

**"CLINICAL STUDY OF OCULAR MANIFESTATIONS IN
PATIENTS WITH TYPE 2 DIABETES MELLITUS."**

By

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In

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LIST OF ABBREVIATIONS:-

| | | |
|-------|---|---|
| BRAO | - | Branch retinal artery occlusion |
| BRVO | - | Branch retinal vein occlusion |
| CSME | - | Clinically significant macular edema |
| CWS | - | Cotton Wool spot |
| DCCT | - | Diabetes control and complication trial |
| DM | - | Diabetes Mellitus |
| DRS | - | Diabetic Retinopathy Study |
| ETDRS | - | Early Treatment Diabetic Retinopathy Study |
| FAZ | - | Foveal avascular zone. |
| H/Ma | - | Hemorrhage/microaneurysm |
| HRC | - | High risk characteristics. |
| IOP | - | Intra ocular pressure |
| IRMA | - | Intra retinal microvascular abnormalities |
| IDDM | - | Insulin dependent diabetes mellitus |
| NIDDM | - | Non insulin dependent diabetes mellitus |
| NPDR | - | Non proliferative diabetic retinopathy |
| NVD | - | New Vessels on disc |
| NVE | - | New vessels elsewhere in the retina. |
| NVI | - | New vessels in the iris |
| NVG | - | Neovascular glaucoma |
| PDR | - | Proliferative diabetic retinopathy |
| POAG | - | Primary open angle glaucoma. |
| PSC | - | Posterior SubCapsular Cataract |
| RD | - | Retinal detachment |
| WESDR | - | Wisconsin Epidemiologic Study of Diabetic Retinopathy |
| VTDR | - | Vision Threatened Diabetic Retinopathy |

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ABSTRACT

Background & Objectives

Diabetes is a metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. ¹

The World Health Organization (WHO) estimates that worldwide, there are currently 220 million people living with diabetes. Diabetes is becoming an important chronic disease in India. Infact, India is infamously known as the Diabetes Capital of the World. In 2011, India had 62.4 million people with type 2 diabetes, compared with 50.8 million the previous year, according to the International Diabetes Federation (IDF) and the Madras Diabetes Research Foundation. The nationwide prevalence of diabetes in India now tops 9%.

The most significant complication of diabetes mellitus involving the eye and which develops in 85% to 100% of all diabetes eventually is retinopathy. Prevalence of all types of Diabetic Retinopathy depends on the duration of diabetes & level of glucose control.² A wide spectrum of ocular conditions other than diabetic retinopathy is associated with diabetes. Significant attention is paid to the retinal complications of diabetes mellitus and their potentially devastating effects on vision. Diabetes mellitus, however, is a multisystem disease, and diabetic eye disease is an end-organ response to the effects of the condition on the human system. Each portion of the eye is susceptible to the harmful effects of diabetes.³

Diabetic eye disease refers to a group of eye problems that people with diabetes may face as a complication of diabetes ranging from subtle lid xanthomas to vision threatening condition. This clinical study of ocular manifestations of patients with type

2 diabetes mellitus is undertaken in view of large spectrum of diabetic eye disease apart from diabetic retinopathy.

Aims and Objectives of this study were

- To determine the common ocular manifestations in diabetes.
- To determine ocular manifestations relating to duration of diabetes and severity of the diabetes

Methods: This is a prospective observational study on patients attending outpatient department and those referred to department of Ophthalmology at B.L.D.E. University's Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur Karnataka from October 2012 to 31st march 2014 fulfilling the inclusion and exclusion criteria.

Results: Diabetic retinopathy was the most common ocular complication occurring in diabetes subjects (36.8%). The prevalence of cataract was 35.4% followed by glaucoma (4.6%) and other ocular pathologies like conjunctivitis, recurrent styes, dacrocystitis, etc. The strongest predictor for the prevalence of retinopathy in persons with type 2 diabetes is the duration of diabetes and was proven statistically significant. Both prevalence and severity of retinopathy correlates with HBA1C level in our study group. The most common type of cataract found was cortical type (41.2%) followed by senile posterior cortical (29.8%). In general, the visual prognosis following cataract surgery in diabetic patients is favourable. Diabetes predisposes to infection in different body parts, and ocular structures are not an exception.

Interpretation & Conclusion :

Diabetic retinopathy was the commonest ocular complication of diabetes, followed by cataract and primary open angle glaucoma. The prevalence and severity of diabetic retinopathy was higher in patients with longer duration of diabetes.

Key words: ocular complications, diabetes, diabetic retinopathy, cataract.

INTRODUCTION

In recent decades, diabetes mellitus has progressed from a disease affecting primarily people in developed countries into a true worldwide epidemic. The World Health Organization (WHO) in 1999 defined diabetes mellitus as “a metabolic disorder of multiple etiology 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, resulting from microvascular and macrovascular complications.⁴

It is estimated that in 2005 nearly 200 million people worldwide had diabetes mellitus. Most of these patients are classified as having type 2 diabetes mellitus and the metabolic syndrome. Most of the increase in total numbers of diabetic patients is expected to occur in developing countries. Worldwide, about 300 million people are expected to have diabetes by 2025, affecting 5.4% of the world’s population .⁵ Changing dietary and exercise trends appear to play a major role in the increasing prevalence of diabetes mellitus.

India is infamously known as the Diabetes Capital of the World. In 2011, India had 62.4 million people with type 2 diabetes, compared with 50.8 million the previous year, according to the International Diabetes Federation (IDF) and the Madras Diabetes Research Foundation. The nationwide prevalence of diabetes in India now tops 9%. By 2030, India will have 100 million people with diabetes.⁶ Diabetes Mellitus being a lifestyle disease, is on the rise in urban areas; Shankar Nethralaya reported that the

prevalence of Diabetes Mellitus in the population older than 40 years, in urban India, was around 28% in 2014.⁷

Diabetic eye disease refers to a group of eye problems that people with diabetes may face as a complication of diabetes ranging from subtle lid xanthomas to vision threatening condition. Some of these are not characteristic while others are pathognomonic for diabetes.

The most important complications are briefly listed as follows.⁸

- **Eyelids** : Xanthelasmata, Blepharitis, Recurrent hordeolum, eczema.
- **Conjunctiva** : Microaneurysms, Venous dilatation.
- **Cornea** : Folds of Descemet's membrane, neurotrophic keratitis.
- **Iris** : Iritis, Iris pigments on lens, Rubeosis Iridis.
- **Pupil** : Rigid pupil, light near dissociation.
- **Lens** : Cataract, refractive errors.
- **Vitreous** : Vitreous haemorrhage, asteroid hyalosis.
- **Retina** : Diabetic retinopathy; retinal vein occlusion, Lipaemia retinalis.
- **Optic nerve** : Ischemic papillitis; Optic atrophy.
- **Extra-ocular Muscles** : Palsy caused by 3rd, 4th, 6th cranial nerve involvement.
- **Orbit** : Mucormycosis.
- **Intra-ocular pressure** : Primary Open angle glaucoma, Neovascular glaucoma.

Diabetic retinopathy is the most well known ocular complication of diabetes and the leading cause of blindness among people 20–64 years of age in the U.S.⁹ It is also 6th most common cause of blindness in India (NPCB).

A Meta-analysis by Yau JY¹⁰ provides a global estimate of the prevalence of DR and the severe stages of DR (PDR, DME) using individual-level data from population-based studies worldwide. On the basis of the data from all 35 studies on more than 20,000 participants with diabetes, they estimated that among individuals with diabetes, the overall prevalence of any DR was 34.6%, PDR was 7.0%, DME was 6.8%, and VTDR was 10.2%. The prevalence of DR in the Chennai Urban Rural Epidemiology (CURES) Eye Study in south India was 17.6 per cent, significantly lower than age-matched western counterparts.¹¹

Numerous studies have shown that the key to decreasing diabetic complications lies with strict glucose control. Intensive glucose control reduced the risk of developing retinopathy by 54%. Neuropathy was reduced by 60% and albuminuria by 54%, respectively. With regards to type 2 diabetes mellitus, the United Kingdom Prospective Diabetes Study (UKPDS) was a randomized clinical trial involving 3867 newly diagnosed patients with type 2 diabetes.¹² After 3 months of diet treatment alone, patients with a mean of two fasting plasma glucose concentrations of 6.1 to 15.0 mmol/L were randomly assigned to either an intensive glycemetic control group or a conventional control group. This study showed a 21% reduction in risk for progression of diabetic retinopathy over a 12-year period in the intensive group. In addition, there was a 29% reduction in the need for retinal photocoagulation in the intensive group compared to the conventional group. Overall, there was a 37% reduction in the risk of an adverse microvascular complication with intensive control that was less strict than current guidelines.

The UKPDS study also demonstrated that glycemetic control appears to diminish with time. Clearly, the best indicator of glycemetic control continues to be hemoglobin

A1C (HBA1C). Skyler and associates have demonstrated that HBA1C levels correlate in a direct relationship with the relative risk of diabetic microvascular complications .¹³ Strict glucose control, weight control, and exercise, remain the essential elements to prevent the complications of diabetic disease.

Treatment interventions at early stages of diabetic retinopathy can reduce burden of blindness due to diabetic retinopathy. With the available cost-effective methods of early screening, appropriate strategies/models need to be developed. Such models need to have a well-developed mode for screening, diagnosis and referral at each hierarchal level beginning from primary health centres to specialized institutes for eye care. The National Program for Control of Blindness of India recommends opportunistic screening for identification of diabetic retinopathy. Every opportunity of contact with high-risk cases for diabetes and/or diabetic retinopathy should be utilized for screening, diagnosis and referral. All the stakeholders including the private sector will need to play a role. Along with this, awareness generation and behaviour change amongst the diabetics and care support systems should also be part of the overall model. A major role can be played by community participation and improving the health seeking behaviour among diabetics in order to reach a larger population and increasing the compliance for continued care.

It is the responsibility of ophthalmology community in creating awareness in the society so as to prevent and or delay these complications and to treat them at the earliest. It is in this context, we have studied the prevalence of diabetic eye complications at our hospital.

AIMS AND OBJECTIVES

- To determine the common ocular manifestations in diabetes.
- To determine ocular manifestations relating to duration of diabetes and severity of the diabetes.

REVIEW OF LITERATURE

LITERATURE SURVEY:

- Negi et al. (2003) have found that recurrent styes and blepharoconjunctivitis are the first indications of diabetes and should prompt tests to exclude it. Xanthelasma, is commonly seen in diabetics, with associated hyperlipidaemia.¹⁴
- Morikubo S ,Takamura Y , have shown that Recurrent corneal erosions in patients with diabetes are usually posttraumatic and the result of apparently mild epithelial breakdown following cataract or vitreoretinal surgery and compared with nondiabetic eyes, eyes of patients with diabetes mellitus showed more damage in corneal endothelial cells due to cataract surgery and a delay in the postoperative recovery of corneal edema.¹⁵
- Kruse A, Thomsen et al. have reported in Diabet Med , in 2006 that diabetes is a risk factor for acute infectious conjunctivitis.¹⁶
- In the Blue Mountains and Beaver Dam Eye studies, participants with diabetes were twice as likely to have glaucoma.¹⁷ Several large epidemiological studies have reported positive associations between diabetes with primary open angle glaucoma (POAG). Glaucoma occurs more often in patients with diabetes (5%) than in the general population (2%). The risk of glaucoma has been reported to be 1.6–4.7 times higher in individuals with diabetes than in nondiabetic individuals.
- Bonavas S, Peponis V et al.¹⁸ did a meta analysis results suggest that diabetic patients are at significantly increased risk of developing primary open-angle glaucoma. Clinicians should be aware of this possibility.

- Data from the Framingham and other eye studies (1985) indicate a three to fourfold increased prevalence of cataract in patients with diabetes under 65, and upto a two fold excess prevalence in patients with 65.
- Numerous studies have documented an association between diabetes and cataracts.¹⁵ This association is supported by an abundance of data from clinical epidemiological studies and basic science studies . Both cross-sectional and prospective data from three population-based studies, the Beaver Dam Eye Study, the Blue Mountains Eye Study, and the Visual Impairment Project, have documented associations between diabetes and both prevalent and incident posterior subcapsular cataract and, less consistently, with prevalent and incident cortical cataracts but not nuclear cataract . The Blue Mountains Eye Study showed that impaired fasting glucose, in the absence of clinical diabetes, was also a risk factor for the development of cortical cataract. There is additional evidence that the risk of cataract increases with increasing diabetes duration and severity of hyperglycemia . Deposition of advanced glycation end products in the lens has been postulated as one possible pathogenic mechanism for diabetic cataract⁶
- Knorz M C, university of heidelberg , Germany have studied that posterior capsular opacification is the frequent complication following extracapsular cataract extraction in diabetics patients.
- Scherzter, Wang et al have shown that between 32% and 43% of neovascular glaucoma cases are caused by proliferative diabetic retinopathy¹⁹

- Bandello F, Menchini F suggest that Diabetic papillopathy is a risk factor for the progression of diabetic retinopathy and, in rare instances, papillopathy can precede the development of AION.²⁰
- Watanabe K showed that in one study 1% of patients with diabetes were found to have cranial nerve palsies, compared with only 0.13% of control subjects. In another population-based study, patients with sixth cranial nerve palsy were six times more likely to have diabetes.²¹
- Prevalence of Diabetic retinopathy in wisconsin epidemiology study of Diabetic retinopathy (WESDR) ²² was 50%. 35-39% in United Kingdom Prospective Diabetes Study (UKPDS) in Diabetes mellitus.

HISTORY

Essential diabetes mellitus was recognized as 'madhumeha' or 'honey urine' and the symptoms of thirst; foul breath and languor was noticed by Sushruta in 5th Century A.D.²³

Aretaeus; a follower of Hippocrates of Alexandria in 2nd Century A.D. gave the name Diabetes (a passer through; siphon like) to the disease.²³

In 1778 John Rollo first noticed that the sufferers from diabetes mellitus are particularly prone to development of cataract.²⁴ Diabetic cataract was first described by Berndt in 1834.

In 1856, Jaeger first described the existence of specific retinal changes in the fundi of diabetes.²⁵

Meckenzie in 1887 presented in clinical form and with complete post mortem pathologic data a beautiful picture of capillary microaneurysms; retinal and vitreous haemorrhage.

Nettleship in 1888 first drew attention describing retinal venous stasis, tortuosity and dilatation of retinal veins.²⁶ In 1890, Hirschberg presented the first comprehensive classification of diabetic retinopathy. He distinguished between exudation and hemorrhagic retinopathy.

In 1921 Sir Fredrick Grass Banting and Charles Herbert Best discovered and isolated Insulin.²³

Salus in 1928 described the condition of rubeosis iridis.²⁷

Clinical application of fundus photography was made by Bedel in 1939 to analyse photographs of 20 diabetics.²⁸

Friedenwald in 1950 introduced the Hotchkiss McManus technique of staining flat retinal preparation. Ashton in 1950 employed this technique and compared the lesions of retina and kidney in diabetics.²⁹

Poulsen in 1953, Kinsel et al in 1954 and Fust et al in 1955 showed that post partum necrosis; hypophysectomy and adrenalectomy have all been followed by improvement in diabetic retinopathy.³⁰

Rucker in 1958 found that 6% of the 335 isolated third nerve lesions and 4% of 409 isolated sixth nerve lesions were in diabetics.³¹

The introduction of photocoagulation by Meyer – Schwickerath in 1959 proved to be an important step in the management of diabetic retinopathy.³²

In 1961; Novotny and Alvis introduced fundus fluorescein angiography.³³

The past decades have witnessed major advances in the treatment and understanding of diabetes. The field has been greatly aided by organizational agreement on an upgraded terminology necessary to properly classify the heterogeneity of the diabetes syndrome.

By the 20th century end, the long term acrimonious debate over the value of good glycemic control of diabetes became resolved with the results of several large well designed and well executed clinical studies. However despite major improvement in the options for care and depth of research in affluent areas, this progress is dampened by the challenge seen in an increasing epidemic of diabetes throughout the world.

A. LIDS:

Blepharitis, recurrent hordeolum and eczema with poor healing tendencies occur in diabetes mellitus . In diabetics gangrene of the lid following hordeolum is an example. Yellowish plaques called xanthelasma which occur due to hyperlipidemia are also common in diabetics mellitus.³⁴

B. CONJUNCTIVA:

Aneurysms ³⁵ of the conjunctival blood vessels are found commonly in diabetics. The significance of conjunctival aneurysms is disputed.

C. CORNEA:

Several corneal abnormalities have been identified in patients with long standing diabetes mellitus. ³⁶ Common corneal complications-

1. Superficial punctate keratopathy
2. Recurrent corneal erosions
3. Persistent epithelial defect
4. Filamentary Keratitis
5. Neurotropic keratopathy
6. Tear film abnormalities
7. Infiltrates
8. Endothelial damage
9. Increased corneal thickness
10. Wrinkling of descemet's membrane (Waite-Beetham lines)

Diabetic patients may have significantly decreased corneal sensitivity, and the severity of the decreased sensitivity is usually correlated positively with the severity of retinopathy. Diabetes has also been reported to be associated with dry eyes, with the severity of dry eyes correlated positively with the severity of diabetic retinopathy. Intrinsic abnormalities of the epithelial basement membrane complexes and impaired epithelial barrier function predispose to superficial punctate keratitis, poor epithelial wound healing after trauma, and persistent epithelial defects. Diabetic patients are prone to recurrent corneal erosions, especially after photocoagulation and vitrectomy.

The poor adhesiveness of the diabetic corneal basement membrane may be related to changes in biochemical composition induced by increased sorbitol and fructose produced by the aldose reductase pathway.

D.IRIS:

Diabetic individuals are prone to both morphologic and vascular alterations. Iris neovascularization (rubeosis iridis) is known to occur due to frequent occlusion of small vessels. It develops as tiny dilated capillary tufts or red spots around the pupillary margin and may be missed unless examined carefully under high magnification. The new vessels grow radially over the surface of the iris towards the angle, sometimes joining dilated blood vessels at the collarette. At this stage intraocular pressure (IOP) may be still normal in some cases and the new vessels may regress either spontaneously with good metabolic control and panretinal photocoagulation of the retina.³⁷

E. PUPIL:

A brisk near response and a sluggish direct response to light is called light near dissociation. The most common cause of light near dissociation is diabetes mellitus. The mechanism is not well understood but may represent a selective neuropathy that involves pupillomotor parasympathetic fibres.³⁸

F. REFRACTIVE CHANGES:

Refractive changes are known to be associated with diabetes mellitus and these can be acute or chronic. Regarding chronic refractive changes in diabetic patients, Duke-Elder reported that hyperglycaemia led to the development of myopia, while hypoglycaemia led to the development of hyperopia. Several papers have reported an abrupt reduction in plasma glucose in diabetic patients with marked hyperglycaemia induced transient hyperopia.³⁹ It has been seen that the degree of hyperopia is highly dependent on the magnitude of the change in plasma glucose concentration. Lenticular swelling has been suggested as a cause of hyperopia in diabetic patients. In patients with diabetes mellitus, excess glucose in the crystalline lens is converted to sorbitol through the action of aldose reductase. Sorbitol, a sugar alcohol, has poor permeability through membranes and tends to accumulate in lens.⁴⁰ When the body rapidly changes from a hyperglycaemic to a hypoglycaemic state, sorbitol, which is less permeable and harder to metabolise, will remain in the lens longer. The difference in osmotic pressure results in the influx of water from the aqueous humour into the lens, causing lenticular swelling with hyperopic refractive changes.

G. LENS:

Diabetic lens is larger than normal, disposed to refractive changes and at increased risk of cataract.

Lens thickness

Lens thickness is greater in diabetics than in normal subjects due to cortical thickening.⁴¹ Diabetic duration was found to be a highly significant determinant of lens dimensions, such that age-related dimensional changes for various biometric parameters were accelerated by between 52% and 121% after the onset of diabetes.⁴² These lens changes are more pronounced in insulin dependent diabetes mellitus.

CATARACT IN DIABETES

Cataract in diabetic patients is a major cause of blindness in developed and developing countries. Recent basic research studies have emphasized the role of the polyol pathway in the initiation of the disease process. Population-based studies have greatly increased our knowledge concerning the association between diabetes and cataract formation and have defined risk factors for the development of cataract.

It has been shown that the intracellular accumulation of sorbitol leads to osmotic changes resulting in hydropic lens fibers that degenerate and form sugar cataracts. In the lens, sorbitol is produced faster than it is converted to fructose by the enzyme sorbitol dehydrogenase. The increased accumulation of sorbitol creates a hyperosmotic effect that results in an infusion of fluid to countervail the osmotic gradient.

Overall risk for cataract formation in diabetes patients is 2-4 times as compared to non diabetes subjects.⁴³ Posterior subcapsular cataracts are particularly common in diabetic patients than other morphological types of cataract.⁴⁴ In type 2 patients increased severity of retinopathy, diuretic usage, lower IOP, smoking, lower diastolic blood pressure was associated with higher incidence of cataract.⁴³ Typically these are snow flake opacities, polychromatic crystals and vacuoles in lenticular cortex. Mechanism behind the cataract formation has been implicates to be the sorbitol pathway.

Cataract Surgery in Diabetes:

In individuals with diabetes, cataract occurs at a younger age and progresses more rapidly, resulting in higher rates of cataract surgery at a relatively young age.¹⁵ In the Wisconsin Epidemiologic Study of Diabetic Retinopathy,²² the 10-year cumulative incidence of cataract surgery was 8% in those with type 1 diabetes and 25% in those with type 2 diabetes. Predictors of cataract surgery included older age, greater severity of diabetic retinopathy, and baseline proteinuria in type 1 diabetes and older age and use of insulin in type 2 diabetes.

While the overall outcomes of cataract surgery are excellent, patients with diabetes may have poorer vision outcomes than those without diabetes, and the worst outcomes may occur in operated eyes with active proliferative retinopathy or preexisting macular edema. To improve cataract surgical outcomes in patients with diabetes, adequate control of diabetic retinopathy with laser treatment before cataract surgery is necessary.

The most devastating postoperative complication is endophthalmitis, a severe intraocular infection.

H.ASTEROID HYALOSIS

These are innumerable small, white or creamy opacities in the vitreous which move through a limited range with the movement of the head. They consist of calcium containing acidific lipids which lie between the vitreous fibrils. A high frequency of diabetes has been found in groups of patients having asteroid hyalosis.⁴⁵

I. DIABETIC RETINOPATHY

EPIDEMIOLOGY

Diabetic retinopathy is among the most common ophthalmic complication of diabetes mellitus.⁴⁶ Studies have reported that the prevalence of diabetic retinopathy in India varies from 20-31%. The estimates of the prevalence of diabetic retinopathy in India suggests that there may be nearly 5.6 million people with diabetic retinopathy in India .

