 99: 58-62.

15. Broadbent DM, Scott JA, Vora JP, Harding SP. The Liverpool diabetic eye study. Prevalence of diabetic eye disease in an inner city population. *Eye*. 1999 Apr; 13(pt 2): 160-165.

16. Klein R, Klein BE, Moss SE, Cruikshanks KJ. The Wisconsin epidemiologic study of diabetic retinopathy: XVII. The 14 year incidence and progression of diabetic retinopathy and associated risk factors in type 1 diabetes. *Ophthalmology* 1998: 105: 977-986.

17. Klein R. Estimates prepared for the diabetes 2000 program, based upon the Wisconsin Epidemiological Study of diabetes retinopathy. San Francisco California. *American academy of ophthalmology*, 1989.

18. Nabatanzi C et al. Prevalence of diabetic retinopathy in Uganda. M.Med dissertation. Makerere University. 1999. (unpublished)

19. Seyoum B et al. Retinopathy in patients in Tikur Ambessa Hospital diabetic clinic. *Ethiop Med J.* April 2001. **392**: 123-131.

20. Kariuki MM, Kollmann KHM, Adala HS. The prevalence, pattern and associations of diabetic retinopathy among black African diabetics attending the medical diabetes clinic at the Kenyatta National Hospital. *German Medical Science*; 2004. DOC **04** dog SO 08.06

21. Githeko A.K. Kollmann KHM. Adala HS. Prevalence, pattern and risk factors of diabetic retinopathy among patients attending rural health institutions in central Kenya. *EAJO.* 2008. Vol 13 No 2: 4-10

22. Nkumbe E, Kollmann KHM, Gackle HC. Fundus findings in black Africans with newly diagnosed diabetes mellitus. German Medical Science; 2004. DOC **04** dog SO 08.06

23. Gichuhi S, Kollmann KHM, Choksey PV. Prevalence of primary open angle glaucoma among black African patients with diabetes mellitus. *German Medical Science*: 2004. DOC **04** dog SO 08.06

24. Wachira J.W, Kollmann KHM, Kimani K. Diabetic retinopathy in pregnant and non-pregnant women of child bearing age in Nairobi, Kenya. M.Med dissertation. University of Nairobi. 2006 (unpublished)

25. American Diabetic Association. Screening for type 2 diabetes. *Diabetes Care* 2004; **27(suppl 1):** 511-514.

26. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes care* Vol. 21; supplement 1.

27. Robert Frank. On the pathogenesis of diabetic retinopathy. A 1990 update. *Ophthalmology* 1991 May; 98 **5:** 586-593.

28. Van Leiden HA, Dekker JM, Moll AC, Nijpels G, et al. Blood pressure, lipids and obesity are associated with retinopathy: the Hoorn study. *Diabetes care* 2002; **25:** 1320-1325.

29. Prevention of blindness from diabetes mellitus: Report of the WHO consultation in Geneva, Switzerland, 9-11 November 2005.

30. Early Treatment of Diabetic Retinopathy Study Research Group: report number 1 Photocoagulation for diabetic macular edema. Arch. Ophthalmol. 1985 Dec 103 **12:** 1796 – 806.

31. Early Treatment of Diabetic Retinopathy Study Research Group: report number2. Treatment techniques and clinical guidelines for photocoagulation of diabetic macular edema. Ophthalmology 1987 July; 94 **7:** 761 – 74.

32. Early Treatment of Diabetic Retinopathy Study Research Group: report number 9. Early photocoagulation for diabetic retinopathy. Ophthalmology 1991 May; 98 **(5 supp):** 766 – 85.

33. Royal college of ophthalmologists. Guidelines for diabetic retinopathy 1997.

34. Moukouri EDN, Mc Moli T, Nouedoui C. Les Aspects Epidemiologiques de la Retinopathie Diabetic a Yaounde. Médecine d'Afrique Noire: 1992, 39 (5).

35. www.medterms.com

36. Thylefors B, Negrel AD, Parajasegaram R, Dadzie KM. Global data on blindness. Bull World Health Organ 1995; 73: 115-121.

37. International Diabetes Federation (IDF), Diabetes Atlas, 2nd and 3rd edition, update Nov.2006.

38. Ophthalmology.2009 Feb; 116(2): 311-8 Epub 2008 Dec 12.

39. Br. J Ophthalmol 2007 91: 415-419. Doi : 10.1136/bjo.2006.104174

40. Arch Ophthalmol.2004; 122(4): 552-563

41. Kohner EM, Aldington SJ, Stratton IM, Manley SE, Hollman RR, Mathews DR, Turner RC. United Kingdom prospective diabetes study 30, diabetic retinopathy at diagnosis of non insulin dependent diabetes mellitus and associated risk factors. Arch Ophthalmol. 1998 Mar; 116(3): 297-303.

42. Segal P, Treisher G, Yalon M, et al. The prevalence of DR: effect of sex, age, duration of disease and mode of therapy. Diabetes Care, 1983; 6: 149-151.

43. R Khandekar, J Allawatii, A J Mohammed, A Al Raisi et al. Diabetic Retinopathy in Oman: a hospital based study. Br J Ophthalmol 2003; 87: 1061-1064.

44. Narendran V, John RK, Raghuram A et al. Diabetic retinopathy among self reported diabetics in southern India: a population based assessment. Br J Ophthalmol 2002: 86: 1014-1018.

