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**THE MAGNITUDE AND PATTERN OF
DIABETIC RETINOPATHY AS SEEN AT
THREE HOSPITALS IN KIGALI,
RWANDA. //**

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DEDICATION

THIS WORK IS DEDICATED TO MY WIFE AND BEST FRIEND CHANTAL AND ALVIN MY SON FOR THE SUPPORT AND INSPIRATION THEY HAVE GIVEN ME DURING THIS WORK AND THE SACRIFICES THEY HAVE HAD TO MAKE FOR ME TO DO MY STUDIES FAR AWAY FROM THEM.

DECLARATION

THIS DISSERTATION IS MY ORIGINAL WORK AND HAS NOT BEEN PRESENTED FOR A DEGREE IN ANY OTHER UNIVERSITY.

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08 / 09 / 2008

APPROVAL

THIS DISSERTATION HAS BEEN SUBMITTED IN PART FULLFILMENT FOR THE AWARD OF THE DEGREE OF MASTERS OF MEDICINE (OPHTHALMOLOGY) WITH OUR APPROVAL AS UNIVERSITY SUPERVISORS.



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1.0 LIST OF ABBREVIATIONS

BCVA	Best Corrected Visual Acuity.
BP	Blood Pressure
CHK	Kigali Central Hospital
CSME	Clinically Significant Macula Edema
DCCT	Diabetes Control and Complications Trial
DR	Diabetic Retinopathy
DRC	Democratic Republic of Congo
DRS	Diabetic Retinopathy Study
ETDRS	Early Treatment of Diabetic Retinopathy Study
FBS	Fasting Blood Sugar
FLA	Fluorescein Angiography
HBA1c	Glycosylated Haemoglobin
HRPDR	High Risk Proliferative Diabetic Retinopathy
IDDM	Insulin Dependent Diabetes Mellitus
IFG	Impaired Fasting Glucose
IGT	Impaired Glucose Tolerance
KFH	King Faisal Hospital
ME	Macula Edema
NHRPDR	Non High Risk Proliferative Diabetic Retinopathy
NIDDM	Non Insulin Dependent Diabetes Mellitus
NPDR	Non Proliferative Diabetic Retinopathy
NVD	Neovascularisation at the disc
NVE	Neovascularisation elsewhere
OHA	Oral Hypoglycaemic Agents
PDR	Proliferative Diabetic Retinopathy
RDA	Rwanda Diabetes Association Clinic
UKPDS	United Kingdom Prospective Diabetes Study
WESDR	Wisconsin Epidemiological Study of Diabetic Retinopathy
USA	United States of America
WHO	World Health Organisation

2.0 ABSTRACT

Objective: To determine the prevalence and pattern of diabetic retinopathy (DR) and its associations in Diabetic patients attending three main diabetes clinics in Kigali, Rwanda.

Design: This was a hospital based cross sectional study.

Subjects: A total of 391 patients aged between 14 – 88 years attending diabetes clinics at three hospitals in Kigali during the month of September to mid October 2007 underwent a detailed eye examination. The blood pressure and fasting blood sugars of all the patients were also measured and DR was clinically graded using the DRS/ETDRS guidelines.

Results: DR was detected in 114 (29.2%) of the 391 patients with diabetes and 237 (60.6%) patients had never had a fundus exam. Severe non proliferative diabetic retinopathy (NPDR) was detected in 7 (1.8%) and clinically significant macula oedema (CSME) in 16 (4.1%) while proliferative diabetic retinopathy (PDR) was found in 18 (4.6%) of all the patients. DR was associated with high blood pressures, long duration of diabetes and high fasting blood sugars. There was no association of DR and sex of the patient.

Conclusion: The prevalence of DR among diabetic patients in this study was 29.2%. The number of diabetics who had a fundus examination for the first time was (60.6%). This number is high and there is need for a better referral system for early screening and management of DR in this population.

3.0 INTRODUCTION

Diabetic retinopathy, a common complication of diabetes is a leading cause of visual loss in diabetics and the most frequent cause of visual loss among working age persons in developed countries.¹

Diabetic retinopathy is responsible for approximately 5% of global blindness and accounts for approximately 2.5 million people blind.² Numerous studies have shown that there is a rising incidence of diabetes and its complications in all age groups and WHO predicts that developing countries will bear the brunt of this epidemic in the 21st century with 80% of all new cases of diabetes expected to appear in the developing countries by 2025.³ The magnitude of visual complications due to diabetes in many developing countries including Rwanda is not known. In the year 2000, it was estimated that Rwanda had approximately 30,000 people with diabetes and this number would increase to 77,000 by the year 2030.⁴

The socioeconomic burden due to the costs of healthcare and loss of productivity is enormous, not to mention the immeasurable misery to the individuals and their families. The annual cost of retinopathy associated disorders in the USA is estimated at more than 620 million dollars.⁵

Despite evidence that early detection and treatment of the vascular retinal changes will prevent or slow progression of blindness/visual impairment from diabetic retinopathy⁶, many diabetics in developing countries are yet to benefit from these findings. There are no treatment facilities for Diabetic retinopathy in Rwanda e.g Laser photocoagulation.

The aim of this study was thus to determine the prevalence of diabetic retinopathy and its associations at three centres treating diabetic patients in the

capital Kigali and hopefully raise awareness about the magnitude of the problem and provide baseline information from which specific interventions can be based.

2.2.1 Introduction

2.2.2 Objectives

The primary objective of this study is to assess the current status of the... (text is very faint and blurry)

2.2.3 Methodology

The study was conducted using a mixed methods approach... (text is very faint and blurry)

The data was analyzed using statistical software... (text is very faint and blurry)

4.0 BACKGROUND

4.1 Diabetes mellitus

4.1.1 Definition

The term diabetes mellitus describes a metabolic disorder of multiple aetiologies characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The disease can lead to serious complications and premature death but people with diabetes can take steps to control the disease and lower the risks of complications. It is among the leading causes of death, disability and economic loss through out the world.^{7,8}

4.1.2 Epidemiology

The prevalence of diabetes mellitus varies widely in different populations. WHO has estimated that there were 171 million people worldwide with diabetes mellitus in 2000 and predicted that 366 million people will have diabetes mellitus by the year 2030.⁹ The International Diabetes Federation has estimated that another 314 million persons have impaired glucose tolerance and that the number will increase to 472 million by 2030.¹⁰

In the United States of America, for example, as much as 7% of the population had diabetes mellitus in 2005. The Centers for Disease Control and Prevention have estimated that 14.6 million persons in the United States have diagnosed diabetes mellitus and an additional 6.2 million have the disease but it has not yet been diagnosed.¹¹ Worldwide the prevalence of diabetes mellitus is estimated at 6%.³

Diabetes is increasing faster in the developing economies than in the developed economies. Seven out of ten countries with the highest number of people living with diabetes are in the developing world and India has the world's largest population with diabetes, approximately 35 million people.³ It was estimated that 26 million people in China had Diabetes mellitus in 2001 and the prevalence has increased markedly recently due to population aging and increases in urban migration.¹² In Africa, there is paucity of data on the prevalence of diabetes mellitus however it is increasing faster in the urban population, Sidibe et al¹³ has estimated that the disease may affect up to 7% of the hospital population.

Persons with diabetes mellitus in developed countries are mostly elderly while most of those in developing countries are younger (45-64 years) thus increasing the impact of diabetes mellitus on those populations and societies.⁹

4.1.3 Clinical features

Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, polyphagia, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor, coma and, in absence of effective treatment, death. Often symptoms are not severe, or may be absent, and consequently hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made.

The long-term effects of diabetes mellitus include progressive development of the specific complications of retinopathy with potential blindness, nephropathy that may lead to renal failure, and/or neuropathy with risk of foot ulcers, amputation, Charcot joints, and features of autonomic dysfunction, including sexual dysfunction. People with diabetes are at increased risk of cardiovascular, peripheral vascular and cerebrovascular disease.

4.1.4 Pathogenesis

Several complex pathogenetic processes are involved in the development of diabetes. These include processes which destroy the beta cells of the pancreas with consequent insulin deficiency, and others that result in resistance to insulin action. The abnormalities of carbohydrate, fat and protein metabolism are due to deficient action of insulin on target tissues resulting from insensitivity or lack of insulin.