Pathogenesis of DR

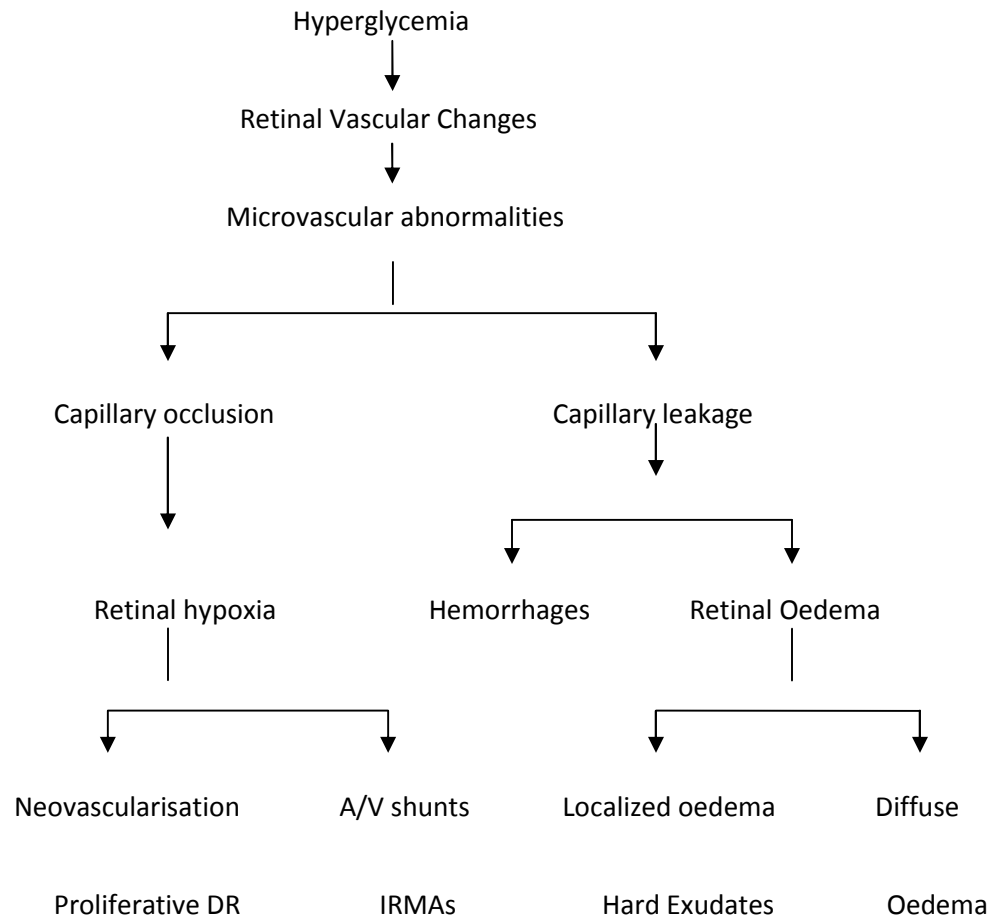


Table No. 1: Pathogenesis of Diabetic Retinopathy

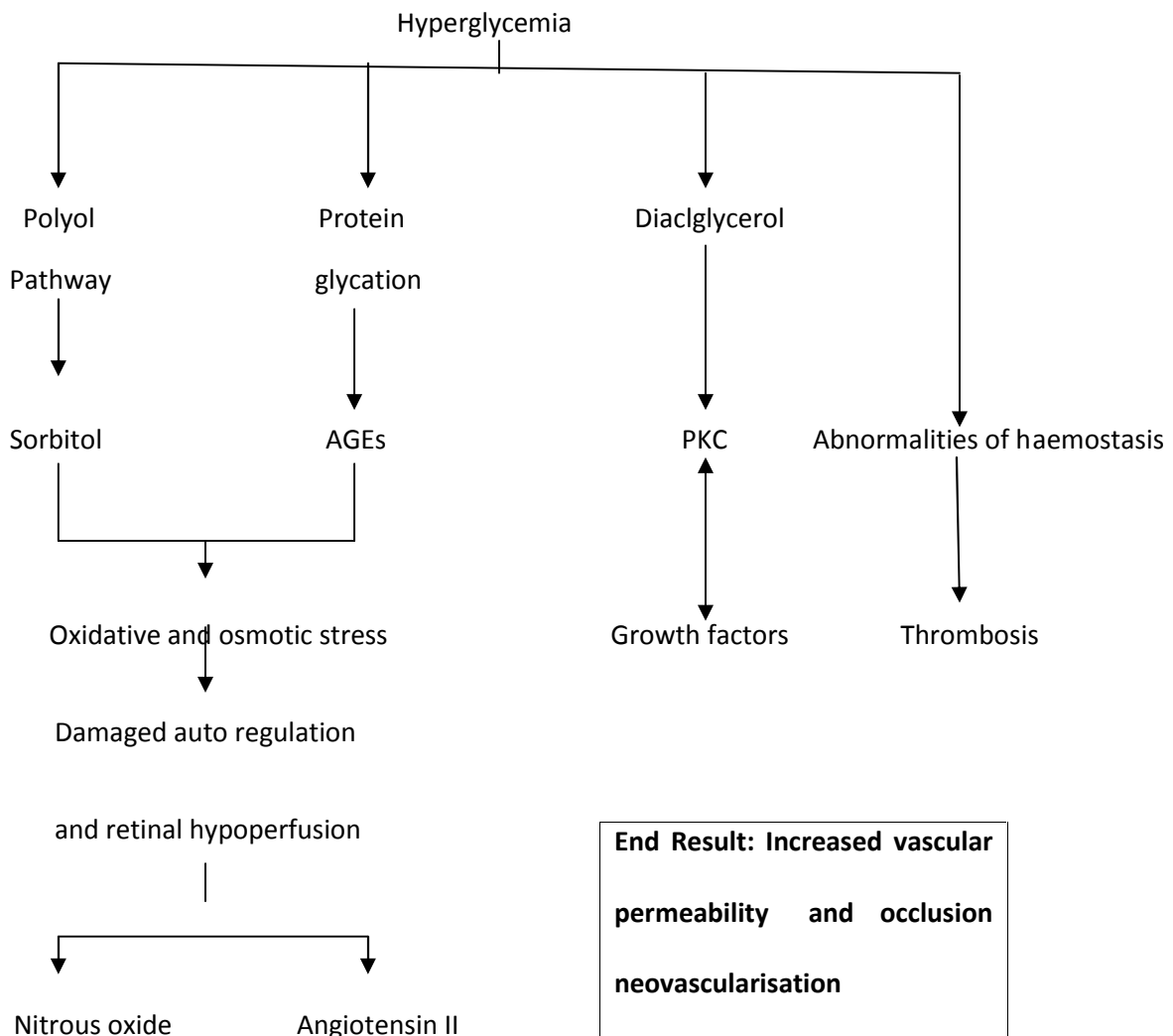


Table No. 2 : Effect of Hyperglycemia on pathogenesis of DR

CLASSIFICATIONS OF RETINOPATHY:

Many classifications have been proposed for diabetic retinopathy. Some of these are

I. Hirschberg's Classification :

In 1980 Hirschberg⁴⁷ recognised three following types

1. Inflammatory – characterised by spots and haemorrhages
2. Haemorrhagic
3. Pigmentary

II. Ballantyne and Michaelson's Classification:

Ballantyne and Michaelson⁴⁸ divided diabetic retinopathy into 5 stages

1. Microlesions –comprised of microaneurysms
2. Macrolesions –dot and blot haemorrhages
3. Vascular changes-Gross changes in veins
4. Destructive changes-intraocular haemorrhages, retinitis proliferans, RD,Glaucoma
5. Mixed forms- diabetic changes associated with arteriosclerosis and /or hypertension.

III. Scott's Classification :⁴⁹

Stage 1a - capillary microaneurysms

Stage 1b - phlebosclerosis; loops; coils; or knots in distended veins

Stage 11a - punctuate haemorrhages with or without discrete flecks of exudates

Stage 11b - larger round or blot haemorrhages, with confluent exudates

Stage 111a - more numerous haemorrhages and exudates

Stage 111b - haemorrhage into the vitreous

Stage 1V - Retinitis proliferans, retinal detachment, gross degenerative changes.

IV. Alaert's and Slosse's Classification :

They suggested a classification⁵⁰ which allows the fact that in some cases there is preponderance of vascular whereas in others exudative lesions are seen, although in most cases they occur side by side.

V. Lee's Classification⁵¹:

1. Angiopathy type-venous dilation; microaneurysms, haemorrhage & neovascularisation
2. Exudative type
3. Proliferative retinopathy
4. Vitreous haemorrhage

VI. Duke Elder Classification⁴⁷ :

1. Pre retinopathic stage- characterised by uniform distension and turgescence of Veins
2. Simple diabetic retinopathy- characterised by microaneurysms, exudates, haemorrhage and venous changes.
3. Proliferative diabetic retinopathy- formation of new vessels in the retina and proliferation into the vitreous

VII. Peyman's Classification⁵²:

1. Non proliferative/simple diabetic retinopathy- characterised by microaneurysms, dot and blot haemorrhages ,exudates.
2. Proliferative/malignant diabetic retinopathy-characterised by new vessel and glial tissue proliferation.

VIII. Clinical classification of diabetic retinopathy-Kanski ⁵³:

1. Background diabetic retinopathy
2. Diabetic maculopathy- focal; diffuse; ischaemic
3. Pre proliferative diabetic retinopathy
4. Proliferative diabetic retinopathy
5. Advanced diabetic eye disease
 - persistent vitreous haemorrhage
 - retinal detachment
 - opaque membrane formation
 - neovascular glaucoma

IX. Modern ETDRS :

The ETDRS in the 1980s developed a staging system which is recognised as the gold standard for clinical trials. The ETDRS classification of DR⁵⁴ is based on the grading of fundus photography of seven stereoscopic fields and the severity of the retinopathy is determined after assessment of these photographs. Each ETDRS grade can predict the prognosis for developing sight-threatening retinopathy. In particular IRMA and venous beading have been identified as high risk for the development of PDR, and cotton wool spots are low risk. The ETDRS grading scheme is too complex to be easily used in clinical practice. Therefore an adaptation of the ETDRS classification is now widely used. This classification system translates features identified by ETDRS as high and low risk into clinical practice. The level of retinopathy represents the risk of developing sight threatening DR and is used to plan follow-up and treatment.

Levels of Retinopathy:

NPDR

A. Mild NPDR

At least one microaneurysm

Definition not met for B, C, D, E, F

B. Moderate NPDR

Standard photograph Fig. No. 1

SE (CWS), VB, and IRMAs definitely present

Definition not met for C, D, E and F

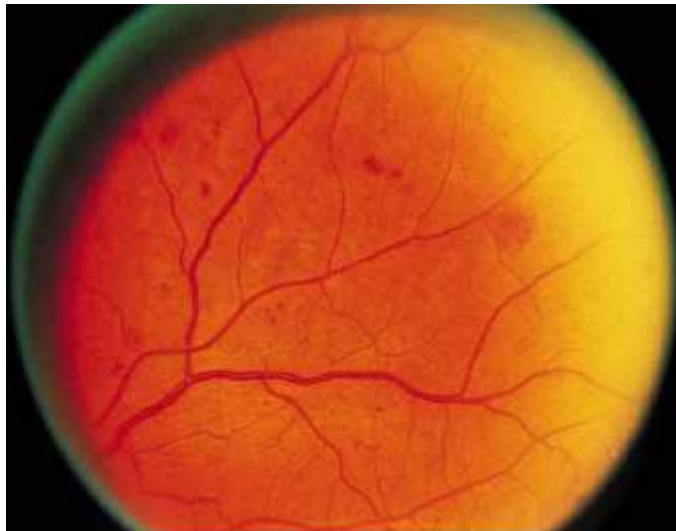


Fig No. 1 showing Moderate NPDR

C. Severe NPDR

H/Ma standard photograph Fig. No. 1 in all four quadrants

VB in two or more quadrants (Fig. No. 2)

IRMA standard photograph (Fig No. 3) in at least one quadrant

D. Very Severe NPDR

Any two or more of C

Definition not met for E, F.

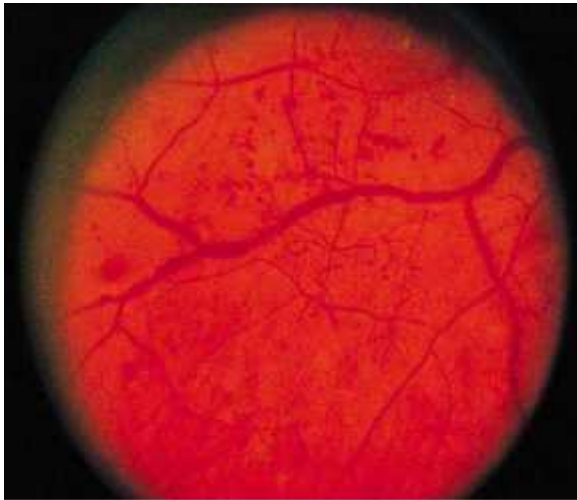


Fig No. 2: Severe NPDR

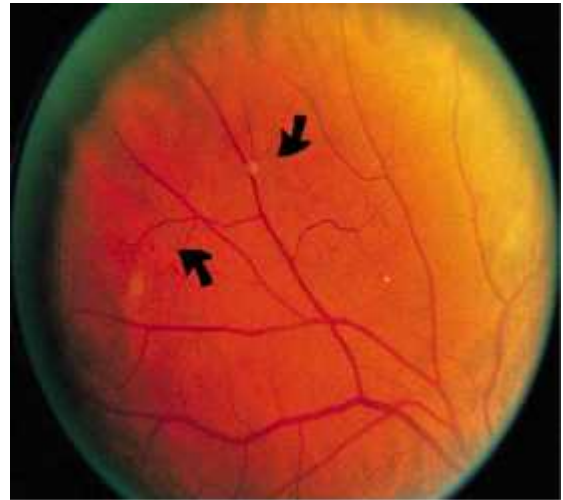


Fig No .3 : VB in ≥ 2 Quadrants

PDR

(Composed of: [1] NVD or NVE, [2] preretinal or vitreous haemorrhage,
[3]fibrous tissue proliferation)

E. Early PDR- New vessels

Definition not met for F

F. High-risk PDR

NVD 1/3-1/2 disc area (Fig. No. 4) or

NVD and vitreous or preretinal haemorrhage or

NVE 1/2 disc area and preretinal or vitreous haemorrhage

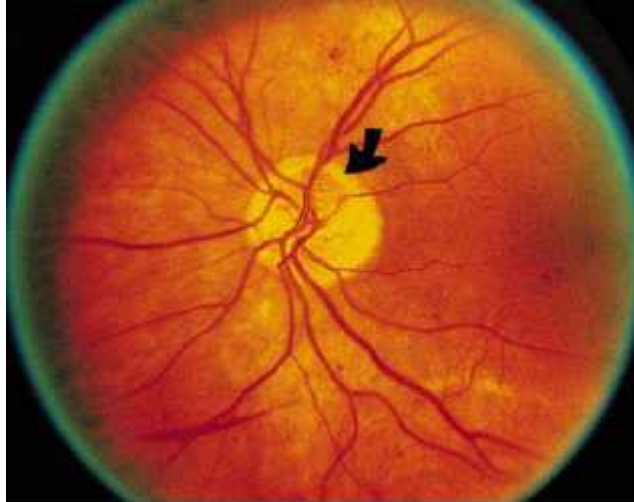


Fig No. 4: High risk PDR

X. International Clinical Diabetic Retinopathy Disease Severity Scale ⁵⁵:

| Disease Severity Level | Clinical features |
|-------------------------------|---|
| No apparent retinopathy | No abnormalities |
| Mild NPDR | Microaneurysm only |
| Moderate NPDR | More than just microaneurysms, but less than severe NPDR |
| Severe NPDR | Any of the following: >20 intra-retinal hemorrhages in each 4 quadrants definite venous beading in ≥ 2 quadrants and no signs of PDR |
| Very severe NPDR | All of the following: >20 intra-retinal hemorrhages in each of 4 Quadrants definite venous beading in ≥ 2 quadrants prominent IRMA in ≥ 1 quadrant |
| PDR | One or more of the following: neovascularisation vitreous/pre-retinal hemorrhage |

Table No. 3: International Clinical Diabetic Retinopathy Disease Severity Scale

CSME :

1. Thickening of the retina located 500 µm from the centre of the macula or
2. Hard exudate with thickening of the adjacent retina located 500 µm from the centre of the macula or
3. A zone of retinal thickening, 1 disc area or larger in size located 1 disc diameter from the centre of the macula.

Diabetic macular oedema: divided into **two broad levels:**

Macular oedema apparently absent- no apparent retinal thickening or hard exudates in the posterior pole and Macular oedema apparently present- some retinal thickening or hard exudates in the posterior pole.

The below table shows the classification system for retinopathy and maculopathy; adapted from Scottish DRS grading scheme.

| Retinopathy | Description |
|--------------------|--|
| RO | No DR |
| R1 mild | Background DR The presence of ≥ 1 of the following: dot hemorrhages microaneurysms hard exudates cotton wool spots superficial/flame shaped hemorrhages |
| R2 (Observable) | Background DR-observable ≥ 4 blot haemorrhages in 1 hemifield only |

| | |
|---------------------|---|
| R3 (referable) | Background DR-referable Any of the following: > 4 blot hemorrhages in both superior & inferior fields venous bleeding IRMA |
| R 4 (proliferative) | PDR Any of the following: active new vessels vitreous hemorrhage refer ophthalmology |
| Maculopathy | Description |
| MO | No blot haemorrhages/hard exudates ≤ 2 disc diameters of the centre of fovea |
| M1 (observable) | Blot hemorrhages /hard exudates within a radius of >1 but <2 disc diameters of centre of fovea |
| M2 (referable) | Blot haemorrhages /hard exudates within a radius of ≤ 1 disc diameter of the centre of fovea |

Table No. 4: Classification system for retinopathy and maculopathy.

Lesions of diabetic retinopathy:

Micro aneurysms: Originally noted by Mackenzie and Nettleship - Seen as red, round intraretinal lesions. Appear to derive from retinal capillaries and found in vicinity of occluded capillaries. Microaneurysms are clinically visible only when they are above 30 microns and the upper limit is 125 microns. More microaneurysms are seen on fundus angiography than clinically. On angiography they appear as hyperfluorescent dots. Generally they fill during early venous stage and some may leak dye to the surrounding retina.

Intraretinal haemorrhages: This appears secondary to ruptured microaneurysms, capillaries and venules. The shape of the intraretinal haemorrhage depends on the location within the retinal layer. Dot and blot haemorrhage – located in deeper layers of retina (between outer plexiform and inner nuclear layer of the retina) Flame shaped haemorrhages – located in the superficial nerve fibre layer Intraretinal haemorrhages resolve in 6-12 week period.

Hard exudates: These are collection of lipid adjacent to microvascular leakage. Intraretinal exudates appears as creamy – yellow flakes, flecks or dots. They may be arranged in individual streaks, in clusters, or in large circinate rings (if so almost always surround leaking microaneurysms or areas of capillary non perfusion). The hard exudates have an affinity for the posterior pole, particularly the macula .⁵⁶

Soft exudates: Also known as cotton wool spots, it is seen secondary to arteriolar closure. It occurs in more advanced NPDR. These are small infarcts of nerve fibre layer. These appear as fluffy white/yellow white striations of nerve fibre layer. Cotton wool spots generally resolve in 2-3 months.

Venous beading and loops

Represent focal areas of venous dilatation. Other venous abnormalities include loops and reduplication of venous segments. The degree of venous beading in ocular fundus images has been shown to be a more powerful predictor of conversion to proliferative diabetic retinopathy than any other type of retinal abnormality. Further, the degree of venous beading has been shown to be well correlated with disease progression. Reduplication of veins is the least common retinal abnormality but is of the most important prognostic significance. These changes are associated with capillary non perfusion and retinal ischemia.⁵⁷

Foveal Avascular Zone Abnormalities: Normally FAZ is approximately 350 to 750 microns in diameter. Abnormalities seen in the FAZ are irregular intercapillary spaces. Decrease vision without macular oedema warrants a suspicious outlook towards the FAZ.

Intraretinal microvascular abnormalities: Intraretinal microvascular abnormalities (IRMA) represent either new vessel growth or remodelling of pre-existing vessels through endothelial cell proliferation within the retinal tissues to act as shunts through areas of nonperfusion. They are irregular, segmental dilatation of the capillary bed running between the arteriole and venule adjacent to capillary non perfusion area. They

stain on fluorescein angiography, but usually do not leak fluorescein. These eyes are at high risk of developing PDR. ⁵⁸

Proliferative lesions: Proliferative diabetic retinopathy .As a response to retinal ischemia there is development of extra retinal fibrovascular proliferation. For the diagnosis of PDR the following lesions are necessary: presence of newly formed blood vessel and/or presence of fibrous tissue arising from retina or optic disc and extending along the inner surface of the retina or into the vitreous cavity. These immature vessels have a propensity to bleed. They also cause development of firm adhesion between retina and vitreous body, and when they contract can cause tractional retinal detachment. ⁵⁸

Neovascularisation of disc (NVD): It is seen as fine vessels bridging across physiological cup. NVD has a tendency to follow temporal arcade. Grows between the internal limiting membrane of the retina and posterior hyaloids face of the vitreous and adheres to it. As NVD matures fibrous tissue grows to accompany it. With time the fibrovascular bridge may form linking the superior and inferior temporal arcade. ⁵⁸



Fig No. 5: Neovascularisation of disc

New vessels arising from elsewhere (NVE): Seen as wheel like networks with vessels radiating like spokes from centre and circumferential vessel bounding the periphery. When vitreous haemorrhage does occur it assumes a classic boat like configuration.

High risk characteristics:

NVD -1/4th TO 1/3rd in extent

NVD with pre-retinal haemorrhage

NVE with vitreous haemorrhage

Advanced diabetic eye:

Advanced diabetic eye disease is the end stage of proliferative retinopathy defined by any of the following potentially blinding complication of proliferative diabetic retinopathy. ⁵⁹

- Severe vitreous haemorrhage

- New vessels in the iris

- Neovascular glaucoma

- Tractional retinal detachment

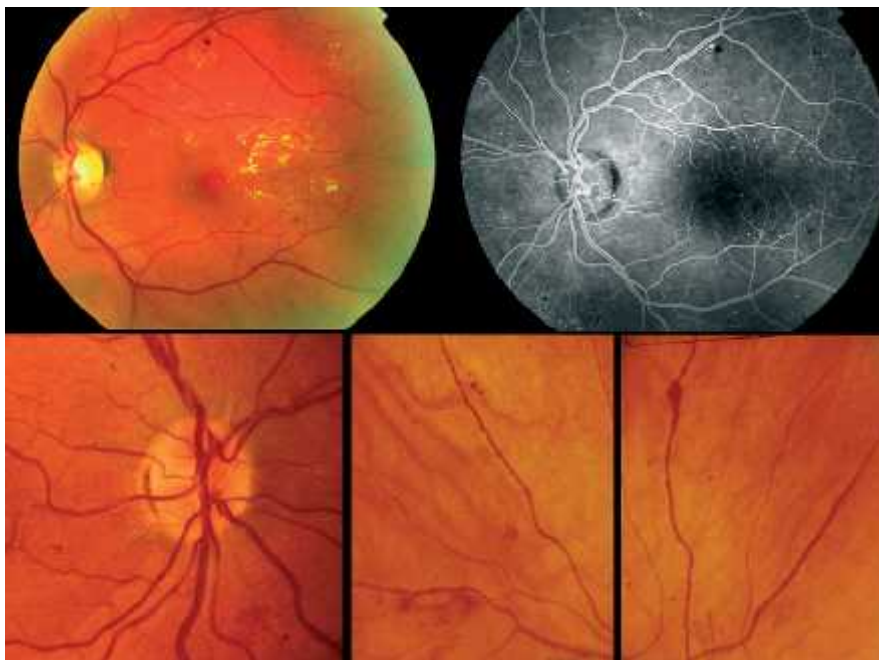


Fig No. 6: Non-proliferative diabetic retinopathy Cardinal signs are retinal microaneurysms, haemorrhages, and hard exudates ; and intraretinal microvascular abnormalities; venous beading ; and venous loop formation.

| Preretinopathy | Mild NPDR | Moderate NPDR | PDR | Advanced DED |
|-----------------------|---------------------------|----------------------|-----------------------------|---------------------|
| | ⇒ | ⇒ | ⇒ | ⇒ |
| Abnormalities of: | Microaneurysms | Venous bleeding | NVE | NVI |
| Electrophysiology | Intra-retinal hemorrhages | Venous loop | NVD | NVG |
| Psychophysics | Cotton wool spots | Cotton wool spots | Fibrovascular proliferation | Vitreous hemorrhage |
| Retinal blood flow | Venous dilatation | Cluster hemorrhages | | TRD |
| Vascular permeability | Exudates | IRMA | | |

Table No.5: Progression of diabetic retinopathy

Glycemic Control:

Glycemic control has a strong influence on many indices of diabetic retinopathy such as prevalence of retinopathy, incidence of retinopathy, progression of retinopathy, need for focal and scatter photocoagulation, and loss of visual acuity.⁶⁰ The influence of glycemic control is apparent in both type 1 and type 2 diabetes. The concept of a laboratory cutpoint for blood glucose normality is vague. However, 5–9.8% of patients over age 40 in developed countries have typical lesions of diabetic retinopathy even though they do not meet criteria for diabetes.⁶¹ Retinopathy consistent with diabetic retinopathy can develop in certain patients whose blood glucoses range in the normal range for the population; in adults over the age of 49, the 5-year incidence of such an event is 10%. This may in part reflect increased genetic susceptibility to the effects of hyperglycemia. Responsiveness to treatments for manifestations of diabetic retinopathy may also depend on glycemic control. Failure of DME to respond to focal/grid laser photocoagulation has been associated with higher glycosylated hemoglobin.⁶²

INVESTIGATIONS for a case of diabetic Retinopathy:

1. Regular:

| Technique | Advantages | Disadvantages | Recommendations |
|--|--|--|--|
| Direct Ophthalmoscopy | -Mobile -Inexpensive | -Requires pupil dilatation -Small field -Low sensitivity -Less effective than slit- lamp biomicroscopy | -Optional for Screening -Pupils must be dilated |
| Indirect Ophthalmoscopy | -Mobile -Large field -Relatively inexpensive | -Requires pupil dilatation -small abnormalities may be missed -Less effective than slit-lamp biomicroscopy | -Optional for Screening -Pupils must be Dilated |
| Slit-Lamp Biomicroscopy | -Large field | -Requires pupil dilatation -Immobile -Requires special lenses | -Required for ophthalmologic examination |
| Non Mydriatic retinal photography | -Large field -non-medical staff can be trained to use -can be digitally stored | -Relatively inexpensive -dark space required | Recommended for screening |

Table No. 6: Investigations available for Diabetic Retinopathy

2. Fluorescein angiography:

Fluorescein angiography ranks as one of the crucial diagnostic aids for detecting ocular pathophysiologic mechanisms. Chao and Flocks provided the earliest description of fluorescein angiography in 1958 and it was introduced into clinical use in 1861 by Novotny and Alvis.⁶³ The interaction of phthalic acid anhydride and resorcinol results in the formation of sodium fluorescein (C₂₀ H₁₀ O₅ Na₂). It has a low molecular weight. It is highly soluble in water. It absorbs light maximally between 480 to 510nm wavelength whilst its emission spectrum peaks at 530nm. Toxic reaction to fluorescein is rare. These mainly include nausea, vomiting, urticaria, skin rashes; hypotension and shock.