45. The DCCT Research group. The effect of intensive treatment of Diabetes on the development and progression of long term complications in insulin dependent diabetes mellitus. N Engl J Med 1993; 329 977-986.

46. United Kingdom prospective diabetes study (UKPDS) group. Intensive blood glucose control with sulphonyureas or insulin compared with conventional treatment and risk of complications in patients with type II diabetes (UKPDS 33). Lancet. 1998; 352: 832-853.

47. Goldstein, D.E., Little, R.R.. Wiedmeyer, H.M.England. J.D.. Rohlfing. C.L.. Wilke. A.L: Is glycohemoglobin testing useful in diabetes mellitus? Lessons from the DCCT. Clin. Chem. 40: 1637-40, 1994.

48. JAPI.VOL.55.June 2007

11.0 APPENDICES

11.1 Appendix I: Diagnostic Criteria

WHO diagnostic criteria (2006) for diabetes are as follows:

<u>Diabetes</u>

Fasting Plasma glucose	$\geq$7.0mmol/l (126mg/dl)
Or 2-hour plasma glucose*	$\geq$11.1mmol/l (200mg/dl)

<u>Impaired glucose tolerance (IGT)</u>

Fasting Plasma glucose	<7.0mmol/l (126mg/dl)
And 2 hour plasma glucose*	$\geq$7.8mmol/l and <11.1mmol/l
	(140mg/dl and 200mg/dl).

<u>Impaired fasting glucose (IFG)</u>

Fasting plasma glucose	6.1-6.9mmol/l (110 mg/dl- 125mg/dl)
2 hour plasma glucose*	<7.8mmol/l (140mg/dl)

* Venous plasma glucose 2 hours after ingestion of 75g oral glucose load (or 1.75g/kg in children). If 2-hour plasma glucose is not measured, status is uncertain as diabetes or IGT cannot be excluded.

11.2   Appendix II: ETDRS classification

ETDRS GRADING OF DIABETIC RETINOPATHY

---

0. Normal

1 Minimal non proliferative diabetic retinopathy (NPDR).

2. Non proliferative diabetic retinopathy without macula edema.

3 NPDR with macular edema that is not clinically significant.

4. NPDR with clinically significant macular edema (CSME)

   CSME is defined by the ETDRS as the following:

5. Severe NPDR (pre-proliferative).

6. Non high risk proliferative diabetic retinopathy without CSME.

7. Non high risk proliferative diabetic retinopathy with clinically significant macula edema. (NHRPDR with CSME).

8. High risk proliferative diabetic retinopathy (HRPDR).

9. High risk proliferative diabetic retinopathy not amenable to   photocoagulation.

11.3 Appendix III: Questionnaire

<u>Questionnaire</u>   Date................

   A-General information

Name.. IP/OP No..............

1-Age (in years)................

2-Sex   a) Male   b) Female

3-Duration of diabetes in years........................

4-Type of diabetes   a) Type 1   B) Type 2

5-Diabetes Treatment: Duration.

   a) Diet...............................   ....

   b) O.H.A...................................   ....

   c) Insulin.........................   ....

6-Any awareness about the effect of DM on the eyes   a) Yes   b) No

7- Any visual complaints a) Yes   b) No

If Yes   a) Reading problem   b) Poor distance vision

 c) Others (specify).. ....

8. Any previous eye examination for the diabetes (a) yes   (b) No

9-Concomitant illness   a) HTN   b) Nephropathy c) Glaucoma   d) Cigarette Smoking   e)
Others_____

Laboratory: Fasting blood sugar.............................

B/P (mmhg)..

B- Ocular Examination,

1. Visual Acuity   OD

                               OS

2. Refraction (when necessary)

                        _OD_   _OS:_

3-Adnexal and anterior segment exam

(Abnormal findings only)

5-Posterior Segment   ....

(Thorough Dilated Exam)

49

Final assessment of Diabetic Retinopathy

DIABETIC RETINOPATHY	RE	LE
0.Normal 1 minimal NPDR		
2. NPDR with macula edema that is not CSME.		
3. NPDR with CSME		
4. Severe NPDR		
5. NHRPDR		
6. NHRPDR with CSME		
7. HRPDR		
8. HRPDR not amenable to photocoagulation		

PLAN (Remarks):

....

MINISTERE DE LA SANTE PUBLIQUE

SECRETARIAT GENERAL

DIRECTION HOPITAL CENTRAL

No 36 DHCY/CM

MINISTRY OF PUBLIC HEALTH

SECRETARY GENERAL'S OFFICE

DEPARTMENT OF CENTRAL HOSPITAL

AUTORISATION DE RECHERCHE

Je soussigné **Pr Pierre MASSO MISSE** certifie qu'une autorisation de mener une enquête a l'hôpital Central de Yaoundé. service d'ophtalmologie est accordée au Dr NJIKAM JUDE ERIC. étudiant a l'UN (KENYA) L'étude porte sur **«the magnitude and pattern of diabetic retinopathy at Central Hospital, Yaounde Cameroun »**

L'éthique et la confidentialité sont à respecter

Au terme de ce Travail, une copie sera déposée à la bibliothèque de l'HCY

Fait à Yaoundé, 06 08 2010

Le Conseiller Médical

Professeur Masso

Ampliations :

- Directeur HCY
- Archives