4.1.5 Classification

Several classifications exist for diabetes. The recommended classification includes both staging of diabetes mellitus based on clinical descriptive criteria and a complementary aetiological classification.

The aetiological type named Type 1 (or IDDM) encompasses the majority of cases which 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 for which neither an aetiology nor a pathogenesis is known (idiopathic).

Type 2 includes the common major form of diabetes 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 hyperglycaemic 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.

4.1.6 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 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 is derived, the current WHO criteria distinguishes a group with significantly increased premature mortality and increased risk of micro vascular and cardiovascular complications.

The current (2006) WHO diagnostic criterion for diabetes is as follows:

Diabetes

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

Impaired glucose tolerance (IGT)

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

Impaired fasting glucose (IFG)

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

* 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.

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).

4.1.7 Treatment

Details about treatment of diabetes mellitus are beyond the scope of this work. However dietary control, weight reduction, exercise, oral hypoglycaemic agents and insulin are the common modes of treatment. Patient education as well, is of paramount importance.

4.1.8 Ocular complications

Diabetes mellitus is associated with several ocular complications of which diabetic retinopathy has the most important effects on the visual system.

Other ocular complications include:

- a) Cataracts: senile cataract has been shown to appear earlier and progress faster in diabetic patients while the less common "true diabetic" cataract may occur from osmotic irregularities.
- b) The impaired circulation in the microvasculature of the diabetic eye may lead to ischemia of the optic disk. This leads to optic neuropathy which may cause permanent visual loss.
- c) Ischemia can also affect cranial nerves innervating the extraocular muscles, leading to any pattern of strabismus and diplopia. The 3rd, 4th and 6th cranial nerves are all susceptible to mononeuropathies from diabetes, by the same mechanisms that lead to peripheral neuropathies.

- d) Patients with diabetes are also at increased risk of primary open angle glaucoma.¹⁴ In addition, ischemic factors may lead to neovascularisation of the anterior chamber angle, leading to neovascular glaucoma.
- e) Other complications include:
- i) Lenticular myopia during hyperglycaemia.
 - ii) Conjunctiva: microaneurysms.
 - iii) Cornea: reduced corneal sensations and reflex tear production.
 - iv) Ciliary body and choroid: thickened basement membrane at the pigment epithelium of the pars plicata, arteriosclerosis of the choroid, obliterated lumen of the choriocapillaries at the macula.
 - v) Vitreous: vitreous haemorrhage and detachment.
 - vi) Central retinal vein occlusion.
 - vii) Pupillary reaction defects.

4.2 Diabetic retinopathy

4.2.1 Definition

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

4.2.2 Epidemiology

Diabetic retinopathy is a leading cause of new onset blindness in industrialised countries and a more frequent cause of blindness in middle income countries. WHO has estimated that Diabetic retinopathy is responsible for 4.8% of the 37 million cases of blindness through out the world.²

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.¹⁵

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 (when diabetes had been diagnosed when they were less than 30 years, presumed type 1).¹⁵ 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,¹⁶ while 84% taking insulin and 53% not taking insulin had some degree of diabetic retinopathy when the duration of diabetes mellitus was 15-20 years.¹⁷

Sixty percent of people who have had insulin dependent diabetes mellitus for 20 years or more will have proliferative diabetic retinopathy¹⁵ while more than 12% of those who have had the condition for 30 years or more, will be blind.¹⁸

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.^{18, 19}

If all patients with proliferative diabetic retinopathy were to receive timely evaluation and treatment, the rate of blindness would be reduced from 50% to less than 5% after 5 years, a greater than 90% reduction in blindness from this disease.²⁰

Worldwide, several studies on the prevalence of diabetic retinopathy have been done. Population based studies tend to show lower prevalence compared to hospital based studies.

In Australia, the Australian Diabetes, Obesity and Life-style study (AusDiab, 2003) reported a prevalence of 15.3%²¹ while in India, the Chennai Urban Rural Epidemiological study (CURES 1, 2005) reported prevalence of 17.6%.²² In Liverpool another population based study, the Liverpool diabetic eye study (1999) found prevalence of 33.6%.²³

In the African set up, mainly hospital based studies have been done. Among others, Dr. Kariuki et al (1999) found prevalence of 49.8% at Kenyatta National Hospital in Nairobi, Kenya²⁴. Kaimbo DK (1995) found prevalence of 32% in Democratic Republic of Congo²⁵ while Seyoum B (2001) in Ethiopia, found prevalence of 37.8%²⁶ and Mhando PA (1980) in Dar es Salaam reported prevalence of 25%.²⁷

4.2.3 Risk factors

Epidemiological surveys have shown that various risk factors known to be associated with DR tend to accelerate its course and increase its severity.

These risk factors include:

1. Duration of the disease

Duration of diabetes mellitus is probably the strongest predictor for development and progression of retinopathy.

The Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR), the widest and most prolonged population based ophthalmological survey, reported that higher prevalence of DR was associated with longer duration of Diabetes.²⁸

A study conducted by Dandona et al²⁹ in type 2 diabetic patients reported that 87.5% of those with diabetes for more than 15 years had DR compared to 18.9% of those who had diabetes for less than 15 years.

2. Glycaemic control

There is strong evidence to suggest that the development and progression of DR is influenced by the level of hyperglycaemia.^{22, 30} The protective effect of glycaemic control on the development and progression of DR has been investigated in both type 1 (WESDR³⁰ and Diabetes Control and Complications Trial, DCCT³¹) and type 2 diabetic patients (United Kingdom Prospective Diabetes Study, UKPDS³²). In the 14 year progression of

retinopathy study (WESDR), the prevalence of retinopathy in type 1 diabetic patients was 12% when glycosylated haemoglobin (HBA1c) was less than 7% as compared to 40.7% when HBA1c levels were greater than 10% and an increased risk of PDR was associated with more severe baseline retinopathy and higher HBA1c levels.³⁰ In the UKPDS, the risk reduction in eye complications for every 1% decrease in HBA1c was 19%. It is thus observed that long term glycaemic control plays an important role in delaying the onset and lowering down the progression of DR.

3. Hypertension

Reports have indicated that high diastolic blood pressures in young individuals and higher systolic blood pressures in older individuals can worsen DR.^{15, 33}

4. Renal disease:

Severe nephropathy is associated with worsening of DR. A link between renal and retinal angiopathy in diabetes has been long recognised. This effect may be mediated through an increase in blood pressure, fibrinogen levels and lipoproteins.³⁴ Cross sectional and longitudinal studies report a relationship between microalbuminuria, proteinuria and retinopathy.^{35, 36, 37}

5. Pregnancy

It is recognised that DR can progress rapidly during pregnancy due to hormonal changes. The progression is usually transient and the long term risk of progression of DR does not appear to be increased by pregnancy.³⁶

Other risk factors that have been shown to be associated with Diabetic retinopathy include: elevated serum lipids^{39, 40}, alcohol^{41, 42}, anaemia^{43, 44} and obesity.⁴⁵

4.2.4 Pathogenesis

Diabetic retinopathy is a microangiopathy affecting the retinal pre-capillary arterioles, capillaries and post-capillary venules with features of both microvascular occlusion and leakage.^{46, 47}

Several factors have 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 – retina, kidneys and nerves- are all freely permeable to glucose.⁴⁵

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 oedema, which may either be diffuse or localized. Chronic localized retinal oedema leads to the deposition of hard exudates at the junction of healthy and oedematous 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 'intraretinal microvascular abnormalities' (IRMA) and neovascularisation, thought to be formed by a vasoformative substance (vascular endothelial growth factor), elaborated by the hypoxic retinal tissue in an attempt to revascularise hypoxic areas of the retina. This substance promotes neovascularisation on the retina, optic nerve head and Iris.

4.2.5 Classification

Different classifications for DR exist depending on the purpose.

However, DR can be classified into early stage Non Proliferative Diabetic Retinopathy (NPDR), and a more advanced stage Proliferative Diabetic Retinopathy (PDR). Macula oedema can be present at any level of DR. NPDR is further classified into mild, moderate, severe and very severe. PDR may be early, high risk or advanced.