The various phases of fluorescein angiography normally observed are as follows.

1. Choroidal phase

- 8 to 10 seconds after injection of dye in antecubital vein. (arm to retina time)
- Seen as a diffuse fluorescent flush
- Patchy filling may be seen

2. Retinal arterial phase

- 8.5 to 11.0 seconds after injection of dye in antecubital vein.
- Temporal artery fills somewhat earlier than the nasal one and the upper artery slightly before the lower one.

3. Capillary phase

- Around the disc are seen the radial peri-papillary capillaries.
- Macula shows the typical capillary pattern with the central avascular Zone (500 microns in diameter).
- No special pattern can be recognised in the middle and far fundus periphery.

4. Venous phase

- Earliest venous filling occurs at the posterior pole.

5. Recirculation phase

- Begins within the first minute after injection.

In diabetic retinopathy, fluorescein angiography is useful for early detection of neovascularization, areas of capillary non perfusion, intraretinal microvascular abnormalities, microaneurysms etc. It is also done before the retina is subjected to photocoagulation.

3. OCT

The development of imaging equipment is allowing new features of diabetic macular oedema to be identified, which may have implications for treatment in the future.⁶⁴ OCT has identified macular pathologies that cannot be observed by clinical examination and therefore have not been part of prognosis and treatment planning in the past.

TREATMENT OF DIABETIC RETINOPATHY

- I. Medical line of treatment
- II. Photocoagulation
- III. Surgical line of treatment

I. Medical line of treatment

Control of diabetes by oral hypoglycaemic drugs, conventional insulin treatment or continuous insulin pumps play an important role. Clofibrate has been claimed to be useful in clearing retinal hard exudates.⁶⁵ Calcium dobesylate has been shown to have a beneficial effect on abnormal retinal capillary permeability.

| Agent | Molecules tried |
|-------------------------------|----------------------------|
| Antiplatelet/anti-coagulants | Aspirin Ticlopidine |
| Aldolase reductase inhibitors | Ponalrestat Tolrestat |
| GH suppressors | Octreotide |
| Anti-angiogenesis | Curcumin |
| ACE inhibitors | Candesartan Perindopril |
| PKC-beta inhibitors | Ruboxistaurin |

Table No. 7: Potential molecules for the management of diabetic retinopathy

II (i) Panretinal Photocoagulation (PRP)

a. Pretreatment Discussion with Patients:

Patients usually need numerous follow-up visits and may require supplementary laser treatment.

- PRP reduces the risk of visual loss and blindness.
- Although laser treatment is effective, some patients may still develop vitreous hemorrhage. The hemorrhage is caused by the diabetes and not by the laser; it may mean the patient needs more laser treatment.
- Laser treatment often reduces peripheral and night vision; treatment may moderately reduce central vision. This short-term side effect is compensated by the significant long-term reduction in severe vision loss and blindness in laser-treated patients.

b. Lenses for PRP:

The three-mirror Goldmann contact lens has a central opening for treating the posterior pole, and side mirrors for treating the mid peripheral and peripheral retina. Disadvantages: small field of view, which requires continual manipulation of the lens to complete treatment. Spot size is set at 500 μ m.

Newer wide-angle contact lenses are often used. Although the image is inverted, there is a large field of view allowing for many burns with the field while easily maintaining orientation to the disc and macula. The optics of these wide-angle lenses will affect the laser spot size on the retina. Wide-angle indirect ophthalmoscopy lenses provide an inverted image, but show a large field of view and a magnification of the spot in the retina. Scatter treatment can be applied to a large area of retina in a single image, and it is easy to visualize the disk and the macula.

c. Technique for PRP:

- i. The pupil should be fully dilated and topical anesthesia is used. Retrobulbar subtenons anesthesia to reduce pain and decrease eye motion can be employed as necessary.
- ii. The most common wavelengths used are Argon green, blue green (generally avoided currently), and 532 green laser, using the slit-lamp delivery system. In case of hazy media, Krypton red or diode red laser (814 nm) can be used. Slit-lamp treatment is most commonly done through a contact lens but can also be performed using indirect ophthalmoscopy. For example, when treatment is given under general anesthetic.

- iii. Typical initial settings on the Argon laser would be 500 μm spot size, a 0.1 second exposure and 250-270 mw power. The power is gradually increased until a whitish reaction is obtained on the retina. The lesions are placed 1 burn width apart.
- iv. A total of 1600-3000 burns are placed in 1 or more sittings, carefully avoiding the macular area and any areas of tractional elevation of the retina. The burns are placed 2 to 3 disc diameters away from the center of the macula and 1 disc diameter away from the disc, usually outside the arcades and extended peripherally up to the equator and beyond.
- v. Laser treatment should not be applied over major retinal veins, preretinal hemorrhages, darkly pigmented chorioretinal scars, or within 1 DD (200-300 μm) of center of macula, so as to avoid risk of hemorrhage or large scotomas.
- vi. Other considerations:
 - Additional photocoagulation is needed if there is evidence of worsening of proliferative DR.
 - Add laser burns in between scars of initial treatment further peripherally and also at the posterior pole, sparing the area within 500-1500 μm from the center of macula.
 - Favor quadrants with active new vessels or areas with intraretinal microvascular abnormalities where scars are more widely spaced and areas of severe ischemia not previously treated, such as the temporal part of the posterior pole.
 - Direct treatment of NVE in between scars is possible.

d. Panretinal (Scatter) Photocoagulation Technique Following Diabetic Retinopathy Clinical Research Network (DRCRNet) Consensus.

Panretinal (scatter) photocoagulation initially consists of 1200 to 1600 burns (or the equivalent area treated with a multi-spot laser), with a spot size on the retina of approximately 500 μm given over 1 to 3 sittings and completed within eight weeks days of initiation, As shown in the below table No.8.

| Burn Characteristic | Direct/Grid Photocoagulation(Modified ETDRS technique) | Mild Macular Grid Photocoagulation technique |
|--|--|--|
| Direct treatment | Directly treat all leaking microaneurysms in areas of retinal thickening 500 to 3000 μm from centre of the macula | Not applicable |
| Change in MA color with direct treatment | Not required, but atleast a mild gray white burn should be evident beneath all microaneurysms. | Not applicable |
| Burn size for direct treatment | 50-100 μm | Not applicable |
| Burn duration for direct treatment | 0.05-0.1 sec | Not applicable |
| Grid treatment | Applied to all areas with diffuse leakage /non-perfusion within area described below for treatment | Applied to entire area described below for treatment |

| | | |
|--------------------------------------|---|---|
| Area considered for grid treatment | -500-300 μm superiorly, nasally and inferiorly from center of macula. -500-3500 μm temporally from macular center. -No burns are placed $\leq 500\mu\text{m}$ of disc | -500-300 μm superiorly, nasally and inferiorly from center of macula. -500-3500 μm temporally from macular center. -No burns are placed $\leq 500\mu\text{m}$ of disc |
| Burn size for grid treatment | 50-100 μm | 50 μm |
| Burn duration for grid treatment | 0.05-0.1 sec | 0.05-0.1 sec |
| Burn intensity | Barely visible (light gray) | Barely visible (light gray) |
| Wavelength(grid and focal treatment) | Green to yellow | Green |

Table No.8: Modified-EDTRS and the Mild Macular Grid Photocoagulation Techniques

II(ii)Laser photocoagulation

Focal laser

Possible mechanisms of action

- Direct closure of leaking microaneurysms
- Laser induced endovascular thrombosis
- Heat-induced contraction of the vessel wall Procedure – compare vascular landmarks on fluorescein angiograms are compared to the fundus.
 - Fundus is precisely located.
 - All fluorescein leaks are directly treated.
 - Laser burns are spaced one burn width apart.

- The end point is whitening or darkening of the treated area.
- Laser parameters- 100-200 microns spot size, 0.1 second duration, 50-100 Mw power

II (iii) Grid laser

Photoreceptor destruction increases inner retinal oxygenation resulting in vasoconstriction, decreased retinal blood flow and decreased vascular leakage

- Retinal pigment epithelium damage causes retinal capillary and venule endothelial dysfunction.
- Photocoagulation debridement of dysfunctioning RPE cells may result in enhanced blood retinal barrier
- Grid photocoagulation is performed in areas of thickened retina showing diffuse leakage or capillary dropout.
- Laser parameters- 100-200 micron spot size, 0.1 to 0.5 sec duration, 50- 100mw power, light intensity burns.
- Grid is not done within 500 microns of the macula/disc margin

Pan retinal photocoagulation for proliferative diabetic retinopathy:

It may alter the extracellular modulating factors responsible for control of intraocular neovascularisation. Destruction of oxygen consuming photoreceptors and RPE may improve inner retinal oxygenation, thus decreasing stimulus for vasoproliferative factors

Procedure – laser spots are placed outside the arcades and extend peripherally up to the equator and beyond

- Laser settings- 300-500micron spot size, 0.1 second, 220-27- mw power
- Power and duration are adjusted till gray white reaction is achieved
- Burns are placed one burn width apart; a total of 1600-2000 burns are applied in 2-3 sittings starting first with the inferior quadrant, because if vitreous haemorrhage occurs then the inferior quadrant will be difficult to treat.

| | |
|-------------------------------------|---|
| Size(on retina) | 500 μ m |
| Exposure | 0.1sec recommended |
| Intensity | Mild white(i.e. 2+ to 3+) |
| Distribution of sessions | Edges 1 burn width apart |
| Number | 1-3 |
| Nasal proximity to disk | No closer than 500 μ m |
| Temporal proximity to center | No closer than 3000 μ m |
| Superior/inferior limit | No further posterior than 1 burn within temporal arcades |
| Extent | Arcades (3000 μ m from macular center) to atleast the equator |
| Total number of burns | 1200-1600n |
| Wavelength | Green or yellow(red can be used if vitreous hemorrhage) |

Table No. 9: The burn characteristics for panretinal photocoagulation

III. Surgical line of treatment

Pituitary ablation by yttrium 90 implantation or hypophysectomy advocated as a treatment for proliferative retinopathy following a case report by Poulstein in 1953 Kohner and co-workers have advocated pituitary ablation as a treatment of choice for diabetic retinopathy characterised by progressive capillary dilatation, capillary occlusion, cotton wool spot formation and severe proliferative diabetic retinopathy. ⁶⁶

(i) Vitrectomy forms a mainstay of the surgical line of proliferative treatment of diabetic retinopathy with its attendant complications.⁶⁷

Indications for vitrectomy in severe diabetic retinopathy

(a) Media opacity

- Non clearing vitreous haemorrhage
- Pre macular haemorrhage

(b) Traction

- Progressive fibrovascular proliferation
- Tractional retinal detachment involving macula
- Combined rhegmatogenous and tractional retinal detachment
- Macular oedema associated with taut posterior hyaloids

(c) Others

- Recurrent vitreous haemorrhage
- Anterior hyaloid fibrovascular proliferation
- Goals of vitrectomy in proliferative diabetic retinopathy
- To clear the media
- To relieve all tractions on the retina
- To reattach retina where needed
- Closure of retinal breaks if any

The aims of vitrectomy:

1. To clear media opacities such as vitreous haemorrhage;
2. To remove proliferative tissue causing tractional forces on the retina;
3. Prevent further neovascularization by laser endophotocoagulation and by removal of vitreous gel so removing the scaffold along which fibrovascular tissue can proliferate;
4. Repair retinal detachment by excising tractional membranes and removing fibrovascular tissue from the surface of the retina.

Surgical technique:

The central core vitreous gel is first removed with the vitrectomy probe. If there is no posterior vitreous separation then this is next elevated in an accessible area without tight vitreo-retinal adhesion. The optic disc is often a good location for this if no posterior vitreous separation exists elsewhere. The neovascular membrane complexes are now dissected away from the retina being careful to ensure that the dissection is in the correct plane and not in a false plane of vitreo-schisis, which can occur in PDR. This can be achieved using an 'en-bloc' dissection then delamination technique using the remaining posterior hyaloids face to exert antero-posterior traction to open up the plane of dissection . Alternatively via circumcision from the posterior hyaloid face and then segmentation of neovascular complexes followed by their delamination from the surface of the retina. 'En-bloc' dissection and delamination involve the use of horizontal cutting scissors to cut through vascular 'pegs' joining the membrane complexes to the retina. Segmentation may involve vertical cutting scissors to divide membranes between pegs in order to relieve traction between these. Once the traction has been relieved the retina is

carefully inspected for signs of retinal breaks. If identified these are treated with laser to form a surrounding chorioretinal adhesion and then gas or oil is used as tamponade. PRP is completed if deficient using indirect or endolaser and often continued up to the pars plana, which possibly reduces the rate of post vitrectomy bleeding.

Complications:

1. Post-vitrectomy vitreous cavity haemorrhage can occur in some patients.(10-20%)
2. Retinal tears and holes
3. Raised IOP
4. Cataracts can develop following vitrectomy surgery, especially in patients over 60 years.
5. Rubeosis-rare.

Visual prognosis post vitrectomy: Overall, about 70–90% of patients will get an improvement in vision with vitrectomy surgery with the final acuity result depending largely on the degree of maculopathy. Less than 5% may be made worse.

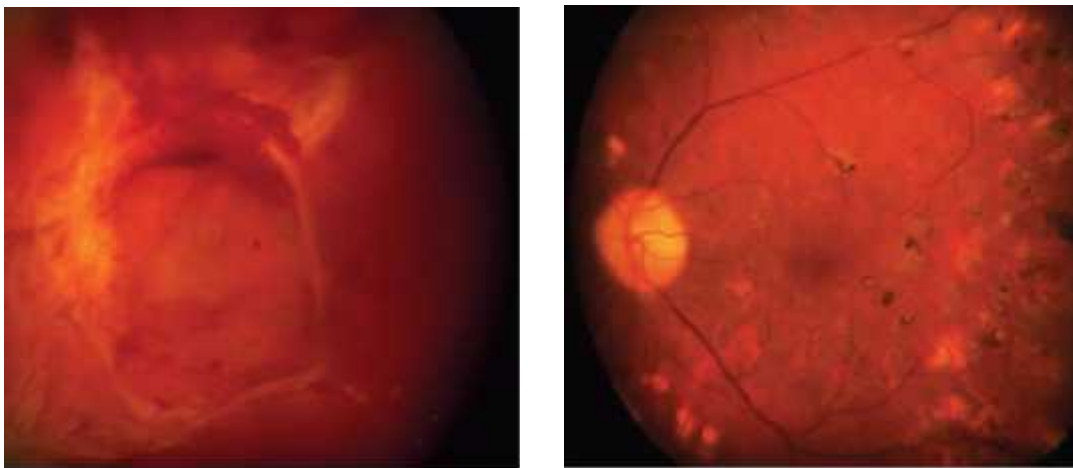


Fig No. 7: (a,b) Pre and post vitrectomy for vitreous haemorrhage and traction retinal detachment. VA postoperatively 6/12.

(ii) Intravitreal drugs:

Intravitreal triamcinolone has been used for macular oedema refractory to laser treatment.⁶⁸ Using OCT has shown this treatment to reduce retinal thickness. Improvement in vision has been recorded in some cases. However, this effect is transient and appears not to be sustained despite repeat injections. A recent randomised clinical trial showed that laser is more effective than intravitreal triamcinolone at preventing visual loss. There are also significant side-effects with intravitreal triamcinolone, with raised intraocular pressure and cataract being very common

Intravitreal injections of anti-VEGF such as ranibizumab, pegaptanib and bevacizumab are currently being explored as therapies for DR. Several trials have shown a beneficial effect when anti-VEGF agents are used alone or combined with laser for macular oedema.⁶⁹

A recent randomised trial of 854 eyes has shown that ranibizumab combined with laser for 'centre involving' diabetic macular oedema (ie DMO that involves the fovea) is more effective than laser alone.⁷⁰ At 1 year there was a significantly greater mean increase in visual acuity in eyes treated with ranibizumab and laser, than those who had only laser. Multiple injections were required, however, and were also necessary in the second year of the study, but the authors report no systemic side-effects attributable to the treatment. There is concern that these drugs can, though rarely, affect patients systemically to increase the risk of stroke and myocardial infarction. Additionally, VEGF may be necessary in maintaining the function of healthy retina. However, it is very likely that anti-VEGF therapy in combination with laser has a place in the management of both PDR and macular oedema.

| Severity of retinopathy | Presence of CSME | Follow-up (Months) | Scatter(Panretinal) Photocoagulation | Fluorescein Angiography | Focal laser |
|-------------------------------------|------------------|--------------------|--------------------------------------|-------------------------|--------------|
| Normal/ Mild NPDR | No | 12 | No | No | No |
| Mild to Mod NPDR | No | 6-12 | No | No | No |
| | Yes | 2-4 | No | Usually | Usually |
| Severe to very Severe NPDR | No | 2-4 | Sometimes | Rarely | No |
| | Yes | 2-4 | Sometimes | Usually | Usually |
| Non high-risk PDR | No | 2-4 | Sometimes | Rarely | No |
| | Yes | 2-4 | Sometimes | Usually | Usually |
| High risk PDR | No | 3-4 | Usually | Rarely | No |
| | Yes | 3-4 | Usually | Usually | Usually |
| High risk PDR Not amenable to PC | - | 1-6 | Not possible | Occasionally | Not possible |

Table No. 10: Management recommendations for patients with Diabetes retinopathy

The recommended follow up regimen for newly diagnosed diabetic patients is as given in table no.11

| | IDDM | NIDDM |
|---------------------------------------|--|--|
| 1st Examination | 5years after onset if patient's age < 30 yr. At puberty after diagnosis | On diagnosis immediately if patient's age >31 yr. |
| Follow-up | Annually* | Annually* |

* normal at first examination

Table No. 11: Ocular examination and follow up schedule in diabetes subjects

J. NEURO OPHTHALMIC ASSOCIATIONS:

Diabetics are at risk to develop cranial mononeuropathies particularly at the oculomotor, trochlear, abducens and facial nerves .The principal metabolic defect implicated are disruption of polyol-myoinositol-aldose reductase pathways, altered lipid metabolism, advanced glycosylated end product formation, increased oxidative stress and diabetes-induced defects in growth factors. These mononeuropathies are caused by microvascular infarction of the peripheral nerves in the brain. These patients usually recover over a period of 2 to 6 months without developing aberrant regeneration.

K. ORBIT:

Patients with diabetes mellitus are known to develop infectious processes including orbital cellulitis. Orbital cellulitis is characterised by proptosis, periorbital swelling, and ophthalmoplegias, including internal ophthalmoplegia. Most patients will have a concomitant sinus disease. Although generally the most common infective organism are bacterial, in any diabetic patient , zygomycosis (or other fungal infections) must be the leading diagnosis until excluded by biopsy. Mucormycosis is though known to occur in patients with ketoacidosis, all patients with diabetes mellitus are at risk of developing this complication

L. INTRAOCULAR PRESSURE:

Primary Open-Angle Glaucoma:

Diabetes was common in participants of the Barbados Eye Survey and participants of the Baltimore Eye Survey, it was unrelated to the prevalence of open angle glaucoma. ⁷¹ In the Beaver Dam Eye Study, older-onset diabetes (> 30 years of age) was associated with a modest increase in the risk of glaucoma. ⁷² In the Blue Mountains Eye

Study, there was a significant association between diabetes (diagnosed from history or from elevated fasting plasma glucose level) and open-angle glaucoma.⁷³ The Ocular Hypertension Treatment Study (OHTS) found that diabetes mellitus appeared to be protective against the development of POAG in patients with ocular hypertension.⁷⁴ When patients are treated medically for POAG, it is important to recognize that the potential side effects of beta-adrenergic antagonists include reduced glucose tolerance and masking of hypoglycemic signs. Therefore, this class of antiglaucoma medications should be used cautiously in diabetic patients.

Angle-Closure Glaucoma (ACG):

One study found that patients with ACG had a higher prevalence of abnormal glucose tolerance test results compared with POAG patients and controls.⁷⁵ Patients with ACG also have a high prevalence of non-insulin-dependent diabetes.⁷⁶ It has been hypothesized that, in some cases, ACG may be a symptom of diabetes, perhaps due to autonomic dysfunction.⁷⁷

Hyperosmotic agents are commonly included in the medical management of acute episodes of elevated IOP. In diabetic patients, isosorbide is preferred to glycerol because isosorbide is not metabolized into sugar, while glycerol is metabolized into sugar and ketone bodies. Glycerol, therefore, can produce hyperglycemia and, rarely, ketoacidosis in diabetic patients

Despite the widespread use of panretinal photocoagulation (PRP), proliferative diabetic retinopathy (PDR) remains a leading cause of neovascular glaucoma. In 1984, Brown and associates reviewed 208 cases of neovascular glaucoma and reported that

36% were caused by central retinal vein occlusion, 32% by diabetic retinopathy, and 13% by carotid occlusive disease.⁷⁸

The reported incidence of any neovascularization of the iris (NVI) ⁷⁹ among diabetic patients ranges from 1% to 17% . In the early stages, NVI usually appears as small vascular tufts either at the pupillary margin or in the anterior chamber angle. As these vessels later spread across the iris surface, they are frequently accompanied by fibrous tissue, which contracts and may cause ectropion uveae.

Management of primary open angle glaucoma in diabetes is same as in otherwise normal patients. If the intraocular pressure cannot be controlled by medical therapy alone, trabeculectomy is the surgery of choice. Glaucoma drainage surgery in diabetic patient is associated with poor prognosis due to increased scarring at the site of surgery, secondary to increased proliferation of tenons capsule.