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 haemorrhages, retinal oedema, hard exudates, dilation 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 ischaemia.
- 2) Increased retinal capillary permeability resulting in macula oedema.

Diabetic macula oedema may manifest as focal or diffuse retinal thickening with or without exudates. Severe NPDR is defined by the Early Treatment of Diabetic Retinopathy Study (ETDRS) by the 4:2:1 rule.

- Diffuse intra retinal haemorrhages and microaneurysms in 4 quadrants.
- Venous beading in 2 quadrants.
- Intraretinal microvascular abnormalities (IRMA) in 1 quadrant.

Any of the above three findings indicates severe NPDR.

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).

The 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 ETDRS classified DR into 9 stages:

1. Normal or minimal non proliferative diabetic retinopathy (No DR) i.e with rare microaneurysms.
2. Non proliferative diabetic retinopathy without macula edema.
- 3 Non proliferative diabetic retinopathy with macular edema that is not clinically significant.
4. Non proliferative diabetic retinopathy with clinically significant macular edema (CSME)

CSME is defined by the ETDRS as the following:

- Thickening of the retina at or within 500 microns of the center of the fovea.

Or

- Hard exudates at or within 500 microns of the center of the fovea, if associated with thickening of the adjacent retina.

Or

- A zone or zones of retinal thickening one disc area or larger, any point of which is within a disc diameter of the center of the macula.

5. Severe non proliferative retinopathy (pre-proliferative).

6. Non high risk proliferative diabetic retinopathy without clinically significant macula oedema (NHRPDR without CSME).

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

8. High risk proliferative diabetic retinopathy (HRPDR). The high risk characteristics for severe visual loss are:

Neovascularisation at the disc (NVD) greater than 1/4 to 1/3 disc area or vitreous and/or preretinal haemorrhage accompanied by new vessels, either NVD or NVE which is \geq 1/4 disc area.

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

4.2.6 Treatment

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%.⁶

The Diabetic Retinopathy Study (1971-1978) demonstrated conclusively that scatter (pan retinal) laser photocoagulation reduces the risk of severe visual loss due to PDR by as much as 60%.⁴⁸

The Early Treatment Diabetic Retinopathy Study (1979-1990) demonstrated that (pan retinal) laser photocoagulation can reduce the risk of severe visual loss (best corrected vision of 5/200 or worse) to less than 2%. It also showed that focal laser photocoagulation can reduce the risk for moderate visual loss from diabetic macular oedema by 50%.^{49, 50}

The Diabetic Retinopathy Vitrectomy Study (1977-1987) provided insight into the timing of vitrectomy surgery to restore useful vision in eyes with non resolving vitreous haemorrhage.^{51, 52} In particular, it highlighted that in certain situations, early vitrectomy resulted in better vision.

The Diabetes Control and Complications Trial (1983-1993) conclusively demonstrated that intensive control of blood glucose as reflected in measurements of glycosylated haemoglobin reduced the risk for progression of diabetic retinopathy.⁵³

4.2.7 Screening

Diabetic retinopathy does not reduce vision in its early stages when treatment is most effective. Preventing blindness from retinopathy thus relies on early detection of asymptomatic disease by fundus examination and instituting appropriate treatment measures immediately.⁵⁴ In the western world, it has been reported that about 26% of patients with type 1 and 36% of patients with type 2 diabetes mellitus have never had their eyes examined.⁵⁵

In a study done by Kariuki et al at Kenyatta National Hospital in Kenya, 82% of the 601 diabetic patients examined were being seen by an ophthalmologist for the first time and 48.6% of them required treatment for diabetic retinopathy.²⁴

The situation in most other African countries might even be worse given the limited number of eye care health professionals available.

This underscores the need for screening services for diabetic retinopathy in many of these countries.

The fundus may be examined by ophthalmoscopy using a slit lamp and either a contact lens or a 90 D lens or by retinal photography.⁴⁸

It has been shown that seven standard field stereoscopic 30° fundus photography or a dilated indirect ophthalmoscopic and stereoscopic macula examination by a retina specialist are the two gold standards for assessing diabetic retinopathy, however digital colour photography can also be used.^{6, 56}

Recently, several new non invasive techniques promise to improve diagnostic sensitivity e.g the optical coherence tomography (OCT).⁵⁷

5.0 RATIONALE

The rationale for this study was the following:

1. The magnitude of visual complications due to DR in Rwanda is not known.
2. 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. The results of this study may encourage better collaboration between ophthalmologists and other disciplines as regards the need for early referrals of diabetic patients for screening.

6.0 OBJECTIVES

6.1 General objective

1. To determine the prevalence and pattern of DR in diabetic patients attending three main diabetes clinics in Kigali, Rwanda.

6.2 Specific objectives

1. To determine the prevalence of DR in patients attending three main diabetes clinics in Kigali.
2. To establish the pattern of DR by standardised grading using the ETDRS guidelines.
3. To determine the association between DR and the following known risk factors.
 - (i) Duration of diabetes.
 - (ii) Glycaemic control.
 - (iii) Blood pressure.

7.0 METHODOLOGY.

7.1 Study design

This was a hospital based cross sectional study.

7.2 Study period

The study was carried out from the beginning of September to mid October 2007.

7.3 Study population

All diabetic patients attending the diabetes clinics at King Faisal Hospital, Rwanda Diabetes Association clinic and Kigali Central Hospital, who gave consent to participate during the period of the study.

7.4 Setting

The study included patients from the following three main centers treating diabetic patients in Kigali.

King Faisal Hospital (KFH) is a semi-private institution that provides medical services mainly to the middle and upper class patients from all over the country. It has a diabetic clinic that runs once a week.

Rwanda Diabetes Association (RDA) clinic was founded in 1997 by members of the association. It is the oldest and has the majority of patients. It caters for diabetic patients from all over the country and of all social classes but mainly of the low socioeconomic status. Its clinic runs daily from Monday to Friday.

Kigali Central Hospital (CHK) is the main public hospital in Kigali and caters for patients of all social classes but mainly of the middle and low socioeconomic status. It has a diabetic clinic that runs once a week.

7.5 Sample size

The sample size was determined by using the following formula

$$n = Z_{\text{crit}}^2 * P (1-P) / D^2$$

Where n = required sample size.

P = prevalence of ocular disorder in people attending (estimated at 30%, taking into consideration other studies done in the region)

D = Precision of the Study set at 0.05

Z_{crit} is the cut off points along the x-axis of the standard normal probability distribution that represents probabilities matching the 95% confidence interval (1.96).

Substituting the above in the formula we get; $n \approx 323$ patients.

Thus the required minimum sample size was 323 patients.

7.6 Sampling method

All patients attending the three clinics during the period of study who gave consent were enrolled.

In total, 391 subjects were examined out the 650 patients who were referred from the three centers, giving us a response rate of 60%.

7.7 Exclusion criteria

1. Opaque ocular media not allowing adequate visualization of the fundus for grading of diabetic retinopathy (unless the opaque media was due to Vitreous hemorrhage secondary to Diabetes).
2. Diabetic children less than 12 years.

3. No consent.

7.8 Examination Procedure

All patients attending the three diabetes clinics during the study period were informed about the study and then referred to the eye clinic at King Faisal Hospital where all ocular examinations were carried out. On arrival, an informed consent was sought and the patients' demographic data entered in a questionnaire. The age of the patient was recorded using the last celebrated birthday.

The fasting blood sugars were measured in the diabetes clinics using a capillary blood sample with a glucometer. (Glucoplus Blood glucose meter, CR 2032, Canada) The measurements were done by a nurse at each of the centers who had previously been trained to use this type of glucometer.

The patient's blood pressure was then measured in sitting position after 5-10 minutes of rest using an automatic wrist BP machine (NISSEI, Model WS-320, Japan). An average of 3 readings was recorded. Hypertension was defined as Systolic BP of ≥ 140 mmhg and a Diastolic BP of ≥ 90 mmhg.

The visual acuity of each eye was then tested separately using a Snellen's chart at 6 meters and subjective refraction performed for those with vision of less than 6/6 by two trained ophthalmic assistants.

This was followed by anterior segment examination using a slit lamp biomicroscope (HAAG-STREIT BERN 900, Switzerland) after which patients were dilated using tropicamide 1% eye drops. One to two eye drops were applied into each eye three times at 5 minute intervals and the posterior segment examined when the pupil was fully dilated.

The fundus was examined using a monocular indirect ophthalmoscope and a pan retinal 20 D Volk lens after which binocular stereoscopic slit lamp examination with a 90 D Volk lens was done. Both slit lamp and fundus examination were

carried out by a final year ophthalmology resident with confirmation of findings by a consultant ophthalmologist.

After examination, the patient's condition was explained to them and those requiring medications or spectacles were given prescriptions. Patients who required treatment with laser photocoagulation and vitrectomy were referred to other countries where these services were available.

7.9 Data analysis and presentation

After cross checking the questionnaires for any missing entries, a data base was created in MS Access where all the data were entered. Data analysis was then carried out using the statistical package for social sciences (SPSS Version 12.0). Analysis involved calculation of frequencies, means, percentage proportion of retinopathy and p –values. The results are presented in forms of tables, histograms and pie charts.

7.10 Ethical considerations

Ethical approval was obtained from the ethics committee in Rwanda and informed consent was obtained from all the patients prior to any examinations.

Those requiring treatment were treated whenever possible or given advise and referrals.

The effects of drugs used were explained to all the patients, patient's data was also kept confidential.

8.0 RESULTS

A total of 391 diabetic patients were examined out of 650 patients referred from all the three diabetic clinics. (Response rate of 60%)

Table 1: Response rate.

Centre	No. of patients examined.	No. of patients referred.	Percent (%)
KFH	40	50	80.0
RDA	267	450	59.3
CHK	84	150	56.0
TOTAL	391	650	60.2

Figure 1: Distribution of patients by the Centres, (n = 391)

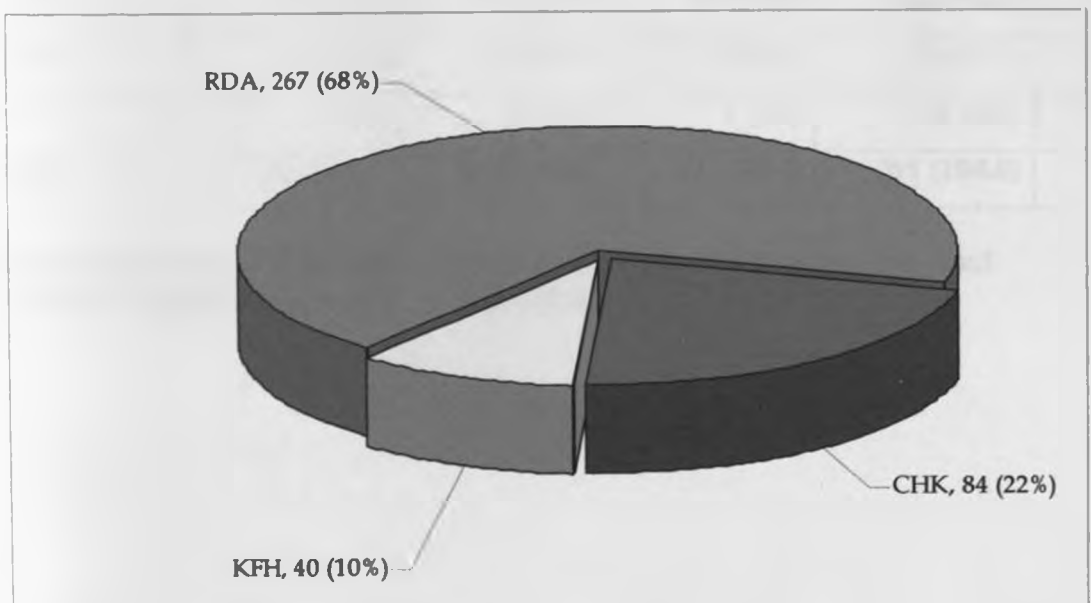


Table 2: Distribution of patients by sex and study centre, (n = 391)

Sex	Study Centre			Total	p-value
	KFH, n (%)	RDA, n (%)	CHK, n (%)		
Male	11 (27.5)	97 (36.3)	24 (28.6)	132 (33.8)	0.286
Female	29 (72.5)	170 (63.7)	60 (71.4)	259 (66.2)	
TOTAL	40 (100.0)	267 (100.0)	84 (100.0)	391 (100.0)	

The majority of study participants were female, ratio of male: female was approximately 1:2.

Table 3: Distribution of patients by age and study centre, (n = 391)

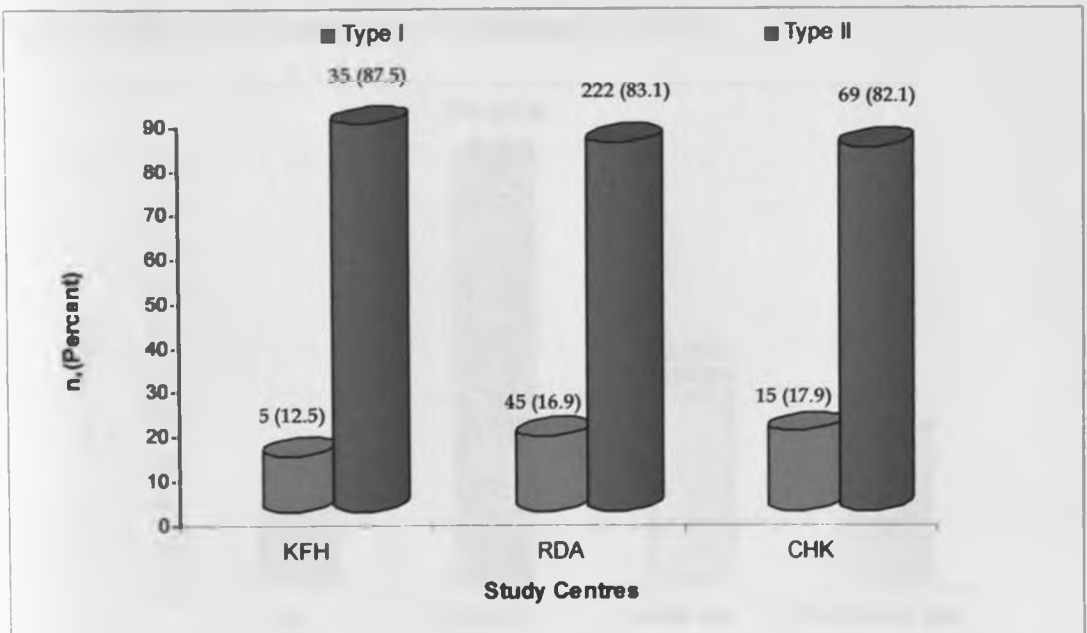
Age (in Years)	Study Centre			Total, n (%)	P-Value
	KFH, n (%)	RDA, n (%)	CHK, n (%)		
≤ 40	4 (10.0)	46 (17.2)	15 (17.9)	65 (16.6)	0.498
41 – 50	11 (27.5)	68 (25.5)	17 (20.2)	96 (24.6)	0.562
51 – 60	11 (27.5)	95 (35.6)	28 (33.3)	134 (34.3)	0.592
61 - 70	12 (30.0)	42 (15.7)	23(27.4)	77 (19.7)	0.104
71+	2 (5.0)	16 (6.0)	1 (1.2)	19 (4.9)	0.203
TOTAL	40 (100.0)	267 (100.0)	84 (100.0)	391 (100.0)	

Most patients were in the age range of 51-60 years (34.3%) and the least number of patients were over 71 years (4.9%).

Table 4: Summary statistics of Age, (n = 391)

Statistic	Study Centre			
	KFH	RDA	CHK	TOTAL
Mean	55.3	51.8	52.7	52.3
Mode	50	54	50	59
Range	25 – 78	14 – 88	20 - 82	14-88

The age distribution between the three study centres was not significantly different. (p -value = 0.226) and the mean age for the whole study population was 52.3 with a range of 14-88.