M. OPTIC NERVE ABNORMALITIES:

Acute optic disc edema associated with diabetes, or diabetic papillopathy, usually occurs in the second to fourth decades of life and generally shows no correlation with the severity of diabetic retinopathy. It is typically associated with mild loss of vision (20/50), and the visual field may be normal or may show defects, such as an increased blind spot, arcuate scotoma, or altitudinal scotoma. Fluorescein angiography usually demonstrates diffuse leakage at the disc. The condition presents bilaterally in approximately 50% of cases,⁸⁰ while in other cases, the second eye may be affected as late as 3 years after initial presentation. The visual prognosis is usually good, with nearly all younger patients recovering to a visual acuity of 20/30. Visual field defects

infrequently persist. While the optic disc appearance usually returns to normal, occasionally, diffuse or segmental atrophy may result.

In diabetic papillopathy, diffuse disc swelling may mimic papilledema of raised intracranial pressure. However, careful visual field testing may demonstrate an arcuate or altitudinal defect, which would be unusual in papilledema. To avoid unnecessary PRP, it is important to differentiate the prominent telangiectasia of disc vessels often seen in diabetic papillopathy from neovascularization of the disc.

Diabetic papillopathy differs from anterior ischemic optic neuropathy (AION). Typical AION is generally seen in middle-aged to elderly frequently hypertensive persons with or without diabetes, and is characterized by acute unilateral moderate-to-marked loss of vision, swelling of the optic disc with variable nerve fiber layer hemorrhages, segmental areas of nonperfusion on fluorescein angiography, poor prognosis for visual recovery, and late optic disc pallor .

N. INFECTIOUS DISEASES:

Endophthalmitis.

The increased risk of postoperative endophthalmitis among diabetic patients is not surprising, because patients with diabetes have been demonstrated to have impaired cellular and humoral immune responses, as well as altered phagocytic capabilities. Further, it is well known that diabetic patients are more likely than nondiabetic patients to experience delayed wound healing . Thus, diabetic patients may be predisposed to wound breakdown or persistent wound defects or both, which, in turn, may increase their risk of developing endophthalmitis.

In a case-control study of endophthalmitis following secondary intraocular lens implantation, 50% of patients had a history of diabetes compared with 5.9% of control patients.⁸¹

Finally, vitrectomy for complications of PDR often requires longer surgical time and more instrument changes passing through the pars plana sclerotomies compared with vitrectomy for other diseases.

Mucormycosis. It is a rare orbital infection that affects diabetic patients, especially those with ketoacidosis. Orbital mucormycosis usually originates in adjacent sinuses and presents with complete internal and external ophthalmoplegia, decreased vision, proptosis, ptosis, and chemosis. Histopathologic hallmarks of the disease are vascular invasion and tissue necrosis. Clinically, affected areas are characterized by black eschars and discharge, although this may be a late finding. Mucormycosis is associated with a significant risk of mortality,⁸² which underscores the importance of prompt diagnosis and treatment with tissue debridement and amphotericin B.

MATERIALS AND METHODS

SOURCE OF DATA

This was a prospective observational study on patients attending outpatient department and those referred to department of Ophthalmology at _____ from October 2012 to 31st march 2014

METHOD OF COLLECTION OF DATA

- Patients were selected on the basis of history, clinical examination and blood investigations
- Patients were labeled as type 2 diabetes mellitus based on the criteria laid down by the American Diabetes Association.
- All subjects were interviewed as per the prepared proforma and the complete ophthalmic examination was done.
- Estimation of RBS at admission and FBS and PPBS second day of admission

SAMPLE SIZE:

With a prevalence rate of type 2 diabetes 9% at 95% confidence interval and +/- 3 margin of error, the calculated sample size was 349.

$$n=4pq/L^2$$

where ; n= sample size,

p= incidence rate,

q= 100-p,

L= allowable error.

STATISTICAL ANALYSIS:

Data was analyzed using following statistical method

- Diagrammatic presentation.
- Mean +/- SD

RESEARCH HYPOTHESIS:

It was an observational study to know the incidence and prevalence of various forms of diabetic eye disease in the study population.

Inclusion criteria

- a) Patient who has been diagnosed type 2 Diabetes Mellitus.
- b) Patients more than 30 years of age

Exclusion criteria

- a) Patient with type 1 diabetes
- b) Patients with known eye disease not related to diabetes.
- c) Patients with hypertension.
- d) Patients who have undergone treatment earlier for any form of diabetic eye disease

INVESTIGATIONS :

- Blood sugar levels(FBS & PPBS)
- Urine sugar, Albumin, Microscopy.
- HBA1C
- Slit Lamp Examination
- Fundoscopic Examination
- IOP
- Fundus Fluorescein Angiography if necessary

Does the study required any investigations or interventions to be conducted on patients or other humans or animals?

YES.

Had ethical clearance been obtained from your institution in case Of 7.3?

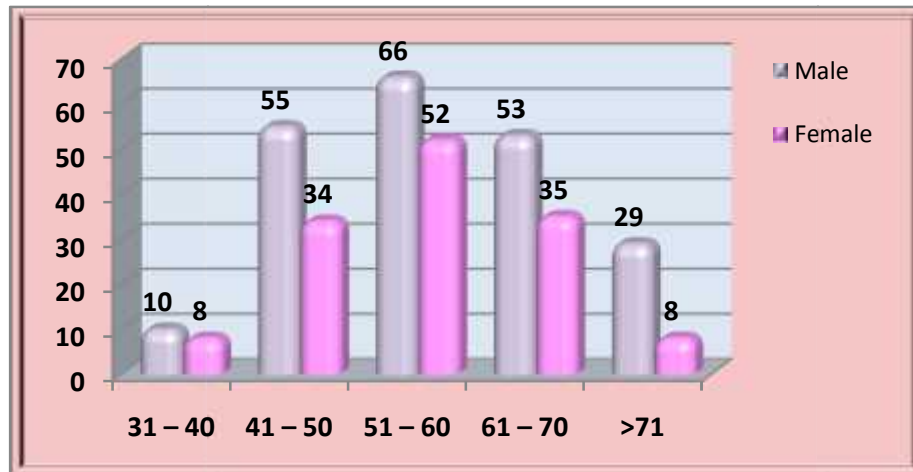
YES

OBSERVATIONS AND RESULTS

This observation study was conducted on totally 350 consecutive patients of NIDDM attending our institute. Out of 350, 211 were males (60.3%) and 139 (39.7%) were females. Among both the sexes, the age groups between 51 to 60 years had maximum number of patients (33.7%).

Table No. 12 .Distribution of Patients according to Age and Sex

| Age in years | Male (%) | Female (%) | Total (%) |
|---------------------|-----------|------------|------------|
| 31 – 40 | 10(4.7%) | 08(6.5%) | 18(5.1%) |
| 41 – 50 | 55(26.1%) | 34(24.5%) | 89(25.4%) |
| 51 – 60 | 66(31.3%) | 52(37.4%) | 118(33.7%) |
| 61 – 70 | 53(25.1%) | 35(25.2%) | 88(10.6%) |
| 71 and above | 29(13.7%) | 08(5.8%) | 37(25.1%) |
| Total | 211(100%) | 139(100%) | 350(100%) |

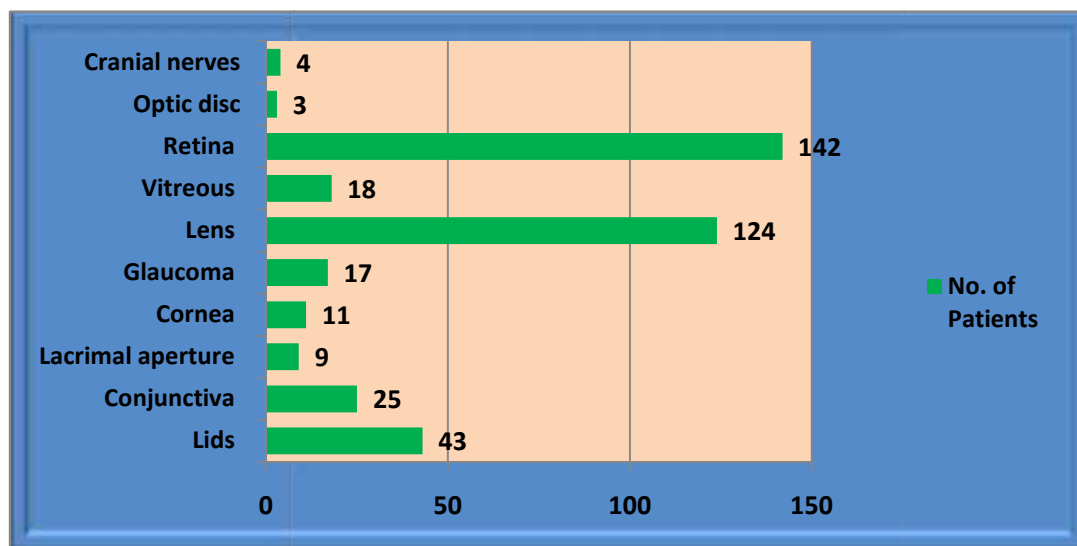


Graph No.1: Distribution of Patients according to AGE and SEX

TABLE NO. 13: We have accumulated data of all the possible diabetic ocular complications in this study population. It shows that the most common prevalent ocular complication is of retina: 142 patients (40.6%) followed by the lens 124(35.4%). We can observe that all the anatomical parts of eye are involved in diabetes.

Table No. 13. Overall prevalence of ocular involvement due to diabetes mellitus

| Sr. No. | Ocular morbidity | No. of Patients | Percentage |
|---------|-------------------|-----------------|------------|
| 1. | Lids | 43 | 12.3% |
| 2. | Conjunctiva | 25 | 7.1% |
| 3. | Lacrimal aperture | 9 | 2.6% |
| 4. | Cornea | 11 | 3.1% |
| 5. | Glaucoma | 17 | 4.6% |
| 6. | Lens | 124 | 35.4% |
| 7. | Vitreous | 18 | 5.2% |
| 8. | Retina | 142 | 40.6% |
| 9. | Optic disc | 3 | 0.9% |
| 10. | Cranial nerves | 4 | 1.1% |

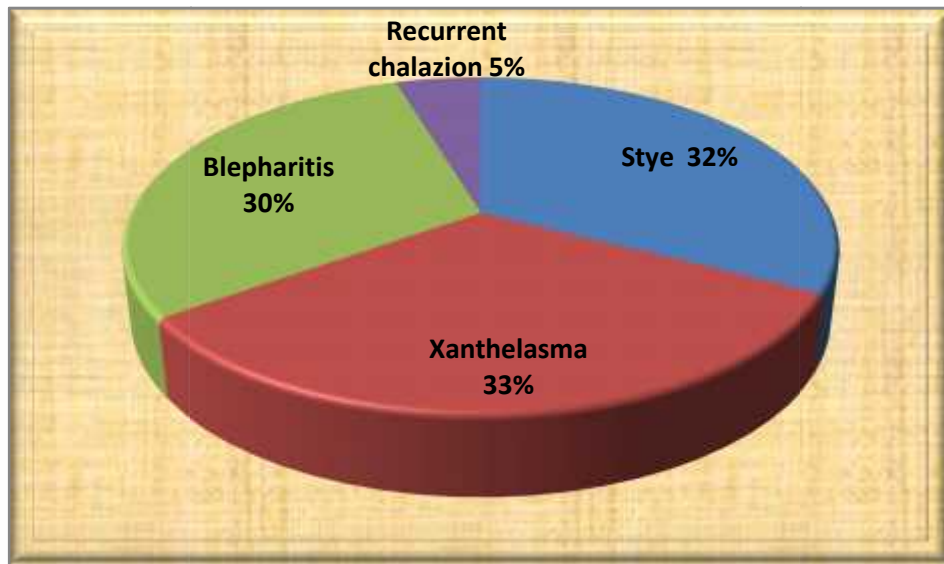


Graph No. 2: Overall prevalence of ocular involvement due to diabetes mellitus

TABLE NO. 14: 43 patients had some eyelid lesions, out of which 14 had Stye and another 14 had Xanthelasma, 13 had Blepharitis and 2 had recurrent chalazion.

Table No. 14. Distribution of patients according to type of lid lesion present

| Type of Lid Lesion | No. of patients | % |
|---------------------|-----------------|-------------|
| Stye | 14 | 4.0 |
| Xanthelasma | 14 | 4.0 |
| Blepharitis | 13 | 3.7 |
| Recurrent chalazion | 02 | 0.6 |
| Total | 43 | 12.3 |

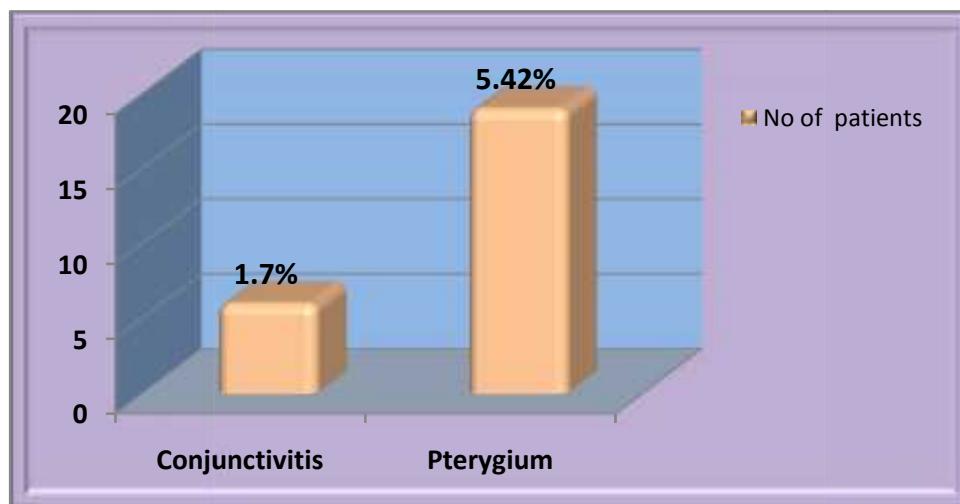


Graph No 3: percentage distribution of patients according to type of lid lesion.

TABLE NO 15: Conjunctival lesions found were pterygium in 19 patients (5.4%) and infective conjunctivitis in 6 patients (1.7%).

Table No. 15. Conjunctival lesions

| Conjunctival lesion | No of patients | % |
|---------------------|----------------|------|
| Conjunctivitis | 06 | 1.71 |
| Pterygium | 19 | 5.42 |

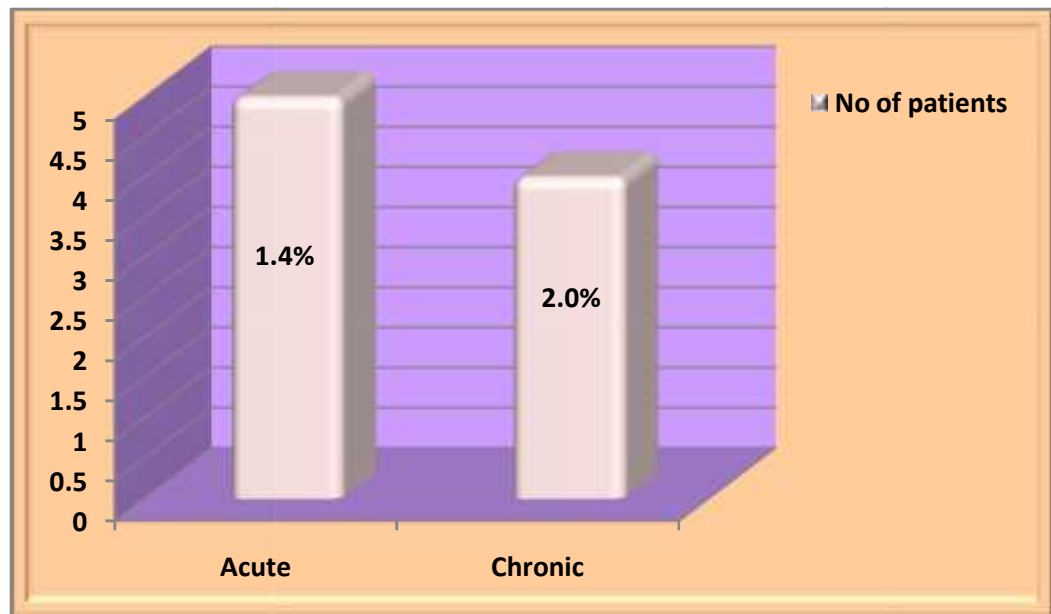


Graph No 4: Frequency and percentage distribution of conjunctival lesions

TABLE NO. 16: Both acute (1.4%) and chronic (1.1%) form of dacryocystitis is found

Table No. 16. Distribution of patients according to lacrimal system affection

| Type of Dacryocystitis | No of patients | Percentage |
|------------------------|----------------|------------|
| Acute | 5 | 1.4% |
| Chronic | 4 | 1.1% |
| Total | 9 | 2.6% |

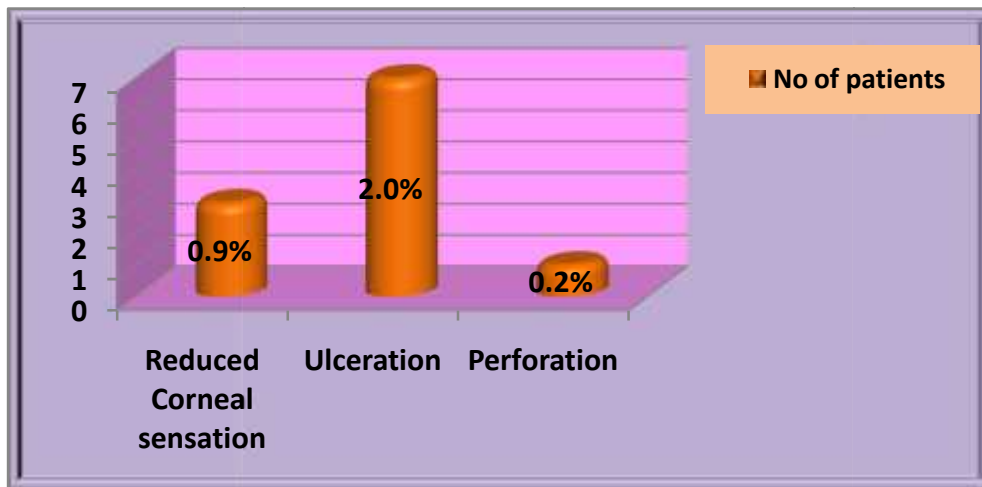


Graph no 5: Distribution of patients according to lacrimal system affection

TABLE NO 17 shows that 7 patients had corneal ulceration (2%), 3 had reduced corneal sensation (0.9%) while 1 patient (0.3%) had a corneal perforation . 4 out of these 7 ulcers were non healing bacterial type.

Table No. 17. Distribution according to type of corneal lesions present.

| Corneal lesion | No of patients | Percentage |
|---------------------------|----------------|------------|
| Reduced Corneal sensation | 03 | 0.9 |
| Ulceration | 07 | 2.0 |
| Perforation | 01 | 0.2 |
| Total | 11 | 3.1 |



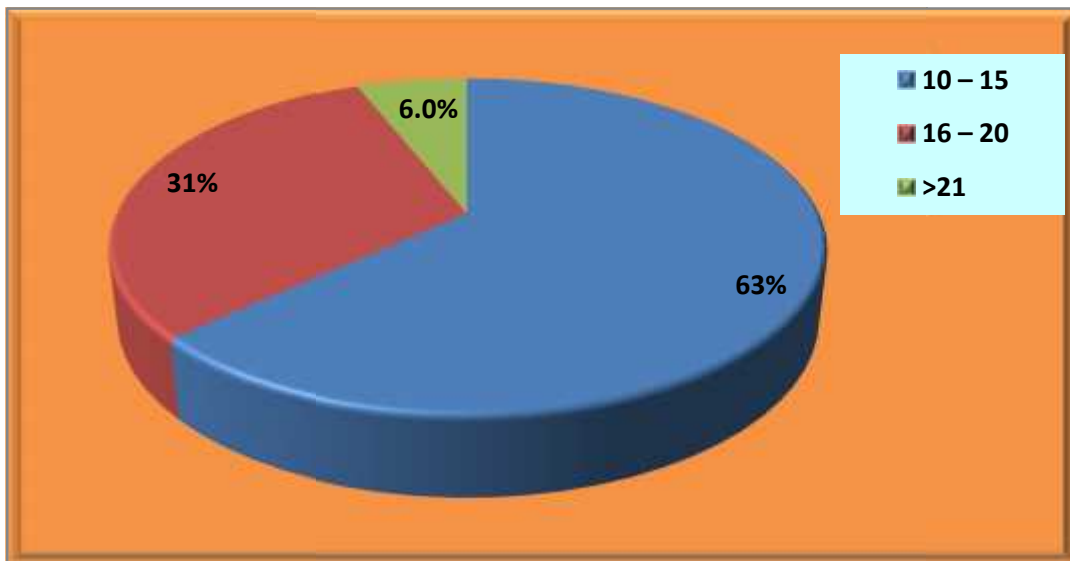
Graph No. 6: Frequency distribution of type of corneal lesions present.

TABLE NO. 18: We measured the IOP in all the patients bilaterally with an indentation tonometer and recorded the findings. None of the 350 patients had hypotonia. Most of these patients had IOP between 10 to 15 mm Hg (63%), while 21 (6%) of them had more 21 mm Hg. These findings are statistically significant ($p < 0.001$), which means presence of intra ocular hypertension in this population is having a positive association.

Table No. 18. Distribution of Patients according to IOP recorded

| IOP mm Hg | No of patients | Percentage |
|--------------|----------------|------------|
| 10 – 15 | 221 | 63 |
| 16 – 20 | 108 | 31 |
| >21 | 21 | 6.0 |
| Total | 350 | 100 |

p-value = 0.0001

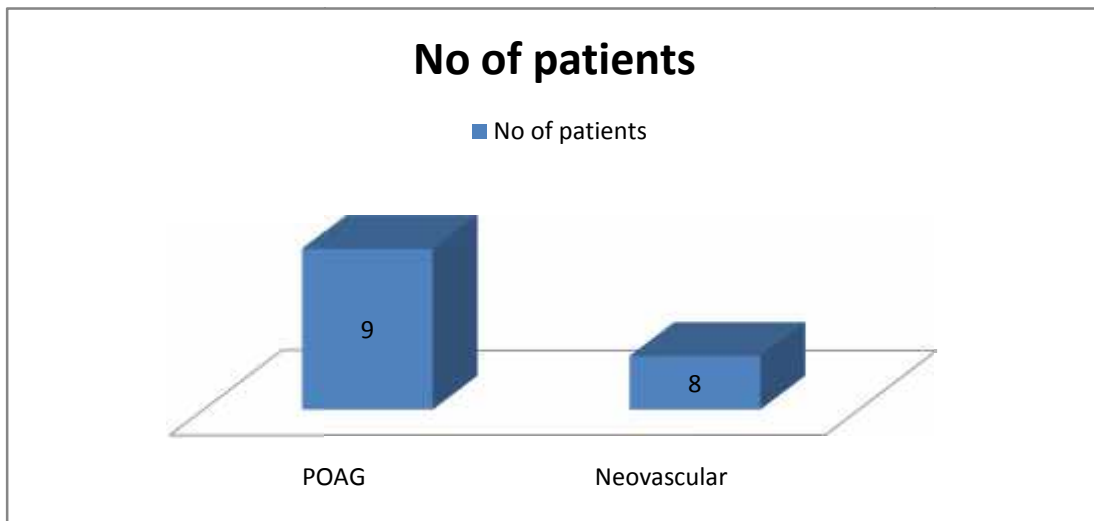


Graph No 7: Percentage Distribution of Patients according to IOP recorded.