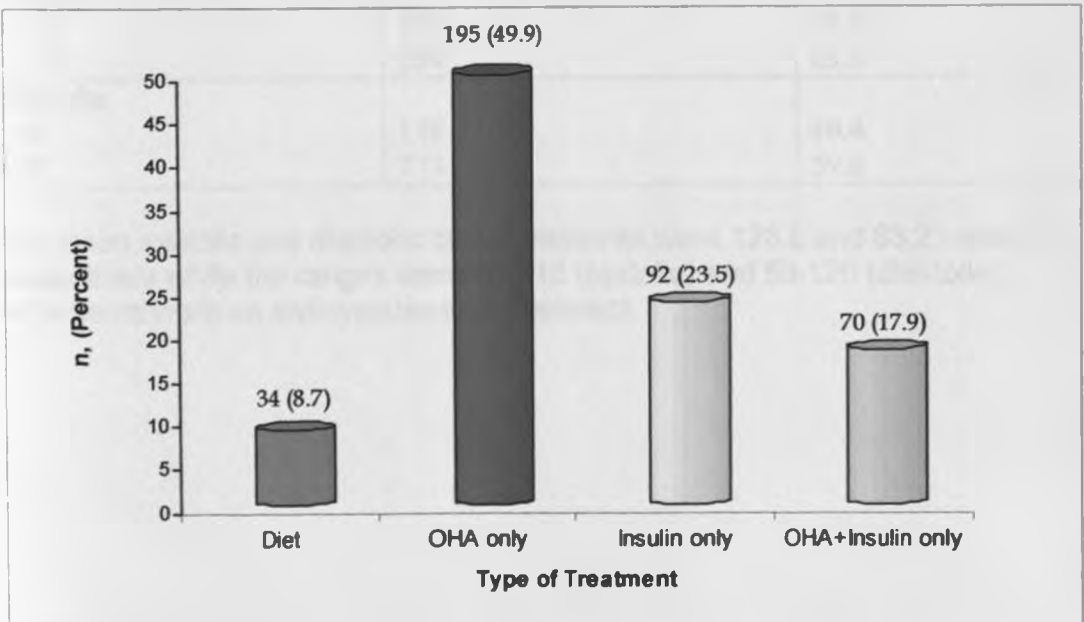
Figure 2: Type of Diabetes by Study Centre (n = 391)

Majority of the patients had type II Diabetes (83.4%).

Table 5: Duration of Diabetes by Study Centre (n = 391)

Duration (in Years)	Study Centre			TOTAL
	KFH, n (%)	RDA, n (%)	CHK, n (%)	
≤ 5	25 (62.5)	141 (52.8)	52 (61.9)	218 (55.8)
> 5 – 10	8 (20.0)	65 (24.3)	19 (22.6)	92 (23.5)
> 10	7 (17.5)	61 (22.9)	13 (15.5)	81 (20.7)
TOTAL	40 (100.0)	267 (100.0)	84 (100.0)	391 (100)

Patients at RDA had a slightly higher duration of diabetes, 47.2% had the disease for >5years compared to 38.1% at CHK and 37.5% at KFH. The differences were however not statistically significant (P -value = 0.144).

Figure 3: Mode of Treatment of Diabetes (n = 391)

Only a small proportion of the study participants (8.7%) were on diabetic diet.

Table 6: Glycaemic status of the study population, (n=391)

Sex	Fasting blood sugar (mg/dl)				
	< 120	> 120 - 140	> 140 - 160	> 160 - 180	> 180
Male, n (%)	47 (37.0)	17 (32.7)	9 (20.0)	9 (33.3)	50 (35.7)
Female, n (%)	80 (63.0)	35 (67.3)	36 (80.0)	18 (66.7)	90(64.3)
TOTAL	127 (100.0)	52 (100.0)	45 (100.0)	27 (100.0)	140 (100.0)

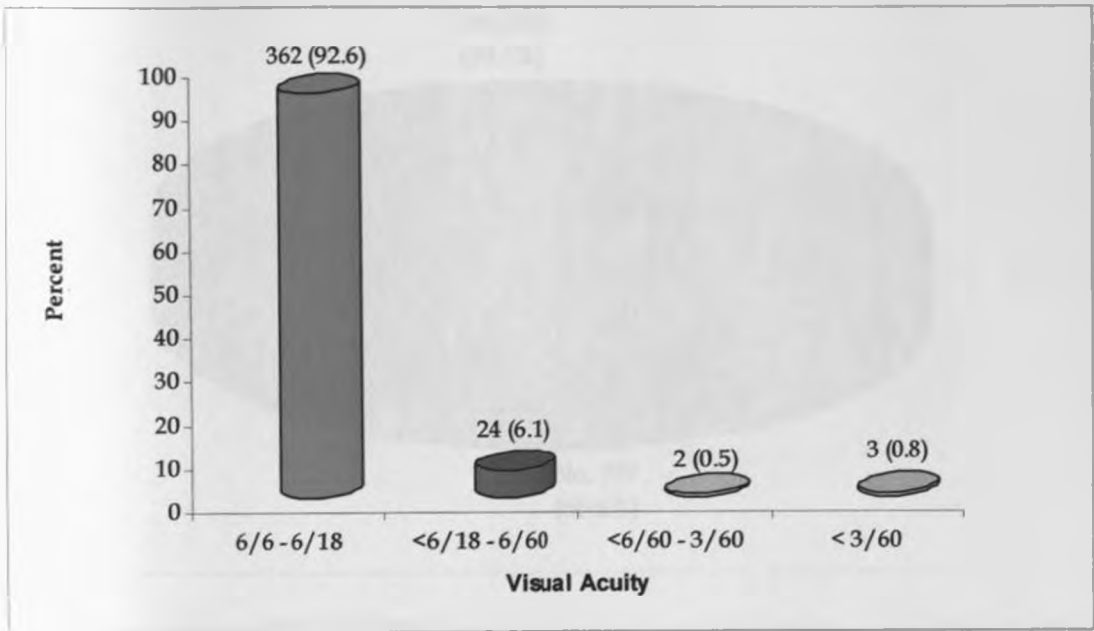
Majority of patients 264 (67.5%) had fasting blood sugars > 120 mg/dl.

Table 7: Characteristics of patients with hypertension, (n=391)

BP (mmhg)	Frequency	Percentage
Systolic		
≥ 140	137	35.0
< 140	254	65.0
Diastolic		
≥ 90	158	40.4
< 90	233	59.6

The mean systolic and diastolic blood pressures were 128.5 and 83.2 mmhg respectively while the ranges were 80-210 (systolic) and 50-120 (diastolic). All patients were on antihypertensive treatment.

Figure 4: Visual Acuity (BCVA) of the study population by WHO classification, (n=391)



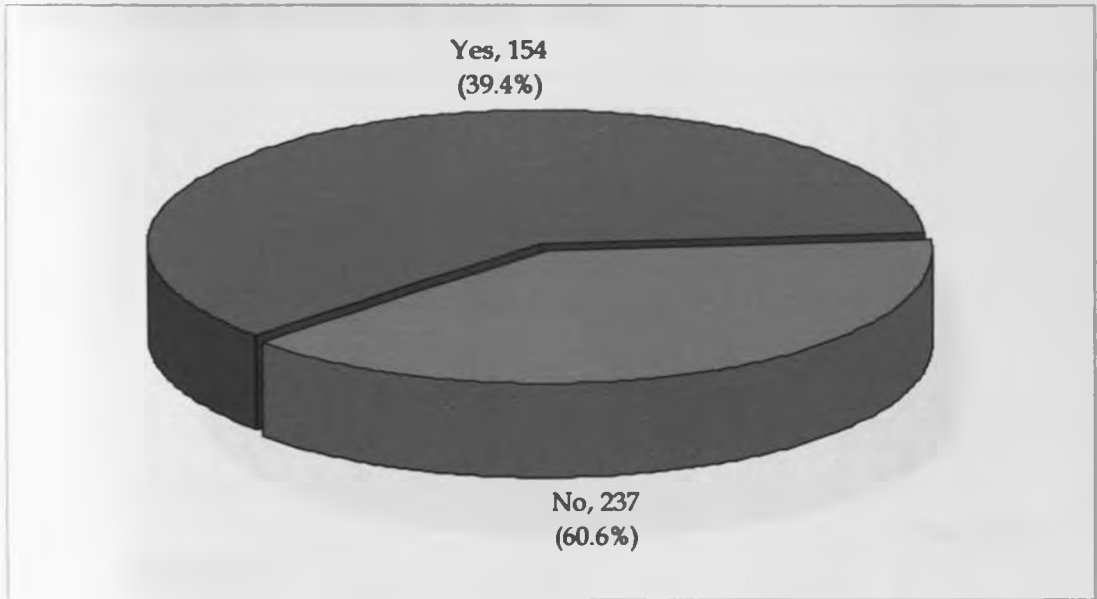
Most of the patients 92.6% had normal vision while 0.8% were blind by WHO classification.