TABLE NO. 19: 9 patients (2.7%) were diagnosed with Primary open angle glaucoma . 8 patients (2.3%) had neovascular glaucoma

Table No. 19. Distribution of patients according to type of glaucoma present.

| Type of glaucoma | No of patients | Percentage |
|--------------------|----------------|------------|
| POAG | 9 | 2.6% |
| Neovascular | 08 | 2.3% |
| Total | 17 | 4.9% |

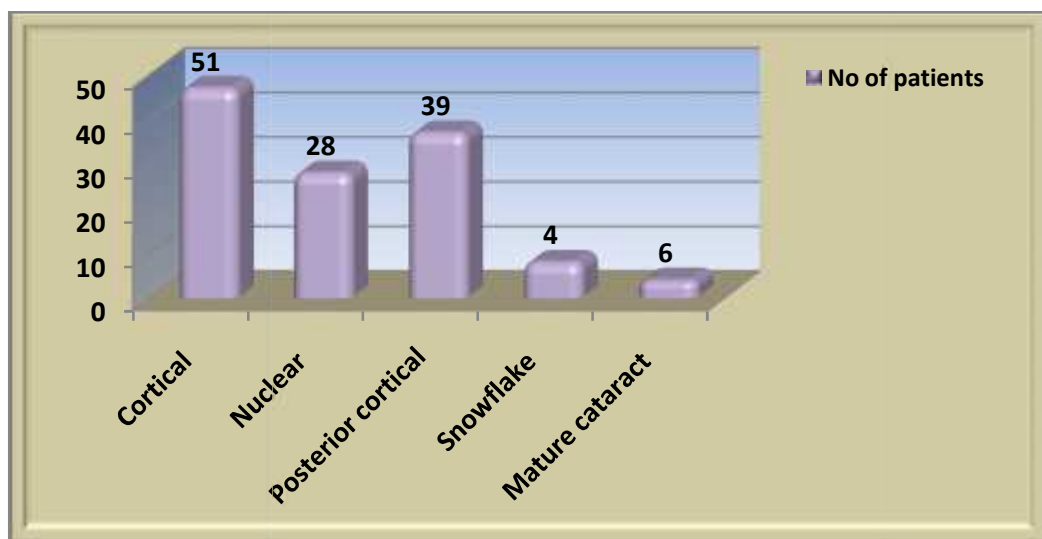


Graph No. 8. Distribution of patients according to type of glaucoma.

TABLE 20: shows the number of patients affected by lenticular opacities. 124 of the total patients had presented with cataract (35.4%) which makes it the second most common mode of ocular diabetic disease in our study. Most common variant found was cortical : 51 of them(41.2%) in the study which is more prevalent than the 39 posterior cortical (31.4%), considered to be a typical diabetic cataract. 28 patients also had nuclear cataract(29.8%). 6 patients had mature cataract (4.8%), while 4 patients (3.2%) had the characteristic snowflake appearing cataract .

Table No. 20. Distribution of patients according to type of cataract present

| Type of cataract | No of patients | Percentage |
|--------------------|----------------|------------|
| Cortical | 51 | 41.2 |
| Nuclear | 28 | 22.6 |
| Posterior cortical | 39 | 31.4 |
| Snowflake | 4 | 3.2 |
| Mature cataract | 6 | 4.8 |
| Total | 124 | 100 |



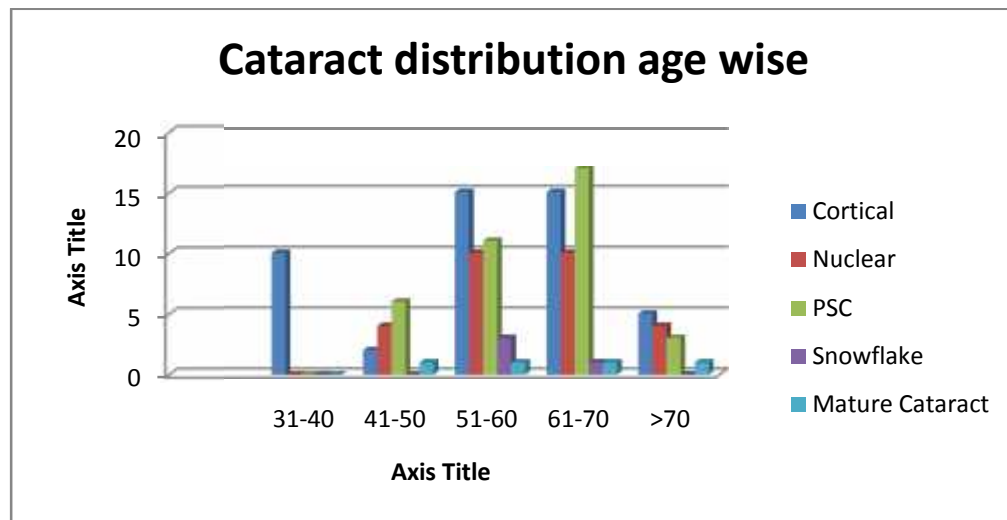
Graph no 9: Distribution of patients according to type of cataract

TABLE NO. 21: It clearly shows that most of the people develop any one of these cataracts in the age group of 51-70 years (70.9%), which coincides with that of non-diabetic population. The finding has been confirmed by statistical significant p- value of 0.008.

Table No. 21: Distribution of cataract according to age

| Age (Years) | Cortical | Nuclear | PSC | Snowflake | Mature Cataract | Total cataract/Study group |
|--------------|----------|---------|-----|-----------|-----------------|----------------------------|
| 31-40 | 10 | 0 | 0 | 0 | 0 | 10 |
| 41-50 | 2 | 4 | 6 | 0 | 1 | 13 |
| 51-60 | 15 | 10 | 11 | 3 | 1 | 40 |
| 61-70 | 19 | 10 | 17 | 1 | 1 | 44 |
| >70 | 5 | 4 | 3 | 0 | 1 | 13 |
| Total | 51 | 28 | 37 | 04 | 04 | 124 |

p-value = 0.008

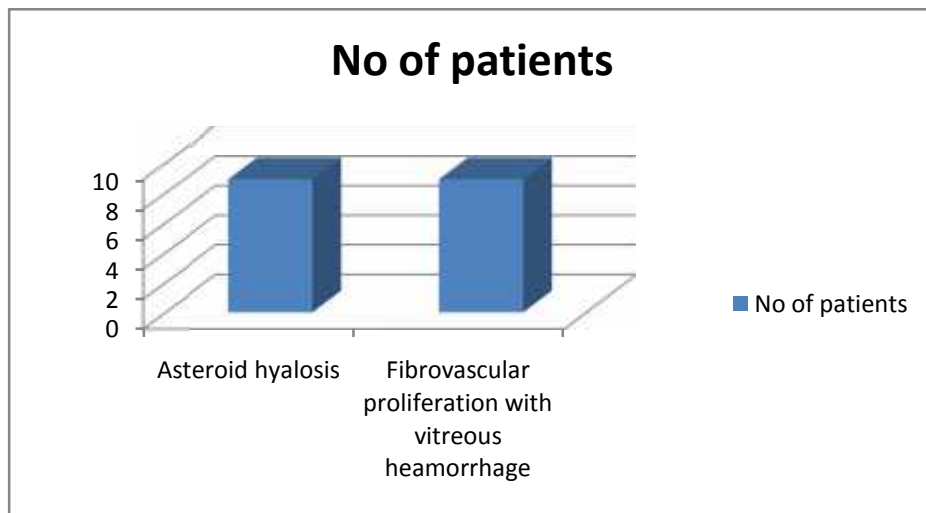


Graph No. 10. Distribution of Cataracts according to age groups

TABLE NO. 22: 9 patients had vitreous hemorrhage at presentation (2.6%), while there were another 9 patients having asteroid hyalosis.

Table No. 22. Distribution of patients according to findings in vitreous

| Vitreous finding | No of patients | % |
|---|----------------|-------------|
| Asteroid hyalosis | 9 | 2.6% |
| Fibrovascular proliferation with vitreous heamorrhage | 9 | 2.6% |
| Total | 18 | 5.2% |

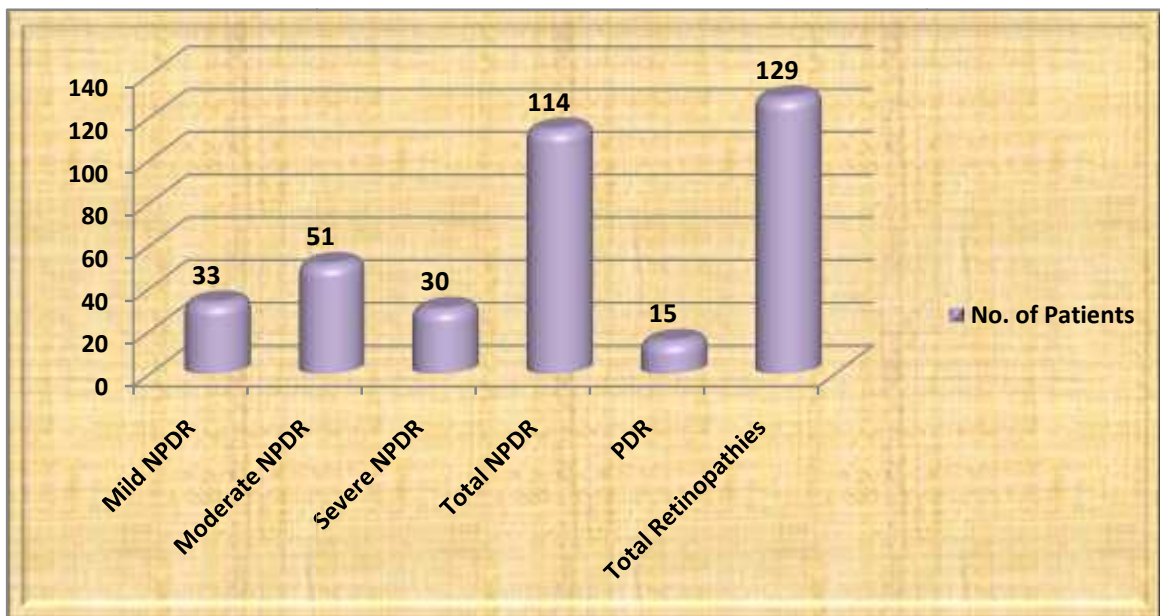


Graph No. 11. Distribution of patients according to findings in vitreous

TABLE NO. 23: We did the fundoscopy of all patients to find out the retinal changes in them. 129 patients were affected by some form of retinopathies (36.8%) making it the most common pathological condition found in the study population. 114 of them (32.6%) had NPDR while 15 had PDR (4.3%). In the NPDR group, 33 of them had mild NPDR (25.6%), 51 had moderate (39.5%) and 30 had severe NPDR (23.5%). 18 of these patients had CSME (5.6%).

Table No. 23. Distribution of patients according to type of retinopathy.

| Type of Retinopathy | No. of Patients | Percentage |
|----------------------------|-----------------|-------------|
| Mild NPDR | 33 | 25.6 |
| Moderate NPDR | 51 | 39.5 |
| Severe NPDR | 30 | 23.2 |
| Total NPDR | 114 | 88.3 |
| PDR | 15 | 11.6 |
| Total Retinopathies | 129 | 100 |



Graph no 12: Distribution of patients according to type of retinopathy

TABLE No. 24: In this study, most of the patients were found to be in the age group of 51-60 years (33%). The average age of the patients studied was 50.9yrs. A significant association was found between age group and retinal complication of diabetes mellitus.(p value=0.001)

Table No. 24: Distribution of patients according to age group.

| Diagnosis | | Age | | | | | Total |
|---------------|------------|-------|-------|-------|-------|------|-------|
| | | 31-40 | 41-50 | 51-60 | 61-70 | >71 | |
| Mild NPDR | Frequency | 01 | 03 | 14 | 10 | 05 | 33 |
| | Percentage | 6.3 | 10.7 | 24.6 | 23.3 | 22.7 | 19.9 |
| Moderate NPDR | Frequency | 00 | 10 | 20 | 12 | 09 | 51 |
| | Percentage | 00 | 35.7 | 35.1 | 27.9 | 40.9 | 30.7 |
| Severe NPDR | Frequency | 12 | 02 | 06 | 08 | 02 | 30 |
| | Percentage | 75.0 | 7.1 | 10.5 | 18.6 | 9.1 | 18.1 |
| PDR | Frequency | 01 | 03 | 04 | 04 | 03 | 15 |
| | Percentage | 6.3 | 10.7 | 7.02 | 9.3 | 13.6 | 9.03 |
| CSME | Frequency | 0 | 6 | 7 | 4 | 1 | 18 |
| | Percentage | 00 | 21.4 | 12.3 | 9.3 | 4.5 | 10.8 |
| OTHERS | Frequency | 2 | 4 | 6 | 5 | 2 | 19 |
| | Percentage | 12.5 | 14.3 | 10.5 | 11.6 | 9.1 | 11.4 |
| TOTAL | | 16 | 28 | 57 | 43 | 22 | 166 |

p-value = 0.0001

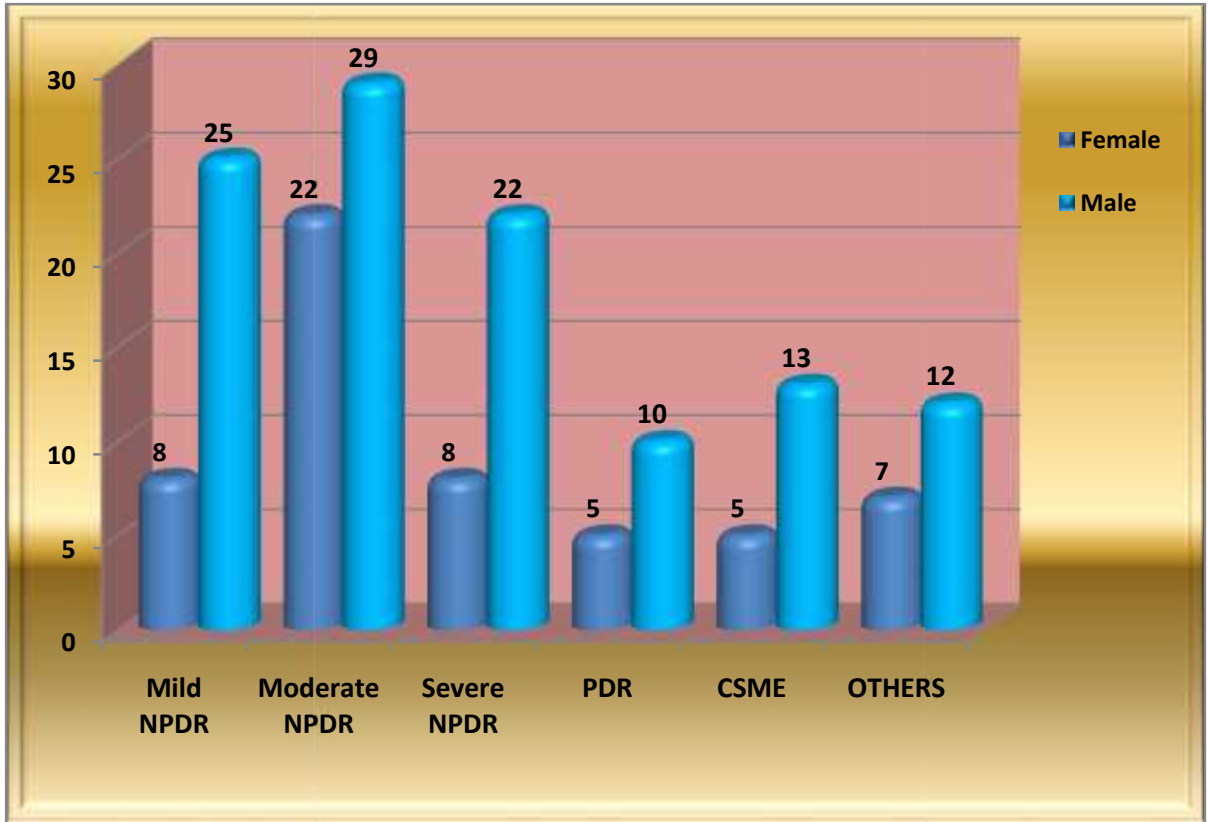
TABLE NO. 25: Shows the prevalence of the same retinal lesions in accordance with the sexual distribution. In the present study 211 patients were male while 139 patients were female. Of these, 111 men and 55 women were affected by some kind of retinal disease. We found significant association between sex and ocular complication of diabetes mellitus (p -value < 0.001) wherein both mild NPDR and severe NPDR were more common in males than in females. Our study showed no difference in prevalence of PDR in either sexes (9% each), while slightly more common CSME in men (11.7%) than in females (9.1%).

The prevalence of combined retinal lesions were however more common in males (211, 52.6%) than females (120, 39.6%)

Table No. 25: Distribution of patients according to sex

| Diagnosis | | Sex | | |
|---------------|------------|--------|------|-------|
| | | Female | Male | Total |
| Mild NPDR | Frequency | 08 | 25 | 33 |
| | Percentage | 14.5 | 22.5 | 19.9 |
| Moderate NPDR | Frequency | 22 | 29 | 51 |
| | Percentage | 40.0 | 26.1 | 30.7 |
| Severe NPDR | Frequency | 08 | 22 | 30 |
| | Percentage | 14.5 | 19.8 | 18.1 |
| PDR | Frequency | 05 | 10 | 15 |
| | Percentage | 9.1 | 9.0 | 9.03 |
| CSME | Frequency | 05 | 13 | 18 |
| | Percentage | 9.1 | 11.7 | 10.8 |
| OTHERS | Frequency | 07 | 12 | 19 |
| | Percentage | 12.7 | 10.8 | 11.4 |
| TOTAL | | 55 | 111 | 166 |

p-value= 0.001



Graph No. 13: Distribution of retinopathy patients according to sex

TABLE NO. 26: Shows the prevalence of retinopathies with the duration of diabetes. 11.7% of people are affected by Mild NPDR within 5 years of getting type 2 Diabetes, which increases significantly to 23.7% and 25% by 10 years and thereafter. Similarly, Moderate NPDR rises from 25% to 28.9% and 36.8% in same interval. The severe NPDR type prevalence rises from 8.3% to 18.8% within 5 years to more than 10 years of diabetes. Also, PDR prevalence increased from 1.7% to 13.2%.

Table No. 26. Co relation between duration of diabetes and type of retinopathy

| Diagnosis | | Duration of DM | | | |
|---------------|------------|----------------|------|------|-------|
| | | 0-5 | 6-10 | >10 | Total |
| Mild NPDR | Frequency | 07 | 09 | 17 | 33 |
| | Percentage | 11.7 | 23.7 | 25.0 | 19.9 |
| Moderate NPDR | Frequency | 15 | 11 | 25 | 51 |
| | Percentage | 25.0 | 28.9 | 36.8 | 30.7 |
| Severe NPDR | Frequency | 17 | 07 | 06 | 30 |
| | Percentage | 8.3 | 12.4 | 18.8 | 18.1 |
| PDR | Frequency | 01 | 05 | 09 | 15 |
| | Percentage | 1.7 | 13.2 | 13.2 | 9.03 |
| CSME | Frequency | 07 | 02 | 09 | 18 |
| | Percentage | 11.7 | 5.3 | 13.2 | 10.8 |
| OTHERS | Frequency | 13 | 04 | 02 | 19 |
| | Percentage | 21.7 | 10.5 | 2.9 | 11.4 |
| TOTAL | | 60 | 38 | 68 | 166 |

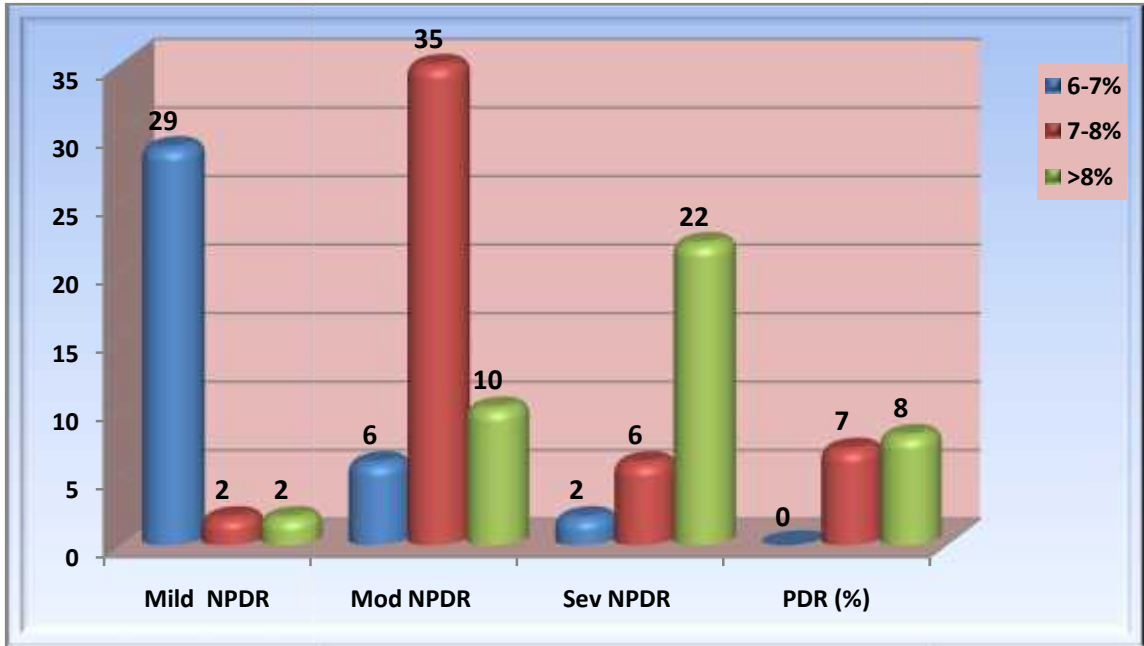
p-value = 0.002

TABLE NO 27: HBA1C has been known as a marker of glycemic control. We used this serum marker to identify the relation between the levels of uncontrolled hyperglycemia with prevalence of different retinopathies. The study yielded a highly significant result. (p- value <0.0001). Our study found that with increasing HBA1C levels, the prevalence of retinopathies increases. From Table No.27, it is clear that 16.1%, 65.8% and 95.5% prevalence was observed for HBA1C of 6-7%, 7-8% and > 8 % respectively. It is also seen that the mild NPDR (87.9%) is found clustering at lower levels of HBA1c (<8 %), moderate NPDR(68.6%) is most prevalent between 7-8 % of HBA1C levels and Severe NPDR(73.3%) is most common at > 8 % levels.