Table 8: WHO grading of VA (BCVA) by Centre (n = 391)

Visual acuity	Study Centre.			P-value
	KFH	RDA	CHK	
6/6 - 6/18	37 (92.5)	245 (91.8)	80 (95.2)	0.320
< 6/18 - 6/60	2 (5.0)	19 (7.1)	3 (3.6)	0.461
< 6/60 - 3/60	-	2 (0.7)	-	-
< 3/60	1 (2.5)	1 (0.4)	1 (1.2)	-

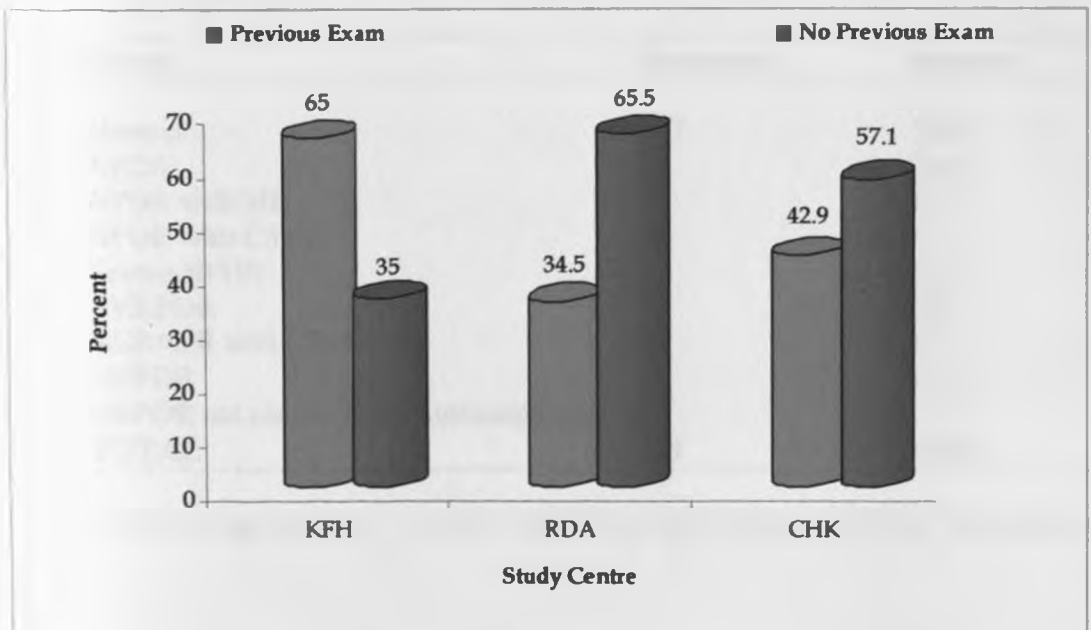
There were no statistically significant differences in the visual acuity at three centres.

Figure 5: History of previous fundus examination (n = 391)



Majority of patients 237 (60.6%) had not undergone a fundus examination before the study.

Figure 6: Previous fundus exam by centre (n = 391)



RDA has no ophthalmologist. It has the highest number of people with no previous fundus exam.

The prevalence of DR in the study population was 29.2% (n=391)

Figure 7: Prevalence of DR by study centre

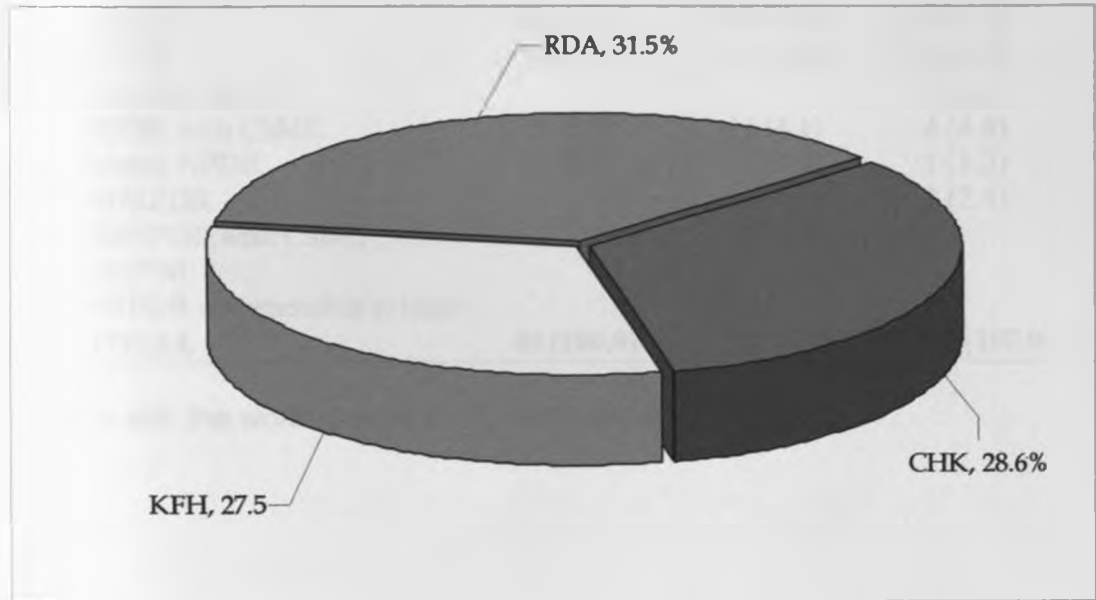


Table 9: Classification of DR in the worst eye (n= 391)

Classification	frequency	percent
1. Normal	277	70.8
2. NPDR	71	18.2
3. NPDR with ME	2	0.5
4. NPDR with CSME	16	4.1
5. Severe NPDR	7	1.8
6. NHRPDR	4	1.0
7. NHRPDR with CSME	2	0.5
8. HRPDR	3	0.8
9. HRPDR not amenable to photocoagulation	9	2.3
TOTAL	391	100.0

Severe NPDR was found in 7 (1.8%), CSME in 16 (4.1%) and PDR in 18 (4.6%).

Table 10: Classification of DR in the Worst Eye in the 3 centres (n = 391)

Classification		KFH	RDA	CHK
1.	Normal	29 (72.5)	183 (68.5)	60 (71.4)
2.	NPDR	9 (22.5)	52 (19.5)	15 (17.9)
3.	NPDR with ME	-	-	2 (2.4)
4.	NPDR with CSME	1 (2.5)	11 (4.1)	4 (4.8)
5.	Severe NPDR	1 (2.5)	5 (1.9)	1 (1.2)
6.	NHRPDR	-	2 (0.7)	2 (2.4)
7.	NHRPDR with CSME	-	2 (0.7)	-
8.	HRPDR	-	3 (1.1)	-
9.	HRPDR not amenable to laser	-	9 (3.4)	-
TOTAL		40 (100.0)	267 (100.0)	84 (100.0)

Patients with the worst grades of DR were found at RDA.

Table 11: DR and patient sex

DR Status	Patient sex		P –value
	Male	Female	
DR	37 (28.0)	77 (29.7)	0.727
No DR	95 (72.0)	182 (70.3)	
TOTAL	132 (100.0)	259 (100.0)	

DR was not associated with sex of the patient.