Table No. 27. Co- relation between HBA1C levels and prevalence of retinopathy

| HBA1C | No. of Patients | Mild NPDR | Mod NPDR (%) | Sev NPDR (%) | PDR (%) | Total |
|---------------|------------------------|------------------|---------------------|---------------------|----------------|--------------|
| 6-7% | 230 | 29(87.9) | 6(11.7) | 02(6.7) | 00(0) | 37(16.1%) |
| 7-8% | 76 | 02(6.1) | 35(68.6) | 06(20.0) | 07(46.7) | 50(65.8%) |
| >8% | 44 | 02(6.1) | 10(19.6) | 22(73.3) | 08(53.3) | 42(95.5%) |
| Total | 350(100) | 33(100) | 51(100.) | 30(100) | 15(100) | 129(100) |

p-value <0.0001



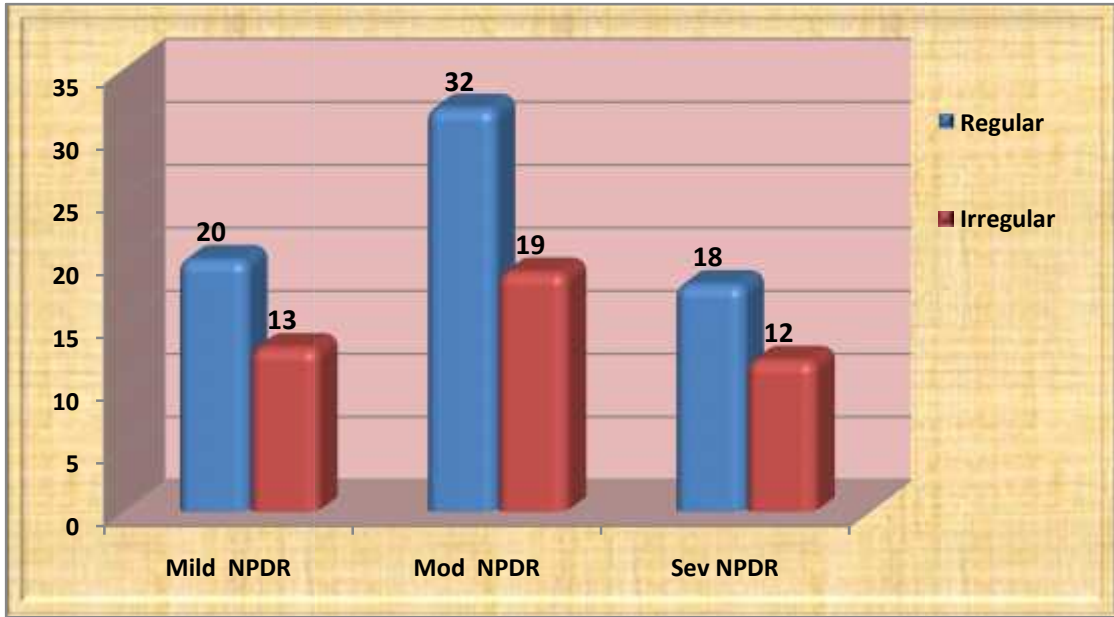
Graph No. 14. Co- relation between HBA1C levels and prevalence of retinopathy

TABLE NO. 28: We also did a comparative study of prevalence of NPDR in patients accordance to their diabetes treatment compliance. Most of these patients had been advised either oral hypoglycemic in the form of metformin, glipizide, gluconorm, etc. or subcutaneous insulin. 250 of our patients (i.e. 65.1%) were taking regular treatment, while 90 (34.9%) of them were not regular with their medications. Apparently, 60.6% of Mild NPDR patients were on regular treatment than 39.3% who were not. In the same way, both moderate and severe variety of NPDR were found more commonly with regular treatment than irregular ones. The p- value of this is 0.964, which indicates dissociation between the two.

Table No. 28. Severity of NPDR versus regular and irregular treatment

| Treatment | | Mild NPDR | Mod NPDR | Severe NPDR | Total |
|-------------------------------|------------------|------------------|-----------------|--------------------|--------------|
| Regular (250/350) | Frequency | 20 | 32 | 18 | 60/249 |
| | % | 60.6 | 62.7 | 60.0 | 73.6 |
| Irregular (90/350) | Frequency | 13 | 19 | 12 | 44/90 |
| | % | 39.3 | 37.3 | 40.0 | 39.4 |
| Total (350) | Frequency | 33 | 51 | 30 | 114 |
| | % | 100 | 100 | 100 | 100 |

Chi square=0.07 p-value=0.964



Graph No. 15. : Severity of NPDR versus regular and irregular treatment

TABLE NO. 29 : Other retinal lesions like ARMD was present in 4 of them, CRVO, BRAO, CRAO & Retinal detachment in 2 each, while 1 patient had BRVO. These 13 patients altogether does not make a significant statistics.

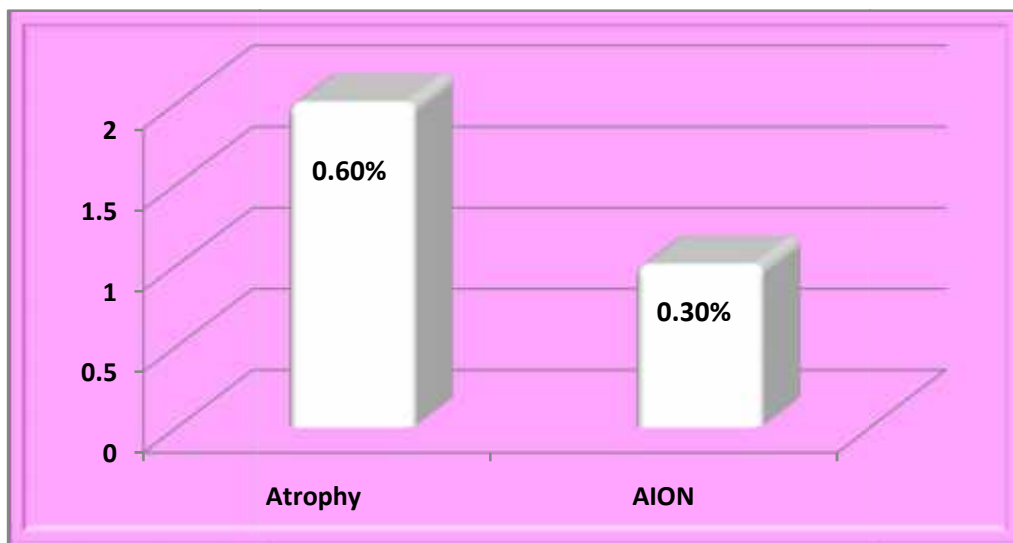
Table No. 29. Distribution of patients according to other retinal lesions

| Other retinal lesions | No of patients | % |
|------------------------------|-----------------------|----------|
| ARMD | 04 | 1.1% |
| CRVO | 02 | 0.6% |
| BRVO | 01 | 0.3% |
| BRAO | 02 | 0.6% |
| CRAO | 02 | 0.6% |
| Retinal detachment | 02 | 0.6% |
| Total | 13 | 3.7% |

TABLE NO.30: 2 of the patients also had optic nerve atrophy while 1 had an acute inflammation

Table No. 30. Number of Optic nerve lesions

| Optic nerve lesions | No of patients | % |
|----------------------------|-----------------------|----------|
| Atrophy | 2 | 0.6% |
| AION | 1 | 0.3% |

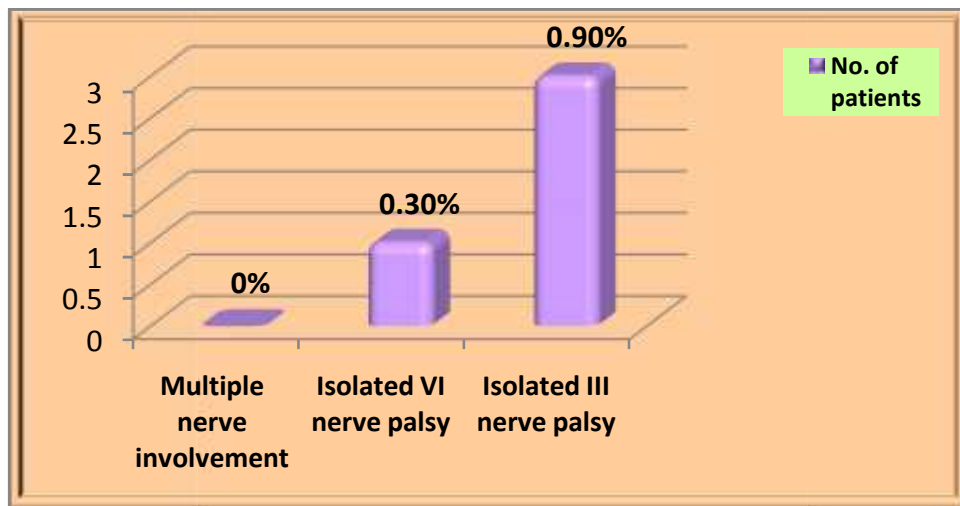


Graph no. 16. Number of Optic nerve lesions.

TABLE NO. 31: There were 3 patients with isolated 3rd cranial nerve palsy with no CNS involvement, while another patient had 6th nerve palsy with no other neurological deficits.

Table No.31. Distribution of patients according to cranial nerve involvement

| Cranial nerves involved | No. of patients | % |
|---|-----------------|------|
| Multiple nerve involvement (III,IV,V,VI,VII) | 0 | 0% |
| Isolated VI nerve palsy | 1 | 0.3% |
| Isolated III nerve palsy | 3 | 0.9% |



Graph No 17. Distribution of patients according to cranial nerve involvement.

DISCUSSION

In this study most of the patients were found to be in the age group of 51-60 years (33.7%). All the patients were aged above 30 years. There were 211 males and 139 females in the study group. The average age of the patients studied was 54.9 years for males and 56.2 years for females. Comparable age distribution was found in the Wisconsin epidemiologic study of diabetic retinopathy.²² The average duration of diabetes in the study group was 6.4 years in males and 7.3 years in females.

In the present study we found retinal lesions were the most common ocular complication occurring in diabetes subjects (40.6%), of which retinopathies of all kind constituted majority of them(36.8%). The prevalence of cataract was 35.4% followed by glaucoma (4.6%) and other ocular pathologies like conjunctivitis, recurrent styes, dacrocystitis, etc. Stanga PE,⁸³ in their review of literature in 1999, have found that retinopathy is the most common ocular complication of long standing diabetes mellitus followed by other lesions like cataract, uveitis, neuro-ophthalmitis, etc. It is generally recognised in existing literature that retinopathy is the most common ocular complication in type 2 diabetes. Our study confirms that diabetic retinopathy in its various forms is the most common complication of diabetic eye disease in India.

Different eye lid lesions were noted in our study such as recurrent stye (4%), Xanthelesma (4%), Blepharitis (3.7%), recurrent chalazion (0.6%), etc. Xanthelasma are rare in the general population. Variable incidence of 0.56%-1.5% has been reported from the western developed nations.⁸⁴ The incidence in our study is 4%, which is explained by the fact that xanthelesma is a cutaneous marker for underlying diabetes and dyslipidemia.

Diabetes predisposes to infection in different body parts, and ocular structures are not any different from it. Thus, recurrent styes (4%), blepharitis (3.7%), conjunctivitis (1.7%) and dacryocystitis (2.6%) can be explained in our group of patients. Kruse, have confirmed in their case control study that diabetes is a risk factor for acute infectious conjunctivitis, with an Odds Ratio (OR) of 1.24 in diabetics.⁸⁵ We did not get a study attributing causal relation or direct association between diabetes and the above said other lesions.

There were 19 patients with Pterygium in our study group, which makes it 5.42% prevalence. Two different studies, one by Asokan, (Chennai group, 2012)⁸⁶ and Hua Zhong⁸⁷ (study of adult Chinese population) have statistically found out no association between prevalence of pterygium and diabetes. We need to find out the disparity among our population with prevalence of pterygium.

Microbial corneal ulcer is a sight-threatening condition and one of the major causes of blindness. Diabetic patients with microbial keratitis are at an increased risk than non-diabetic persons and require immediate medical attention. In this context, recording the local microbiological status of microbial corneal keratitis would be more useful for clinicians / ophthalmologists as to provide effective treatment. The present study evaluated presence of corneal ulcers in the study population, but did not record the microbiology of diabetic subjects who were diagnosed with corneal ulcer of bacterial / fungal etiology. There were 7 patients with corneal ulcer of which 5 were bacterial type and 1 was nonhealing. There was 1 patient presenting with corneal perforation. This signifies the need for appropriate microbiological diagnosis of microbial corneal ulceration in order to provide prompt and effective medical remedy and to avoid empirical treatment.

Nielsen found that the prevalence rate of primary open angle glaucoma and ocular hypertension was 6.0% and 3.0%, respectively in diabetes type 2 patients. Neovascular glaucoma occurred in 2.3% of all diabetics.⁸⁸ In our study, we had 21 patients, i.e. 6% with ocular hypertension (IOP > 21mmHg), 8 patients (2.3%) were of Neovascular type and rest being POAG (2.6%). Thus, the prevalence is similar in both studies.

Cataract:

Persons with diabetes mellitus have been found to be at increased risk of developing cataracts when compared with non diabetic persons. The report by Klein describes the study of characteristics which may be related to this problem in a population-based sample of diabetic persons. Prevalence of surgical aphakia and cataract increased with increasing age in both younger and older onset diabetic persons.⁸⁹ Nearly two thirds of the Indian diabetic population showed evidence of cataract; mixed cataracts were more common than the monotypes ones in a recent study by Shankar Nethralaya.⁹⁰ 35.4% of our study population had cataract which is lesser than the Shankar Nethralaya group. 20 patients in our group had mixed type of cataracts (14.7%) which is lesser than the total number of monotypes. The most common type of cataract found was cortical type (41.2%) followed by senile posterior cortical (29.8%). The characteristic snow flake cataract of diabetes is found in only 3.2% of cataract patients, which is of similar lower incidence in many studies.

Data from the Framingham and other eye studies indicate a three to fourfold increased prevalence of cataract in patients with diabetes under the age of 65, and up to a twofold excess prevalence in patients above 65. The risk is increased in patients with longer duration of diabetes and in those with poor metabolic control. Cataracts may be reversible in young diabetics with improvement in metabolic control. The most frequently seen type of cataract in diabetics is the age-related or senile variety, which tends to occur earlier and progresses more rapidly than in

nondiabetics. Presence of mostly cortical variant in our study could reflect environmental or genetic factors. Most of the people develop any one of these cataracts in the age group of 51-70 years (42.7%), which coincides with that of 3 times that of non-diabetic population.⁹¹

Recent studies on cataract surgery in diabetics tend to report a lower incidence of complications and better visual outcomes. This trend of improvement may be due to better preoperative management of retinopathy, evolutions in operative techniques and appreciation of the importance of systemic factors such as glycemic and hypertensive control.

In general, the visual prognosis following cataract surgery in diabetic patients is favorable. Diabetic patients with little or no retinopathy enjoy the same good prognosis as individuals without diabetes. However, in the presence of significant diabetic retinopathy, postoperative visual acuity may be suboptimal and the results of surgery may be disappointing. Presence of CSME and poor preoperative visual acuity (reflecting diabetic maculopathy, ischemia and traction) have been recognized as risk factors for poor postoperative visual acuity following cataract surgery.⁹²

We operated on all patients with visual acuity of < 6/36, 2 patients had posterior capsular opacity, 1 had conjunctivitis, and rest did not have any major problems. We infer that cataract surgery in diabetes is safe and good results can be expected as in any other group.

Retinopathy:

The prevalence of retinopathy in our study population was 36.8%, of which NPDR were 32.6 % and PDR were 4.3%. Studies have reported that the prevalence of diabetic retinopathy in India varies from 20-31 %. The Aravind Eye Disease Survey in southern India reported a retinopathy prevalence of 27% in a population aged 30 years or older with self-reported diabetes,⁹³ similar to the 22% prevalence reported from another population-based study in an urban population in Hyderabad, India.⁹⁴ The prevalence of DR in the Chennai Urban Rural Epidemiology (CURES) Eye Study in south India was

17.6 per cent, significantly lower than age-matched western counterparts. Thus, our study shows a higher prevalence of retinopathies than other Indian studies.

A recent pooled study examined the prevalence of retinopathy among people 40 years and older from eight population-based studies, including the WESDR.⁹⁵ The studies included in these meta-analysis used standardized methods to grade retinopathy from fundus photographs and estimated an overall prevalence of retinopathy of about 40% and a prevalence of sight-threatening disease.

The prevalence of NPDR was significantly higher in female diabetes patients (34.3%) than in male diabetes patients (31.8%). Wisconsin Epidemiological study of Diabetic Retinopathy had also found higher prevalence of Diabetic Retinopathy in females. There was no significant difference in prevalence of PDR, CSME or other lesions. Thus, females are at higher risk of NPDR than males.

In the younger onset group in the WESDR, the prevalence of any retinopathy was 8% among participants with diabetes duration of 3 years, 25% for 5 years, 60% for 10 years, and 80% for 15 years.²² In the present study, the prevalence of proliferative retinopathy was 1.7% for those with diabetes duration of 5 years, increasing to 13.2% for 10 years. In our study, the prevalence of NPDR varied from 26.1% in persons who had diabetes for less than five years to 32.3% in persons who had diabetes for 5 to 10 or more years and 78.7% in more than 10 years.

Increased incidence of CSME was noted as the duration of diabetes increased (11.7% to 13.2% over the same duration intervals of diabetes.) Similar increased incidence of CSME with increased duration of diabetes was noted in a study by Varma.⁹⁶ The findings are thus consistent with the fact that the strongest predictor for the prevalence of

retinopathy in persons with type 2 diabetes is the duration of diabetes and was proven statistically significant (p-value < 0.002)

Two landmark multicentered clinical trials, the DCCT and the UKPDS assessed the relationship between glycemic control and vascular complications of diabetes. One of the most important predictive factors for diabetic retinopathy is the level of glycemic control. The WESDR showed that both the younger-onset and older-onset patients with diabetes who had no retinopathy had significantly lower mean glycosylated hemoglobin values than those patients with retinopathy.⁹⁵ Patients with higher glycosylated hemoglobin values were shown to have a higher risk of retinopathy, such that those with mean HbA1c levels over 12% were 3.2 times more likely to have retinopathy after 4 years than subjects with HbA1c levels under 12%.⁹⁷ Our study population exhibits a similar pattern : 16.1% of diabetic patients with HBA1C between 6-7% had some form of DR, while the prevalence rises to 65.8% and 95.5% with HBA1C of 7-8% and more than 8% (i.e. uncontrolled type) respectively. It is also seen that the mild NPDR is found clustering at lower levels of HBA1c (<7 %), moderate NPDR is most prevalent between 7-8 % of HBA1C levels and Severe NPDR is most common at > 8 % levels. Thus, both prevalence and severity of retinopathy correlates with HBA1C level in our study group.

In our study, subjects taking regular treatment (oral tablets/insulin) had a combined NPDR prevalence of 24% which is lower when compared to the group not taking treatment regularly (48.9%). Regular treatment and follow up should be stressed in the management of diabetes mellitus. In a study conducted by Alan MJ⁹⁸ Compared with individuals with continuous follow-up, patients with irregular clinical visits were more likely to be from families of lower socioeconomic class levels, have a parental history of

separation and divorce, and were members of families that reported being least openly expressive of positive emotions. Poor glycemic control in year 1 was associated with irregular follow-up in years 2 through 4. Patients with irregular follow-up continued to have worse glycemic control in years 2 through 4 than patients with continuous follow-up. Retinopathy occurred more frequently among those in the irregular follow-up group.

Rush JA showed that diabetes is the underlying cause in 25–30% of patients aged 45 years and older who develop acute extra ocular muscle palsy.⁹⁹ In a study by Watanabe K, 1% of patients with diabetes were found to have cranial nerve palsies, compared with only 0.13% of control subjects.²¹ 1.1% of our patients (i.e. 4 of them) had cranial nerve palsy, same as with the Watanabe study.

We found a prevalence of 0.3% BRVO amongst diabetics in our study while BRVO were detected in 0.79% in a study conducted by Kawasaki R.¹⁰⁰

CONCLUSION

- Retinal lesions (like Retinopathies, CSME, BRVO, BRAO, ARMD and RD) were the most common ocular complication occurring in diabetes subjects (40.6%), of which retinopathies of all kind constituted majority of them (36.8%).
- The prevalence of cataract was 35.4% followed by glaucoma (4.6%) and other ocular pathologies like conjunctivitis, repeated styes, dacrocystitis, etc
- Xanthelesma are rare in general population (0.56%-1.5%), but are found more commonly in diabetic patients (4%).
- Diabetes predisposes to infection in different body parts, and ocular structures are not an exception. We could not find a attributable cause, but others have found diabetes as a risk factor for such infective pathologies.
- Pterygium was found to have a high prevalence in our study (5.42%), but others have not found an association of it with diabetes.
- Diabetic patients with microbial keratitis are at an increased risk than non-diabetic persons and require immediate medical attention.
- The prevalence of Primary open angle glaucoma was 2.6% and 2.3% had neovascular glaucoma.
- 35.4% of our study population had cataract which is lesser than the Shankar Nethralaya group. Monotypes were more common than the mixed type of cataracts.
- The most common type of cataract found was cortical type (41.2%) followed by senile posterior cortical (29.8%). In general, the visual prognosis following cataract surgery in diabetic patients is favorable.

- The prevalence of retinopathy in our study population was 36.8%, of which NPDR were 32.6% and PDR were 4.3%. Our study shows a higher prevalence of retinopathies than other Indian studies
- The prevalence of NPDR was significantly higher in female diabetes patients (34.3%) than in male diabetes patients (31.8%).
- The prevalence of NPDR varied from 26.1% in persons who had diabetes for less than five years to 32.3% in persons who had diabetes for 5 to 10 or more years and 78.7% in more than 10 years.
- The strongest predictor for the prevalence of retinopathy in persons with type 2 diabetes is the duration of diabetes and was proven statistically significant (p-value < 0.0001).
- 16.1% of diabetic patients with HBA1C between 6-7% had some form of DR, while the prevalence rises to 65.8% and 95.5% with HBA1C of 7-8% and more than 8% (i.e. uncontrolled type) respectively. It is also seen that the mild NPDR is found clustering at lower levels of HBA1c (<7 %), moderate NPDR is most prevalent between 7-8 % of HBA1C levels and Severe NPDR is most prevalent at > 8 % levels. Thus, both prevalence and severity of retinopathy correlates with HBA1C level in our study group.
- 1.1% of our patients (i.e. 4 of them) had cranial nerve palsy, same as with the Watanabe study and higher than in general population (0.13%).

SUMMARY

Diabetes is a group of metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. The nationwide prevalence of diabetes in India now tops 9%. By 2030, India will have 100 million people with diabetes. Diabetes mellitus, is a multisystem disease, and diabetic eye disease is an end-organ response to the effects of the condition on the human system. Each portion of the eye is susceptible to the harmful effects of diabetes. This clinical study of ocular manifestations of patients with type 2 diabetes mellitus was undertaken in view of large spectrum of diabetic eye disease apart from diabetic retinopathy.

This was a prospective observational study on 350 type 2 diabetes patients. Collected data of each patient including detailed diabetes history, ocular complaints and did complete ophthalmological examination. The data gathered was tabulated into a master chart and statistical analysis done with SPSS for relevant tables.

Significant Results included:

- All ocular structures can be affected by some diabetic complication.
- The most common prevalent ocular complication in type 2 diabetes is of retina: 142 patients (40.6%) followed by the lens 124(35.4%).
- Diabetics have been found to be at increased risk of developing cataracts when compared with nondiabetic persons. 35.4% of our study population had cataract which is lesser than the other South Indian studies.
- The most common type of cataract found was cortical type (41.2%) followed by senile posterior cortical (29.8%).

- The prevalence of retinopathy in our study population was 36.8%, of which NPDR were 32.5% and PDR were 4.3%. Our study shows a higher prevalence of retinopathies than other Indian studies (20-31%).
- The strongest predictor for the prevalence of retinopathy in persons with type 2 diabetes is the duration of diabetes.
- Patients with higher glycosylated hemoglobin values (HbA1C >8 %) were shown to have a higher risk of retinopathy.

Appropriate treatment of diabetes and optimal metabolic control are key goals in the prevention of many ocular complications of diabetes. At the same time, following the various guidelines to screen and detect these complications will make a difference in the final outcome of the patients.

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ANNEXURE I- ETHICAL CLEARANCE



B.L.D.E. UNIVERSITY'S
SHRI B.M. PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2022 at 3.30 pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title clinical study of ocular manifestation
in patients with type 2 diabetes mellitus

Name of P.G. student Dr. Shilpa M. Lemaran
Ophthalmology

Name of Guide/Co-investigator Dr. Vallabha. K
Prof & HOD. Ophthalmology

DR. TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI B.M. PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization

- 1) Copy of Synopsis/Research project.
- 2) Copy of informed consent form
- 3) Any other relevant documents.

ANNEXURE 2-

INFORMED CONSENT FORM

Title of the study: “A Clinical Study of the Ocular Manifestations in patients with type 2

Diabetes Mellitus.”

Name of the participant: _____

Name of the principal investigator:

Name of the institution:

Hospital and Research Centre

I _____ aged _____ yrs attending ophthalmology OPD in SHRI BLDEA Medical College, Hospital and Research Center, BIJAPUR, have been explained in my own language the need for clinical examination and evaluation of my eyes and investigations (fundus fluorescein angiography) that I may have to undergo as a participant in the study.

Consent form for Fundus Fluorescein Angiography:

Fluorescein angiography is a diagnostic test which uses special camera to photograph the structures of the back of the eye. This test is used to detect any leakage or damage to the blood vessels that nourish the retina.