Table 12: DR and Best Corrected Visual Acuity (BCVA), (n = 391)

VA	DR Status		Total	P-value
	+ Ve, n (%)	- Ve, n (%)		
6/6 – 6/18	97 (26.8)	265 (73.2)	362 (100.0)	<0.001
< 6/18 – 6/60	14 (58.3)	10 (41.7)	24 (100.0)	0.001
< 6/60 – 3/60	2 (100.0)	-	2 (100.0)	-
< 3/60	1 (33.3)	2* (66.7)	3 (100.0)	0.564
TOTAL	114 (29.2)	277 (70.8)	391 (100.0)	

Visual acuity was significantly associated with DR status, patients with visual impairment and severe visual impairment were more likely to have DR.

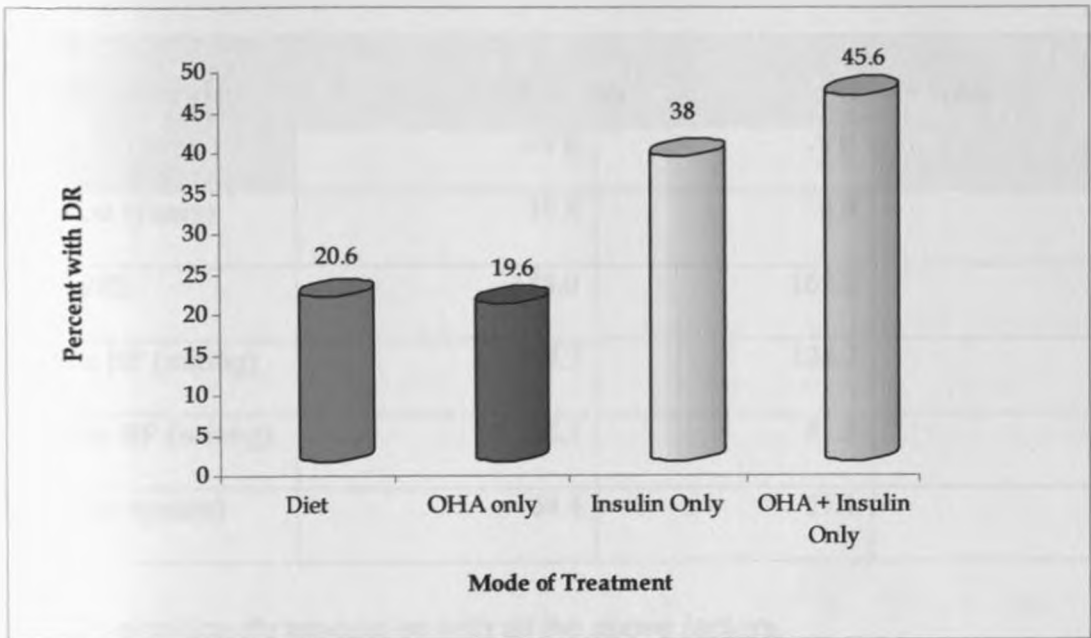
* These 2 patients had dense cataracts.

Table 13: DR and Type of Diabetes, (n=391)

DR Status	Type of DM		P-value
	I, n (%)	II, n (%)	
+ VE	12 (18.5)	102 (31.3)	0.038
- VE	53 (81.5)	224 (68.7)	
TOTAL	65 (100.0)	326 (100.0)	

DR was significantly associated with type 2 diabetes, (p -value=0.038).

Figure 8: DR and mode of treatment



Patients on combined OHA and Insulin had the highest prevalence of retinopathy 45.6%.

Table 14: DR and duration of diabetes

Duration (years)	DR Status		P -value
	DR	No DR	
≤ 5	29 (25.4)	189 (68.2)	0.081
> 5 – 10	37 (32.5)	55 (19.9)	0.011
> 10	48 (42.1)	33 (11.9)	< 0.001
TOTAL	114 (29.2)	277 (70.8)	

DR is significantly associated with duration of diabetes, the longer the duration, the higher the chances of having DR.

Table 15: Relationship between DR and duration of diabetes, FBS, BP & Mean age

Variable (mean)	DR Status		P -value
	+VE	-VE	
Duration (years)	10.8	4.9	< 0.001
FBS (g/dl)	188.0	162.2	0.010
Systolic BP (mmhg)	137.7	124.7	< 0.001
Diastolic BP (mmhg)	87.1	81.5	< 0.001
Mean age (years)	54.4	51.1	0.002

DR was significantly associated with all the above factors.

9.0 DISCUSSION

Diabetic retinopathy is increasingly becoming a major cause of blindness in the world in the age group 20 – 60 years⁶¹ and the incidence of diabetes mellitus and its complications is rising in developing countries³. Measures to reduce visual disabilities and improve the 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 visual complications due to diabetes in Rwanda has not been described. This study is thus the first to describe the prevalence, pattern and associations of diabetic retinopathy in patients attending three major diabetic clinics in the capital Kigali.

Three hundred and ninety one (391) patients were examined with the majority 267 (68%) being recruited from RDA clinic and the least, 40 (10%) from KFH clinic. This reflects the numbers of patients seen by these clinics and could probably be due to the differences in costs of healthcare at the three different hospitals with RDA offering subsidised prices while the other two hospitals do not. However, this finding could also have a bearing on the length of time these clinics have been in existence with RDA being the oldest and KFH, the youngest.

Of all the patients examined, 132 (33.8%) were male while 259 (66.2%) were female giving a ratio of Male: Female of 1: 2. This finding is probably due to the fact that women are usually affected by diabetes more than men and also probably due to the fact that there are more women than men in the general Rwandese population (ratio of male: female = 10:12).

Diabetic retinopathy (DR) was detected in 114 (29.2%) out of the 391 patients examined. Though various reports give different figures for the prevalence of diabetic retinopathy (depending on the methodology and population sample), the prevalence reported in this study correlates fairly well with other findings in this

region. In 1995, Kaimbo DK et al reported a prevalence of DR of 32% in the Democratic republic of Congo (DRC) ²⁵ while an older study done in 1980 by Mhando PA et al in Tanzania found a prevalence of 25%.²⁷ The prevalence found in this study is however lower than that reported by Kariuki et al (1999) of 49.8% in patients attending Kenyatta National Hospital, Kenya²⁴ and also lower than that reported by Seyoum B et al in Ethiopia (2001) of 37.8%²⁶ In India the Chennai Urban Rural Epidemiological Study (CURES 1, 2005) reported a prevalence of 17.6%²² while the Liverpool Diabetic eye study found a prevalence of 33.6%.²³

Of the three centres studied, Rwanda Diabetic Association (RDA) clinic had the highest prevalence of Diabetic Retinopathy (DR) of 31.5% followed by Kigali Central Hospital (CHK), 28.6% and lastly King Faisal Hospital (KFH), 27.5%. The differences in the prevalence at the three centres could have been due to the slightly higher duration of diabetes mellitus in patients who had DR and were attending the RDA clinic, (mean duration of 11.4 years), compared to those at CHK (8.8 years) and KFH (9.6 years) and also probably due to the poor glycaemic control as evidenced in the slightly higher mean fasting blood sugars of 171.9 g/dl at RDA compared to 169.7 g/dl at CHK and 162.3 g/dl at KFH, although these differences were not statistically significant.

Severe non proliferative diabetic retinopathy (NPDR) was found in 7 (1.8%) of the 391 diabetic patients while proliferative diabetic retinopathy (PDR) was found in 18 (4.6%) and macula oedema in 18 (4.6%). Vision threatening retinopathy (defined as presence of PDR or macula oedema) was present in 36 (9.2%) of all the diabetic patients. Only 3 (8.3%) of the 36 patients with vision threatening retinopathy had had laser photocoagulation done (this was done outside Rwanda). The prevalence of PDR found in this study (4.6%) is lower than what was found in Kenya (12.1%) by Kariuki ²⁴ but higher than what was found in Ethiopia (1.7%) by Seyoum .²⁶

Males and Females were equally affected by DR, with 37 (28.0%) of the males and 77 (29.7%) of the females having DR. This finding is consistent with other studies which have reported no association between DR and sex.^{24, 33, 58}

Presence of DR was associated with a higher mean age of 54.4 years where as those with no DR had a mean age of 51.1 years (p –value 0.002). The same finding has been reported in a study carried out in Oman.⁵⁹ However this finding could be associated with the longer duration of diabetes in the older population.