Flourescein dye glows in visible light and is injected into a vein in the arm of the patient ,the dye reaches the retinal vessels through the vascular system. This test does not involve the use of X-ray or any other harmful form of radiation.

Fundus fluorescein angiography is considered to be a very safe test and side effects are uncommon. However the possibility of side effects cannot be completely ruled out. Fluorescein does not contain iodine and hence can be used in patients allergic to iodine. Some patients can experience slight nausea and vomiting. some patients who are allergic to the dye can develop rashes, which require medications such as antihistaminics and steroids. Very rarely a life threatening allergic reaction known as anaphylaxis can occur which requires prompt medical treatment. There may be infiltration of the dye into the skin at the site of injection, which could cause discolouration of the skin at that site. Flourescein dye will turn a patients urine orange and may slightly discolour the skin as well for a brief period of time.

I understand that fluorescein angiography is only a test and not a treatment modality . I understand the risks involved with this test and hereby give consent to undergo this test.

I hereby also give my consent to be included as a participant in the study

“A Clinical Study of the Ocular Manifestation in patients with Diabetes Mellitus type 2.”

Name of the patient _____

Date _____

Signature of the patient _____

Time _____

Name of the impartial Witness _____

Signature of the impartial witness _____

Name of Investigator _____

Signature of the Investigator _____

ANNEXURE IX

PROFORMA

NAME :

AGE/SEX :

I.P. NO:

OCCUPATION :

ADDRESS:

SOCIO ECONOMIC STATUS:

CHIEF COMPLAINTS

OCULAR SYMPTOMATOLOGY

DURATION

INSULIN TREATMENT

VISUAL ACQUITY

RE:

LE:

PH:

PH:

INSULIN: REGULAR / IRREGULAR

PAST HISTORY: HTN / TB / DRUG ALLERGY / NEPHROPATHY

HISTORY OF SIMILAR COMPLAINTS

HISTORY OF OCULAR DISEASES

PREVIOUS OCULAR CHECK UP: YES/NO

FAMILY HISTORY:

OCULAR EXAMINATION:

LOCAL OCULAR EXAMINATION:

Head posture:

Facial Symmetry:

Ocular position:

Ocular movements:

Vision:

Lids:

*Stye

*Xanthelasma

*Blepharitis

*Others:

*None:

Conjunctiva:

*Microaneurysm

Cornea:

*Sensations

*Erosions

*Others

Anterior Chamber (A/C)

*Depth:

*Angle:

*Neovascularisation

*Glaucoma; Present / Absent:

Type, if present:

Iris:

*Pattern:

*Ectropion Uveae:

*Neovascularisation:

*Iridocyclitis:

Pupil:

*Reaction: Direct: Indirect:

*Response to mydriatics:

Lens:

*Cataract:

Type:

IOP:

Sac Syringing:

Vitreous (SLE):

*Asteroid Hyalosis:

*Haemorrhages:

*Fibrovascular Proliferations:

Fundus:

*Retinopathy:

*Maculopathy:

INVESTIGATIONS

RBS

FBS

URINE ALBUMIN

IOP

FUNDUS FLUORESCEIN ANGIOGRAPHY(whenever indicated):

TREATMENT ADVISED

NONE

MEDICAL MANAGEMENT

FOCAL LASER

PRP

VITRECTOMY

CATARACT SURGERY

REFERRED

ANNEXURE 4 –

KEYS TO MASTER CHART

| | | |
|----------|---|--------------------------------|
| Ac | - | Acute |
| Chr | - | Chronic |
| CC | - | Cortical Cataract |
| Dacr | - | Dacrocystitis |
| M | - | male |
| F | - | female |
| FBS | - | Fasting Blood Sugar |
| RE | - | right eye |
| RFT | - | Renal Function Test |
| LE | - | left eye |
| POAG | - | Primary Open Angle Glaucoma |
| Pseudo P | - | Pseudo Phakia |
| PSC | - | posterior subcapsular cataract |
| PRP | - | Pan Retinal Photocoagulation |
| NAD | - | Nothing Abnormal Detected |

| | | |
|-------|---|---|
| NS | - | Nuclear sclerosis |
| CF | - | Counting fingers |
| HM | - | Hand movements |
| PL PR | - | Perception of light ,projection of rays |
| PPBS | - | Post Prandial Blood Sugar |
| m | - | Meter |
| VL | - | Vitreous leak |
| ME | - | Macular Edema |
| PCO | - | Posterior capsular opacification |
| CSME | - | Cystoid macular edema |
| RD | - | Retinal detachment |
| IP | - | iris prolapsed |
| ARM | - | Age related maculopathy |
| ARM D | - | Age related macular degeneration |
| BRVO | - | Branch retinal vein occlusion |

ANNEXURE 5-

MASTER CHART

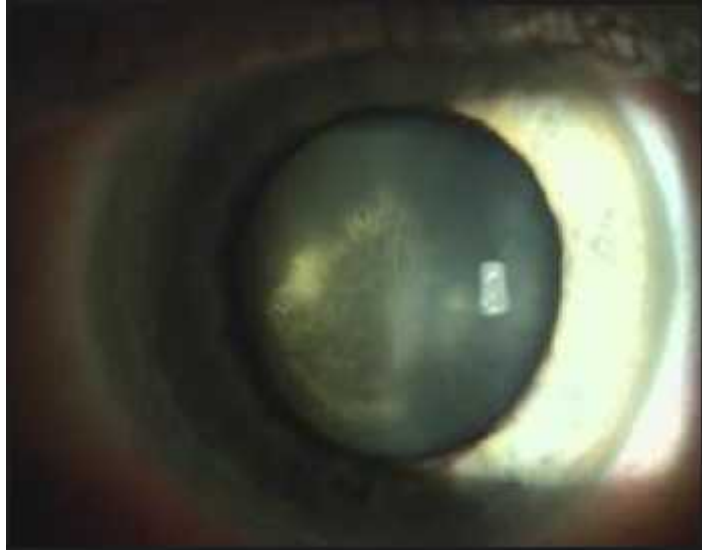


Fig No. 8 POSTERIOR SUB CAPSULAR CATARACT

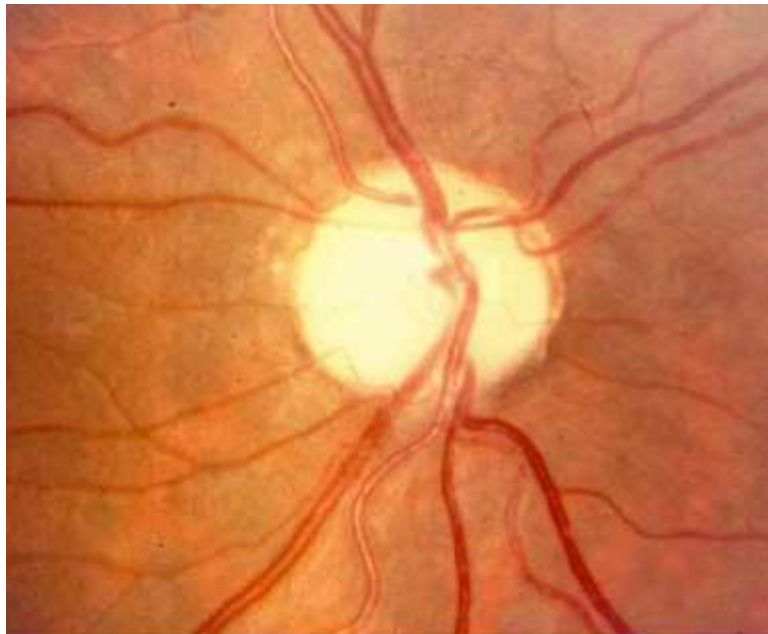


Fig No 9 PRIMARY OPTIC ATROPHY

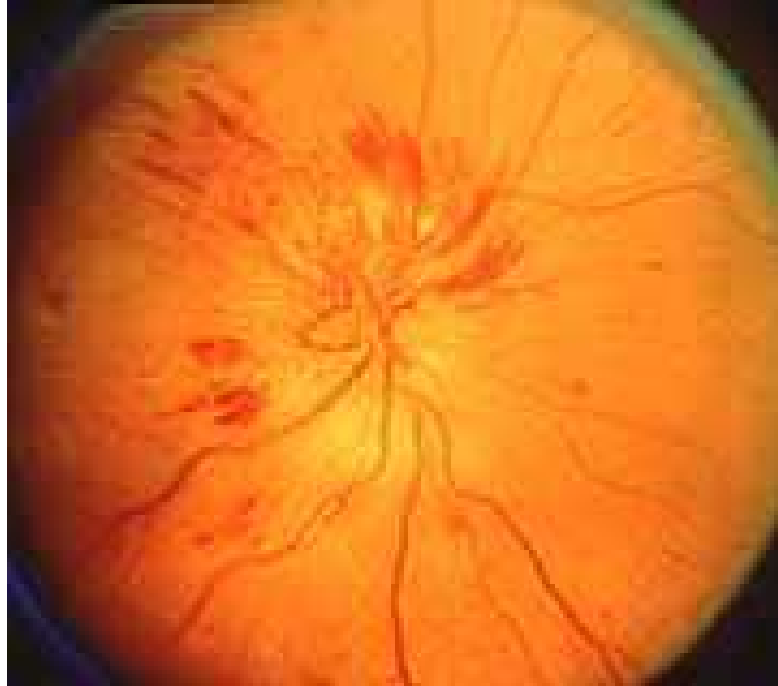


Fig No 10 NON ARTERITIC AION



Fig. NO. 11 CSME WITH VITREOUS HAEMORRHAGE

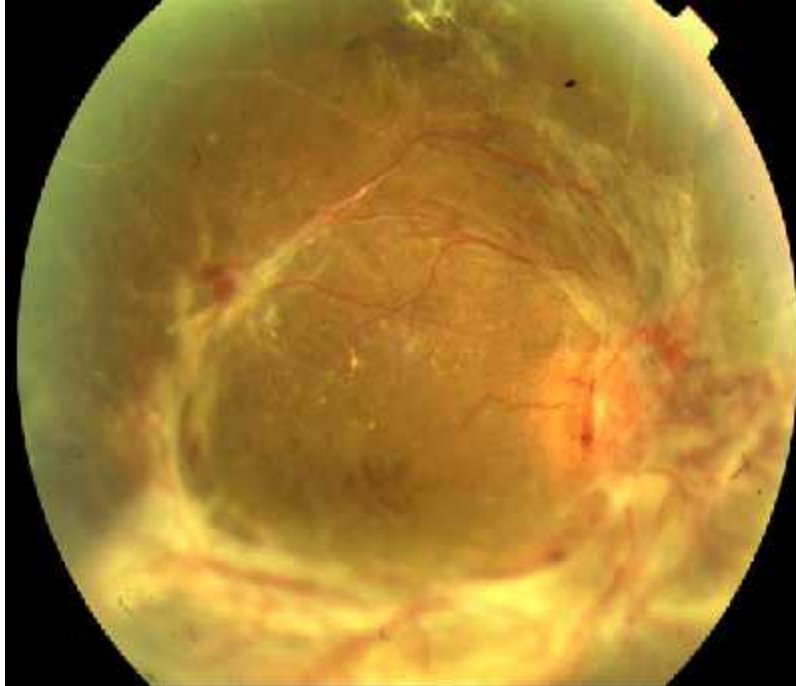


Fig. NO 12 PROLIFERATIVE DIABETIC RETINOPATHY

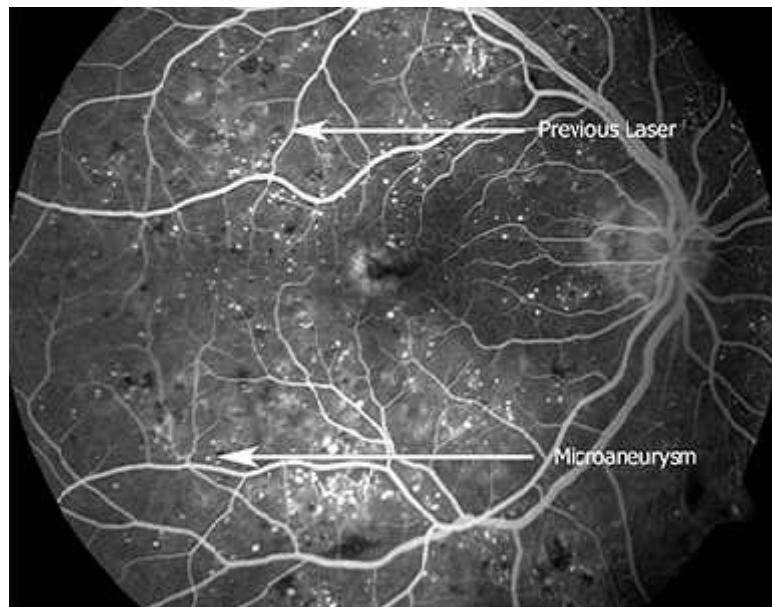


Fig. NO 13 FUNDUS FLUORESCIN ANGIOGRAPHY

| SL NO. | NAME | AGE/SEX | IP/OP NO. | Duration of DM | TREATMENT | HBA1C | FBS/PPBS | URINE ALBUMIN | Vision | | Pin Hole | | IOP | | EYE LID | | CONJUNCTIVA | | CORNEA | | PUPIL | | LENS | | RETINA | | DIAGNOSIS | | TREATMENT | | | | | |
|--------|-------------|---------|-----------|----------------|-----------|-------|----------|---------------|------------|---------|----------|-------|------|------|-------------|-------------|----------------|-----------|--------|--------|--------|--------|-------------------|-------------------|--------------------|--------------------------------|--|----------------------------------|-------------|-------------|-------------|----------|-----------|----------|
| | | | | | | | | | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RE | LE | RIGHT EYE | LEFT EYE | RIGHT EYE | LEFT EYE |
| | | | | | | | | | Reg/irreg. | | | | | | | | | | | | | | | | | | | | | | | | | |
| 1 | Yallappa | 41/M | 103948 | 1yr | Regular | 7.4 | 262/305 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | | |
| 2 | Chidanand | 75/M | 17887 | 15yrs | irregular | 7.1 | 178/234 | Nil | 6/9 | 6/6 | 6/6 | 6/6 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | PDR | PDR | PDR | PDR | PDR | PDR | Normal | Med mag | | | | |
| 3 | Mohanchara | 48/M | 134428 | 18yrs | Regular | 6 | 108/150 | Nil | 6/12 | 6/12 | 6/6 | 6/9 | 14.6 | 17.3 | normal | normal | Pterigium | Pterigium | normal | normal | normal | normal | normal | Normal | Normal | Pterygium | Pterygium | Med mag | Med Mag | | | | | |
| 4 | Gangabai | 55/F | 176678 | 3yrs | Regular | 6.6 | 156/235 | Nil | 6/36 | 6/36 | 6/9 | 6/9 | 14.6 | 15.9 | normal | normal | normal | normal | normal | normal | normal | normal | PSC | PSC | Normal | Normal | cortical cataract | cortical cataract | Med mag | Med Mag | | | | |
| 5 | B B Jadhav | 65/M | 156049 | 7yrs | irregular | 7 | 141/229 | Nil | 6/6 | 6/9 | 6/6 | 6/6 | 12 | 12 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | |
| 6 | Gurappa G | 52/M | 176303 | 6yrs | Regular | 6 | 112/123 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 13.4 | 12.2 | normal | normal | normal | normal | normal | normal | normal | normal | PSC | PSC | Normal | Normal | PSC | Normal | Med mag | Med Mag | | | | |
| 7 | Annasahet | 46/M | 9382 | 3yrs | Regular | 6.8 | 156/186 | | 6/36 | 6/36 | 6/9 | 6/9 | 12.2 | 12.2 | normal | normal | Pterigium | Pterigium | normal | normal | normal | normal | normal | Normal | Normal | Pterygium with PSC with Mild | Pterygium with PSC with PSC | Med mag | Med Mag | | | | | |
| 8 | Vitabai | 55/F | 9704 | 5yrs | irregular | 6.6 | 266/281 | 1+ | 6/24 | 6/9 | 6/24 | 6/18 | 12 | 10 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | MILD NPDR + APMD | MILD NPDR + APMD | Mild NPDR | Mild NPDR | Med mag | Med Mag | | | |
| 9 | Bhimanna | 75/M | 176490 | 17yrs | Regular | 7 | 237/337 | 2+ | 6/36 | 6/12 | 6/9 | 6/12 | 15.9 | 18.9 | normal | normal | normal | normal | normal | normal | normal | normal | PseudoP | PseudoP | Mild NPDR | Mild NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag | | | | |
| 10 | S M Shirha | 55/M | 136431 | 8yrs | irregular | 7.5 | 160/300 | Nil | CF | 6/9 | NI | 6/6 | 13.2 | 13.2 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Focal Laser | Focal Laser | | | |
| 11 | Sundraww | 45/F | 132313 | 6yrs | Regular | 6 | 299/388 | Nil | 6/9 | 6/6 | 6/6 | 6/6 | 17.3 | 17.3 | normal | normal | normal | normal | ulcer | normal | normal | normal | normal | Normal | Normal | Cor ulcer | Normal | Med mag | Med Mag | | | | | |
| 12 | Basanna N | 58/M | 111224 | 10yrs | Regular | 6 | 150/192 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | |
| 13 | S S Kedach | 70/M | 284098 | 30yrs | irregular | 7.5 | 170/310 | 2+ | 6/60 | 6/36 | 6/36 | 6/12 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | PSC | PSC | Mod NPDR | Mod NPDR | PSC with Mod NPDR | PSC with NPDR | Med mag | Med Mag | | | | |
| 14 | Basayya Hi | 62/M | 294043 | 5mon | Regular | 7.8 | 156/220 | Nil | 6/36 | 6/60 | 6/18 | 6/36 | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Mod NPDR | Mod NPDR | Mod NPDR IT tributary | Mod NPDR | Mod NPDR with Arterial occlusion | Med mag | Focal Laser | | | | |
| 15 | M R Bhavini | 80/M | 291776 | 4yrs | Regular | 6 | 130/198 | Nil | 6/36 | 6/6 | 6/12 | 6/6 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag | | | | |
| 16 | Mohan Ksh | 38/M | 192220 | 2yrs | Regular | 6 | 103/163 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 17.3 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | Cortical | normal | Normal | Normal | Cortical | Normal | Med mag | Med Mag | | | | |
| 17 | Kamanna | 61/M | 132290 | 5yrs | Regular | 6 | 236/266 | Nil | 6/36 | 6/36 | 6/24 | 6/24 | 10.2 | 12.2 | normal | normal | Pterigium | normal | normal | normal | normal | normal | NS1 | NS2 | Normal | Normal | Pterygium | Normal | Med mag | Med Mag | | | | |
| 18 | Rayappa M | 47/M | 132242 | 1yr | Reg | 6 | 110/134 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 17.6 | 17.6 | Stye | Chalazion | Conjunctivitis | normal | normal | normal | normal | normal | normal | Normal | Normal | Stye+Conjunctivitis | Chalazion | Med mag | Med Mag | | | | | |
| 19 | Shivamurti | 48/F | 107762 | 2yrs | Reg | 6.6 | 82/200 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.6 | 14.2 | Ac Dacr | normal | normal | normal | ulcer | normal | normal | normal | normal | Normal | Normal | Ac Dacr | Cor Ulcer | Med mag | Med Mag | | | | | |
| 20 | Uma | 62/F | 161798 | 13 yrs | Regular | 6.4 | 150/312 | 1+ | CF- 3m | PL | CF-3m | PL | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | normal | PseudoP | NS 2 | Mod NPDR with CSME | Mod NPDR | Normal | Normal | PRP | Focal Laser | | | | |
| 21 | Chayavva | 35/F | 8688 | 6yrs | Regular | 6.6 | 356/500 | 1+ | 6/60 | 6/6 | 6/36 | 6/60 | 12.2 | 12.2 | Blepharitis | Blepharitis | normal | normal | normal | normal | normal | normal | cortical cataract | Cortical cataract | Mild NPDR | Blepharitis with cataract with | Blepharitis with cataract with mild NPDR | Med mag | Med Mag | | | | | |
| 22 | Dundava | 55/F | 7192 | 8yrs | Regular | 8.8 | 198/309 | Nil | 6/36 | CF-3m | 6/12 | NI | 10.2 | 12.2 | normal | normal | normal | normal | normal | normal | normal | normal | normal | normal | Sev NPDR | Sev NPDR | Sev NPDR | Sev NPDR | Med mag | Med Mag | | | | |
| 23 | Umarmath | 58/F | 81443 | 12 yrs | Regular | 6.5 | 168/200 | Nil | 6/36 | 6/36 | 6/24 | 6/24 | 20.6 | 22.6 | Xanthelesma | Xanthelesma | normal | normal | normal | normal | normal | normal | Snow Flake | Snow flake | Mod NPDR | Mod NPDR | Xa | Normal | Focal Laser | Focal Laser | | | | |
| 24 | Jayalaxmi | 58/F | 81502 | 3yrs | Regular | 6.6 | 101/222 | Nil | 6/60 | 6/9 | 6/60 | 6/9 | 15.9 | 15.9 | normal | normal | normal | normal | normal | normal | normal | normal | NS3 | Early cortical | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | | |
| 25 | Gangabai | 55/F | 139499 | 10yrs | Regular | 6.1 | 152/232 | Nil | 6/60 | 6/18 | 6/18 | 6/9 | 12.2 | 12.2 | Blepharitis | Blepharitis | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | DES | DES | Med mag | Med Mag | | | | |
| 26 | Hulawwa B | 52/F | 139519 | 1yr | Regular | 6 | 96/117 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 8.9 | 7.6 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | |
| 27 | Kusuma Al | 59/F | 139521 | 3yrs | Regular | 6.4 | 180/263 | Nil | 6/12 | 6/12 | 6/12 | 6/12 | 22.4 | 20.6 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | POAG | POAG | Med mag | Med Mag | | | | |
| 28 | Sharda Gu | 50/F | 139520 | 4yrs | Regular | 6 | 227/340 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | |
| 29 | Vijaylaxmi | 40/F | 139588 | 10yrs | Regular | 6 | 113/237 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 11.2 | 12.2 | normal | normal | Pterigium | Pterigium | normal | normal | normal | normal | Cortical | normal | Normal | Normal | Pterygium+CC | Pterygium | Med mag | Med Mag | | | | |
| 30 | Sujatha M | 35/F | 139482 | 2yrs | Regular | 6.2 | 223/343 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.6 | 12.3 | Ac Dacr | Stye | normal | normal | ulcer | normal | normal | normal | Cortical | Cortical | Normal | Normal | Ac Dacr+CC | Stye+Cortical cataract+Mild NPDR | Med mag | Med Mag | | | | |
| 31 | chandrak | 40/F | 139441 | 4yrs | Regular | 6.6 | 209/334 | Nil | 6/9 | CF-2M | 6/9 | CF-2M | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Cortical | Normal | Normal | Normal | AIOL+Cortical | Med mag | Med Mag | | | | |
| 32 | Shardabai | 62/F | 134970 | 1yr | Regular | 7.8 | 145/205 | Nil | 6/60 | CF-3m | 6/36 | 6/60 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | PSC | PSC with NS3 | Normal | Normal | PSC | PSC with NS3 | Med mag | Med Mag | | | | |
| 33 | Mahadevi | 60/F | 192240 | 8yrs | Regular | 7.8 | 174/324 | Nil | 6/36 | 6/36 | 6/36 | 6/18 | 14.6 | 15.9 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag | | | | |
| 34 | Shantabai | 45/F | 176293 | 7yrs | Regular | 9 | 136/222 | Nil | 6/36 | 6/9 | 6/18 | 6/9 | 14.6 | 12.3 | normal | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR | Sev NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag | | | |
| 35 | Gangabai V | 55/F | 176678 | 3yrs | Regular | 6.6 | 125/239 | 1+ | 6/36 | 6/36 | 6/9 | 6/9 | 11.2 | 12.2 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Mild NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag | | | | |
| 36 | Mahadevi | 48/M | 1922298 | 6mon | Regular | 6.1 | 95/115 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 11.2 | 12.2 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | | |
| 37 | Kantabai | 60/F | 3085 | 20yrs | irregular | 9.7 | 247/437 | 2+ | 6/36 | 6/12 | 6/36 | 6/12 | 14.6 | 13.4 | normal | normal | normal | normal | normal | normal | normal | normal | Cortical Cataract | Cortical cataract | Severe NPDR | Severe NPDR | Cortical cataract with sev NPDR | Cortical Cataract with Sev NPDR | Focal Laser | Focal Laser | | | | |
| 38 | Hanumant | 61/F | 37324 | 5yrs | Regular | 8.8 | 166/240 | 1+ | 6/60 | CF-1.5m | 6/24 | NI | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Severe NPDR | Severe NPDR | Severe NPDR | Severe NPDR | PRP | PRP | | | | |
| 39 | Prabhavati | 41/F | 7103 | 2yrs | Regular | 6.4 | 164/347 | Nil | 6/6 | 6/9 | 6/6 | 6/6 | 13.4 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | | |
| 40 | Mallawwa | 54/F | 7146 | 5yrs | Regular | 7.6 | 130/190 | Nil | 6/24 | 6/24 | 6/12 | 6/12 | 17.3 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | cortical cataract | cortical cataract | Mod NPDR | Mod NPDR | Cortical cataract with Mod NPDR | Cortical cataract with Mod NPDR | Med mag | Med Mag | | | | |
| 41 | Basavraj K | 66/F | 2494 | 15yrs | irregular | 7.5 | 121/230 | 1+ | 6/12 | 6/18 | 6/6 | 6/12 | 13.4 | 14.6 | Stye | Chr Dacr | Conjunctivitis | normal | normal | normal | normal | normal | Normal | Cortical cataract | Normal | Stye+Conjunctivitis | Chr Dacr+CC | Med mag | Med Mag | | | | | |
| 42 | Jamabai ch | 46/F | 347658 | 1.5yrs | Regular | 8.5 | 159/229 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.2 | 11.2 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR | Sev NPDR | Sev NPDR | Sev NPDR | PRP | PRP | | | | |
| 43 | Chandappa | 60/M | 330212 | 5yrs | REGULAR | 7 | 69/89 | Nil | 6/60 | cf-1.5m | 6/36 | NI | 17.3 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | Pseudo P | Cortical cataract | Mild NPDR | Mild NPDR | Pseudo P with mild NPDR | Mild NPDR | Med mag | Med Mag | | | | |
| 44 | Neelawwa | 60/F | 338940 | 4yrs | Regular | 6.9 | 130/240 | Nil | 6/12 | 6/12 | 6/6 | 6/6 | 18.9 | 15.4 | Xanthelesma | Xanthelesma | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR | Sev NPDR | Sev NPDR | Sev NPDR | Focal Laser | Focal Laser | | | | |
| 45 | Kasturi | 62/F | 27488 | 20yrs | irregular | 7.6 | 217/335 | 2+ | 6/36 | CF-2m | 6/24 | NI | 12.2 | 14.6 | normal | normal | Pterigium | normal | normal | normal | normal | normal | Normal | PSC | Mod NPDR | Mod NPDR | Pterygium+Mod NPDR | Med mag | Med Mag | | | | | |
| 46 | Mallikarj | 45/M | 339252 | 10yrs | Regular | 8.4 | 196/268 | 1+ | 6/36 | 6/6 | 6/18 | 6/6 | 10.2 | 11.2 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Severe NPDR | Very Sev NPDR | Sev NPDR | PRP | PRP | | | | | |
| 47 | Rajashekh | 65/M | 34682 | 5yrs | Regular | 8.8 | 140/266 | Nil | 6/12 | 6/12 | 6/6 | 6/6 | 13.4 | 18.3 | Xanthelesma | Xanthelesma | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | ARM | Med mag | Med Mag | | | |
| 48 | Shivaputra | 60/M | 325647 | 1yr | Regular | 6.2 | 116/225 | Nil | 6/60 | 6/60 | 6/24 | 6/12 | 15.9 | 14.6 | normal | normal | normal | normal | normal | normal | normal | normal | Normal | NS3 | Normal | Normal | Normal | Normal | Med mag | Med Mag | | | | |
| 49 | Hanumant | 55/M | 3524 | 5yrs | Regular | 8.2 | 233/298 | 1+ | 6/60 | 6/36 | 6/24 | 6/36 | 14.6 | 17.3 | Blepharitis | Blepharitis | normal | normal | normal | normal | normal | normal | cortical cataract | cortical cataract | Mod NPDR | Mod NPDR | Cortical cataract with Mod NPDR | Cortical cataract with Mod NPDR | Med mag | Med Mag | | | | |
| 50 | Mallappa B | 65/M | 3641 | 4yrs | Regular | 6.5 | 160/290 | Nil | 6/12 | 6/12 | 6/9 | | | | | | | | | | | | | | | | | | | | | | | |