The type of Diabetes was associated with development of retinopathy, 12 (18.5%) patients of those with type 1 diabetes had DR while 102 (31.3%) of those with type 2 diabetes had DR. (In this study, Type 1 diabetes was defined as any patient whose age was ≤ 40 years and type 2 diabetes as anyone > 40 years of age at the time of diagnosis). Patients with type 1 diabetes are known to have higher risk for DR than those with type 2 diabetes.⁶⁰ However in our study patients with type 2 diabetes had a higher prevalence of DR (31.3%) compared to those with type 1 diabetes (18.5%). This could probably be explained by the relatively shorter duration of the disease in type 1 patients with a mean duration of 3.6 years compared to those with type 2 diabetes who had a mean duration of 7.2 years (p –value < 0.001). Similarly, in the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR)¹⁵ type 1 diabetic patients who had had diabetes for < 5 yrs duration had a prevalence of 13% compared to 20-24% in those with type 2 diabetes who had had the disease for the same duration.

Duration of diabetes is probably the most important predictor for development of DR. The WESDR reported that a higher prevalence of DR was associated with longer duration of diabetes.²⁸ Several other studies have shown similar association between DR and duration of diabetes.²⁹ According to a study done in Southern India, the prevalence of DR was 7% in individuals with a short duration of diabetes (< 10 years), 26% in those with 10 -14 years duration and 63% in those with 15 years or more duration of diabetes.⁶⁰ Similar findings are reported

in this study where patients with diabetic retinopathy had a statistically significant higher mean duration of diabetes of 10.8 years compared to 4.9 years in those with no diabetic retinopathy (p -value < 0.001).

It has also been observed that long term glycaemic control plays an important role in delaying the onset and lowering down the progression of DR. This protective effect of glycaemic control on the development and progression of DR has been investigated in both type 1^{30, 31} and type 2 diabetic patients.³² In this study, the fasting blood sugars of 391 patients were measured. The mean FBS was 169.5 mg/dl (9.4mmol/l). Patients who had diabetic retinopathy had statistically significant higher mean fasting blood sugars (188.0 mg/dl) compared to those who had no DR (162.2 mg/dl). Although measurement of glycaemic control is best demonstrated by measuring the glycosylated haemoglobin (HBA1c), measurement of fasting blood sugars done in this study showed that higher levels of FBS were also associated with development of DR as has been previously shown in other studies.³³ It was our desire to measure the HBA1c levels of the patients in this study but this was not possible due to lack of facilities to do this test in Rwanda.

The mean systolic and diastolic blood pressures in the study population were 128.5 mmhg and 83.2 mmhg respectively. Patients with DR had higher mean systolic and diastolic blood pressures (137.7 mmhg and 87.1 mmhg respectively) as compared to those with no DR who had mean systolic and diastolic blood pressures of 124.7 mmhg and 81.5 mmhg respectively (p -value < 0.001). These findings correlate well with other reports which have shown that higher systolic and diastolic blood pressures are associated with DR.^{15, 33}

The mode of treatment of diabetes was associated with presence or absence of DR. Patients whose mode of treatment was a combination of oral hypoglycaemic agents and Insulin, had a higher prevalence of DR (48.6%) and were followed by those on Insulin only (38.0%). This could be a reflection of the severity of the

disease and hence the need for combination therapy. This association between DR and mode of treatment has also been previously described in other studies.²⁴

Only 154 patients out of the 391 (39.4%) had had a fundus exam. The remaining 237 (60.6%) had their first fundus exam during this study. However, 65% of the patients attending KFH clinic had had a fundus exam while 42.9% of those at CHK had also had this exam. The lowest percentage (34.5%) of patients with a previous fundus exam was found at RDA clinic. This is probably because this clinic does not have an ophthalmologist and only refers its patients to other centres where these services are available. Thus many patients may end up not going there, especially if their vision is still perceived to be good. This finding however emphasises the need for a proper referral system so that patients could be screened early enough for DR when it can still be prevented or treated.

Study limitations

The following limitations could have affected this study.

1. The duration of diabetes was based on self report by patients without confirmation of medical records and this could have introduced some bias.
2. It is also possible that some patients may have been misclassified as either type 1 or type 2, since classification does not only depend on the age at diagnosis but mainly on the biochemical abnormalities in the body.
3. It was in the original design of this study to measure the glycosylated haemoglobin (HBA1c) since it offers a more reliable measure of the degree of blood sugar control, however this was not done as no such facility was available in Rwanda and it was not financially feasible to send the samples outside the country.

10.0 CONCLUSIONS

1. The prevalence of DR was 29.2% while Visually threatening DR (i.e. Macula oedema and proliferative diabetic retinopathy) was found in 31.6% of those with DR.
2. Poor glycaemic control, longer duration of diabetes mellitus and hypertension were associated with DR.
3. The majority of patients (60.6%) had never had a fundus examination.
4. Patients with DR were more likely to have poor vision compared to those without DR.
5. Majority of patients were poorly controlled and were not on dietary management for control of their diabetes.

11.0 RECOMMENDATIONS

1. There is need for treatment facilities for DR in the country like equipment and personnel to perform laser photocoagulation, flourescein angiography and Vitrectomy. At the moment, these facilities are not available in Rwanda and only very few of the patients who require these facilities can afford to seek treatment in other countries where they are available.
2. There is also need for an early referral system of all diabetic patients to an ophthalmologist for proper fundus examination. Early referrals would help to detect the disease in its early stages when it is still possible to treat it and thus halt its progression. This however requires a multidisciplinary approach for it to be successful.
3. 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.
4. Many of the study participants were not on diabetic diet for treatment of their disease. Physicians in Rwanda can help control the onset of DR with better glycaemic control and control of blood pressure.

Suggested further research

1. A study to investigate other associations of diabetic retinopathy like nephropathy, pregnancy, hyperlipidaemia and glycaemic control (using HBA1c) in this population.
2. Studies to investigate the cost effectiveness of routine screening of diabetics and the socioeconomic burden of visual loss as a result of diabetic retinopathy in the Rwandan population.
3. Studies to determine the magnitude of the problem in type 1 diabetic patients with an adequate sample size.
4. Studies to determine the prevalence of DR in newly diagnosed diabetic patients in this population.

APPENDIX 1: THE MANAGEMENT RECOMMENDATIONS FOR DIABETIC RETINOPATHY

GRADE	FOLLOW UP(months)	LASER	FLA
1	12	No	No
2	6 – 12	No	No
3	4 – 6	No	Occasionally
4	2 – 4	Yes	Yes
5	3 – 4	?	Occasionally
6	3 – 4*	Yes	Occasionally
7	3 – 4*	Yes	Yes
8	3 – 4	Yes	Occasionally
9	1 – 6	Not possible, vitrectomy indicated in some cases.	No

* If photocoagulation is deferred, follow up in 2 – 3 months.

? Value in treatment not certain.

APPENDIX 2: GRADING OF DIABETIC RETINOPATHY

GRADING OF DIABETIC RETINOPATHY

1. Normal or 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)
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.

APPENDIX 3: QUESTIONNAIRE

DATE..... IP/OP No.....

STUDY CENTRE: A) KFH B) RDA C) CHK

NAME..... ADDRESS.....

AGE..... SEX.....

TYPE OF DIABETES 1) TYPE 1 2) TYPE 2

DURATION OF DIABETES.....

DIABETES TREATMENT: DURATION.

DIET.....

O.H.A.....

INSULIN.....

BLOOD PRESSURE (mmhg)..... Treatment: Yes NO.

PREVIOUS FUNDUS EXAM: YES NO

VISUAL ACUITY: RE..... LE.....

REFRACTION: RE.....

LE.....

LABORATORY

1. FASTING BLOOD SUGAR.....

GRADING OF DIABETIC RETINOPATHY

DIABETIC RETINOPATHY	RE	LE
1. Normal or minimal NPDR		
2. NPDR without macula edema		
3. NPDR with macula edema that is not clinically significant.		
4. NPDR with CSME		
5. Severe NPDR		
6. NHRPDR		
7. NHRPDR with CSME		
8. HRPDR		
9. HRPDR not amenable to photocoagulation		

PLAN.....

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