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|-----|-------------|------|--------|--------|-----------|------|---------|-----|-------|---------|------|-------|------|------|-------------|-------------|----------------|---------------|--------|--------|--------------------|--------------------|----------------------|--------------------|-----------------------------------|-----------------------------------|---------------------------------------|-------------------|-------------|-------------|
| 77 | Appasaheb | 66/M | 330302 | 1yr | Regular | 6.3 | 150/188 | Nil | CF-2m | CF-2M | 6/18 | 6/18 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | NS2 | NS2 | Normal | Normal | Cataract NS2 | Cataract NS2 | Med mag | Med Mag | | |
| 78 | Kalappa Ba | 48/M | 29756 | 4yrs | Regular | 6 | 166/189 | Nil | 6/9 | 6/12 | 6/9 | 6/9 | 13.2 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 79 | Ramesh Ar | 25/M | 29647 | New | Regular | 7 | 200/330 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 12.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 80 | Paradappa | 65/M | 29846 | 4yrs | Regular | 6.5 | 180/220 | 1+ | 6/60 | 6/60 | 6/18 | 6/18 | 14.2 | 14.2 | Xanthelesma | Xanthelesma | normal | normal | normal | normal | Normal | Nuclear cataract | Nuclear cataract | Normal | Xanthelesma with Nuclear cataract | Xanthelesma with Nuclear cataract | Med mag | Med Mag | | |
| 81 | B B Budhih | 51/M | 44964 | 10yrs | Regular | 8.8 | 160/220 | 1+ | 6/18 | 6/9 | 6/6 | 6/6 | 13.4 | 14.2 | normal | normal | normal | normal | normal | normal | normal | cortical cataract | cortical cataract | Sev NPDR | Severe NPDR | Cortical cataract with sev NPDR | Cortical cataract with sev NPDR | PRP | PRP | |
| 82 | Rabiya Ina | 55/F | 40730 | 1.5yrs | Regular | 6 | 112/180 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 12.2 | 13.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 83 | Mallangou | 55/M | 41024 | 4yrs | Regular | 6 | 113/186 | Nil | 6/6 | CF-F | 6/6 | NI | 14.2 | 15.9 | stye | normal | normal | normal | normal | normal | Normal | Cortical Cataract | Normal | Normal | Stye | Cortical cataract | Med Mag | Med Mag | | |
| 84 | M S Patil | 55/M | 41062 | 4yrs | Regular | 6.2 | 136/180 | Nil | CF-3M | CF-2M | 6/60 | CF-3M | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Cortical cataract | Normal | Normal | Cortical cataract | Cortical cataract | Med mag | Med Mag | |
| 85 | Appanna | 80/M | 37309 | 10yrs | irregular | 8.8 | 188/218 | 1+ | 6/24 | 6/36 | 6/18 | 6/24 | 18.9 | 20.6 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Cortical cataract | CSME | CSME | Cortical cataract with CSME | Cortical Cataract wuth CSME | PRP | PRP | |
| 86 | G H Matti | 62/M | 44893 | 3yrs | Regular | 6 | 115/158 | Nil | 6/6 | 6/12 | 6/6 | 6/9 | 12.2 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 87 | Appa sahe | 60/M | 4545 | 10yrs | irregular | 7.9 | 96/170 | Nil | 6/18 | 6/12 | 6/9 | 6/9 | 14.6 | 14.6 | normal | normal | normal | normal | normal | normal | Normal | Pseudo P | PseudoP | Mod NPDR | Mod NPDR | Pseudo P with Mod NPDR | Pseudo P with Mod NPDR | Med mag | Med Mag | |
| 88 | Basappa K | 66/M | 2252 | 10yrs | Regular | 8.6 | 230/330 | 1+ | 6/24 | 6/24 | 6/18 | 6/18 | 16.2 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Cortical cataract | Early PDR | Early PDR | Cortical cataract with PDR | Cortical cataract with PDR | Med mag | Med Mag | |
| 89 | Siddappa | 64/M | 21358 | 4yrs | Regular | 6 | 137/157 | Nil | 6/12 | 6/18 | 6/9 | 6/12 | 12.2 | 14.2 | chalazion | Chalazion | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Chalazion | Chalazion | Med mag | Med Mag | | |
| 90 | Basavraj t | 47/M | 4148 | 4 mont | Regular | 8.3 | 137/190 | Nil | 6/6 | 6/6 | 6/5 | 6/5 | 13.9 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Normal | Normal | Normal | Cortical cataract | Cortical cataract | Med mag | Med Mag | |
| 91 | Vishalaxm | 50/F | 4046 | 14yrs | irregular | 9 | 110/210 | Nil | 6/60 | 6/60 | 6/36 | 6/36 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Early PDR | Early PDR | PDR | PDR | Med mag | Med Mag | |
| 92 | Krishna | 50/M | 34221 | 8yrs | Regular | 9.7 | 170/224 | Nil | 6/6 | 6/18 | 6/6 | 6/9 | 13.9 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR with CSME | Sev NPDR with CSME | Sev NPDR with CSME | Sev NPDR with CSME | Focal Laser | Focal Laser | | |
| 93 | Awarappa | 60/M | 22118 | 1yr | Regular | 6 | 123/156 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 12.2 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 94 | Digambaba | 65/F | 17061 | 6/3F | irregular | 6.4 | 167/210 | Nil | 6/18 | CF-3m | 6/9 | 6/60 | 13.9 | 13.9 | normal | normal | normal | normal | normal | normal | Normal | Pseudo P | Cortical cataract | Normal | Normal | Pseudo P | Cortical Cataract | Med mag | Med Mag | |
| 95 | Mahadevi | 68/F | 9581 | 3yrs | Regular | 8.8 | 126/180 | Nil | 6/18 | 6/18 | 6/9 | 6/9 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag | |
| 96 | S Muliman | 48/M | 34619 | 2yrs | Regular | 8.5 | 136/196 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR with CSME | Sev NPDR with CSME | Sev NPDR with CSME | Sev NPDR with CSME | Focal Laser | Focal Laser | | |
| 97 | Savithri | 37/F | 24733 | 6yrs | Regular | 7.6 | 126/172 | Nil | 6/12 | 6/12 | 6/6 | 6/6 | 15.9 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Cortical | PDR | Normal | PDR | Cortical | Cortical | Med mag | Med Mag | |
| 98 | Shivanand | 58/M | 19790 | 13yrs | Regular | 8.2 | 260/310 | Nil | 6/12 | 6/24 | 6/9 | 6/18 | 18.2 | 18.2 | normal | normal | normal | normal | normal | normal | Irregular c | Pin point | Pseudo P | Pseudo P | PDR with CSME | Early PDR | Pseudo P with PDR with CSME | Pseudo P with PDR | Focal Laser | Focal Laser |
| 99 | Anand | 47/M | 5037 | 2yrs | Regular | 6.8 | 160/250 | Nil | 6/36 | 6/36 | 6/24 | 6/24 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Normal | Normal | Normal | CC | Focal Laser | Focal Laser | | |
| 100 | Sushadevi | 60/M | 20537 | 2yrs | Regular | 7.2 | 110/168 | Nil | 6/24 | 6/24 | 6/12 | 6/12 | 14.2 | 15.9 | normal | normal | normal | normal | normal | normal | Cortical witj NSII | Cortical with NSII | Myopic fundus | Myopic fundus | Cortical witj NSII | Cortical witj NSII | Med mag | Med Mag | | |
| 101 | GURUJING | 80/f | 230761 | 1.5yrs | regular | 7 | 116/156 | Nil | 6/9 | 6/18 | 6/9 | 6/9 | 12.2 | 12.2 | Act Dacr | normal | normal | normal | Ulcer | normal | Normal | Pseudo P | Pseudo P | Normal | Normal | Act Dacr | Cor Ulcer | Med mag | Med Mag | |
| 102 | M B Patted | 74/M | 36258 | 12yrs | irregular | 6.9 | 121/143 | 2+ | 6/60 | 6/36 | 6/24 | 6/18 | 18.9 | 18.9 | normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Mild NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag | | |
| 103 | Shashikala | 35/F | 26970 | 2yrs | Regular | 6 | 122/180 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Cortical | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 104 | Sanabai Pa | 70/F | 268812 | 2yrs | Regular | 6.3 | 101/150 | Nil | 6/60 | 6/60 | 6/18 | 6/18 | 12.2 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Choroidal atrophy | Choroidal atrophy | Med mag | Med Mag | | |
| 105 | Sidangou | 50/F | 270108 | 5yrs | regular | 6 | 115/170 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 12.2 | 10.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 106 | Sumashi G | 47/F | 238663 | 11yrs | Regular | 10.2 | 80/220 | Nil | CF-3m | CF-3m | 6/9 | 6/9 | 15.9 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Vitreous hemorrhage | Vitreous hemorrhage | Focal Laser | Focal Laser | |
| 107 | Annapurna | 60/F | 223043 | 2yrs | Regular | 7.1 | 86/200 | Nil | CF-2m | CF-1.5m | NI | 6/60 | 13.2 | 13.2 | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Cortical cataract | Normal | Normal | Cortical cataract | Cortical cataract | Med mag | Med Mag | |
| 108 | Heeranna | 60/M | 19817 | 1.5yrs | Regular | 6 | 90/123 | Nil | 6/60 | 6/60 | 6/36 | 6/36 | 10.9 | 10.9 | normal | normal | Conjunctivitis | Conjunctiviti | normal | normal | normal | normal | SIMC | SIMC | Normal | Conjunc+SIMC | Conjunc+SIMC | Med mag | Med Mag | |
| 109 | Rajaram | 75/M | 19290 | 5yrs | Regular | 6 | 104/140 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.9 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 110 | Geeta Patil | 43/F | 215514 | New | | 6.6 | 183/225 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 10.3 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 111 | Ansuysaba | 58/F | 215507 | 4yrs | irregular | 6.3 | 124/217 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 12.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 112 | Paramanna | 80/M | 18547 | 14yrs | irregular | 8.9 | 103/140 | Nil | 6/9 | 6/9 | 6/6 | 6/9 | 18.9 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag | | |
| 113 | Basamma | 70/F | 215524 | 6yrs | irregular | 6.8 | 126/186 | 1+ | CF-3M | CF-3m | 6/60 | 6/60 | 12.2 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 114 | Bhimappa | 65/M | 19563 | 7yrs | Regular | 6 | 116/166 | Nil | 6/60 | 6/60 | 6/60 | 6/60 | 18.9 | 18.9 | normal | normal | normal | normal | normal | normal | Normal | PSC | PSC | Mild NPDR | Mild NPDR | PSC | PSC | Med mag | Med Mag | |
| 115 | Drakshaya | 55/F | 215560 | 2yrs | regular | 6.4 | 145/222 | Nil | 6/12 | 6/9 | 6/6 | 6/6 | 13.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 116 | Shrinivas D | 83/M | 215522 | 14yrs | Regular | 6.1 | 146/286 | 2+ | 6/12 | 6/9 | 6/6 | 6/6 | 15.9 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag | |
| 117 | Shantappa | 73/M | 21549 | 2yrs | Regular | 6 | 95/116 | Nil | 6/24 | 6/24 | 6/9 | 6/6 | 13.2 | 14.2 | normal | normal | normal | normal | normal | normal | Cortical + PSC | Cortical cataract | Normal | Normal | Cortical + PSC | Cortical cataract | Med mag | Med Mag | | |
| 118 | Siddhango | 50/M | 21573 | 1yr | Regular | 6 | 96/159 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 119 | S M Birada | 50/M | 19855 | 5yrs | Regular | 6 | 108/150 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 10.3 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | CRAO | Normal | Med mag | Med Mag | |
| 120 | Siddlingapa | 45/M | 230777 | 4yrs | Regular | 6.2 | 100/156 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Normal | Normal | Mild NPDR | Med mag | Med Mag | | |
| 121 | Lakshmbiba | 55/F | 231130 | 3yrs | Regular | 6.2 | 116/178 | Nil | 6/18 | 6/12 | 6/9 | 6/9 | 10.3 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 122 | Siddappa | 49/M | 27842 | 4yrs | Regular | 6.6 | 93/201 | Nil | CF-2m | CF-2M | 6/12 | 6/18 | 14.2 | 14.2 | normal | normal | normal | normal | normal | normal | Normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag | |
| 123 | Neermala | 51/F | 208120 | 12yrs | irregular | 6.6 | 131/208 | 1+ | 6/6 | 6/6 | 6/6 | 6/6 | 18.9 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 124 | Sharanapp | 71/M | 207843 | 5yrs | Regular | 8.6 | 188/227 | Nil | 6/24 | 6/36 | 6/12 | 6/12 | 13.2 | 15.9 | normal | normal | normal | normal | normal | normal | Normal | Pseudo P | PSC + Cortical | Sev NPDR | Sev NPDR | Pseudo P with sev NPDR | PSC + Cortical cataract with Sev NPDR | Focal Laser | Focal Laser | |
| 125 | Hazarthi C | 60/F | 18167 | New | | 6.2 | 90/180 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 10.3 | 12.2 | Stye | stye | normal | normal | normal | normal | Normal | Normal | Mild NPDR + Microane | Mild NPDR | Stye + Mild NPDR | Stye + Mild NPDR | Med mag | Med Mag | | |
| 126 | Sharanapp | 65/M | 17692 | 1yr | irregular | 6.9 | 178/340 | Nil | CF-3M | CF-3m | 6/60 | 6/60 | 14.2 | 15.9 | normal | normal | normal | normal | normal | normal | Cortical cataract | cortical cataract | Normal | Normal | Cortical cataract | Cortical cataract | Med mag | Med Mag | | |
| 127 | Shantabai | 60/F | 211153 | 2yrs | Regular | 6.2 | 110/178 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.9 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 128 | Basavraj H | 47/F | 210226 | 1yr | Regular | 6.2 | 121/205 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 12.2 | 12.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | | |
| 129 | Mallikarjur | 55/M | 17378 | New | | 8.6 | 230/319 | Nil | 6/36 | 6/24 | 6/24 | 6/18 | 17.3 | 24.6 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | POAG | Med mag | Med Mag | | |
| 130 | Sangappa | 75/M | 19090 | 8yrs | irregular | 7.2 | 230/345 | 1+ | 6/36 | 6/60 | 6/24 | 6/36 | 15.9 | 18.2 | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Mod NPDR | Med mag | Med Mag | | |
| 131 | Laxmibai D | 54/F | 215487 | 7yrs | Regular | 6.2 | 110/160 | 1+ | CF-3M | CF-1m | 6/2 | | | | | | | | | | | | | | | | | | | |

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|-----|-----------------|--------|---------|---------|-----------|---------|---------|------|-------|---------|-------|-------|------|--------|-------------|-------------|-----------|-----------|--------|--------|--------|--------------------|-------------------|---------------|----------------|------------------------------|----------------------------------|------------|-------------|
| 159 | Madivalent | 50/F | 27219 | 13yrs | irregular | 7.4 | 166/238 | 2+ | 6/36 | 6/60 | 6/24 | 6/12 | 15.9 | 15.9 | stye | Stye | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Stye | Med mag | Med Mag |
| 160 | Kasturibai | 65/F | 305407 | 14 yrs | Regular | 7.2 | 136/196 | 1+ | 6/24 | 6/24 | 6/9 | 6/9 | 13.2 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Pseudo P | Pseudo P | Mod NPDR | Mod NPDR | Pseudo P + Mod NPDR | Pseudo P + Mod NPDR | Med mag | Med Mag |
| 161 | Annaraj | 65/M | 19404 | 5yrs | Regular | 6.9 | 110/170 | Nil | CF-1m | cf-1.5m | 6/60 | 6/60 | 18.9 | 15.9 | normal | normal | normal | normal | normal | normal | normal | PSC+NSII Cataract | PSC+NSII Cataract | Mild NPDR | Mild NPDR | PSC+NSII+Mild NPDR | PSC+NSII+Mild NPDR | Med mag | Med Mag |
| 162 | Vishwanath | 41/M | 139603 | 5yrs | Regular | 7 | 116/160 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 13.2 | 10.3 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 163 | Shantabai | 60/F | 139639 | 6yrs | irregular | 6.7 | 183/410 | Nil | CF-3M | CF-3m | NI | NI | 14.2 | 15.9 | Normal | Stye | normal | normal | normal | normal | normal | Cortical cataract | Cortical cataract | Mild NPDR | Mild NPDR | Cortical Cataract+Mild NPDR | Stye+Cortical cataract+Mild NPDR | Med mag | Med Mag |
| 164 | Mallappa | 55/M | 139470 | 4yrs | irregular | 8.6 | 170/280 | Nil | 6/36 | 6/18 | 6/9 | 6/9 | 13.4 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Cortical cataract | Cortical cataract | Normal | Normal | Cortical cataract | Cortical cataract | Med mag | Med Mag |
| 165 | Danakka B | 57/F | 155196 | 8mon | Regular | 6.6 | 170/204 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 15.9 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 166 | Sadashiv | 55/M | 265431 | 4yrs | Regular | 6.6 | 186/240 | Nil | 6/36 | 6/36 | 6/9 | 6/9 | 16.2 | 15.9 | Blepharitis | Blepharitis | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Blepharitis | Blepharitis | Med mag | Med Mag |
| 167 | Ashok | 48/M | 274335 | 6yrs | irregular | 8 | 120/168 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 10.3 | 10.3 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 168 | Sangappa | 53/M | 274317 | 7yrs | irregular | 8.2 | 160/266 | Nil | 6/24 | CF-3m | 6/18 | 6/6 | 15.9 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Cortical Cataract | Cortical Cataract | Early PDR | Early PDR+CSME | Cortical cataract+Early PDR | Cortical cataract+PDE+CSME | PRP | PRP |
| 169 | Sharada | 51/F | 272777 | 6yrs | irregular | 6.6 | 171/290 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 13.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Mild NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag |
| 170 | Shettappa | 50/M | 36354 | 2yrs | Regular | 6.8 | 110/156 | Nil | 6/24 | 6/18 | 6/9 | 6/6 | 14.2 | 14.2 | Blepharitis | Xanthelasma | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Blepharitis | Xanthelasma | Med mag | Med Mag |
| 171 | Prakash Ra | 37/M | 313289 | 2yrs | Regular | 7.8 | 100/146 | Nil | 6/36 | 6/24 | 6/9 | 6/9 | 15.9 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Cortical | Normal | Normal | Cortical | Med mag | Med Mag | |
| 172 | Shankarga | 56/M | 313763 | 12yrs | Regular | 7.6 | 103/235 | Nil | CF-2m | 6/9 | CF-2m | 6/6 | 10.3 | 13.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag |
| 173 | Gurupada | 61/M | 313328 | 1.5yrs | Regular | 6.3 | 118/179 | Nil | 6/12 | 6/12 | 6/6 | 6/6 | 13.2 | 13.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 174 | Bhagirathi | 69/F | 254120 | 8yrs | Regular | 6 | 106/180 | Nil | 6/24 | 6/6 | 6/9 | 6/6 | 15.9 | 17.9 | Stye | normal | normal | normal | normal | normal | normal | Cortical cataract | PSC | Normal | Normal | Stye+Cortical cataract | PSC | Med mag | Med Mag |
| 175 | Raju Dalaw | 47/M | 313290 | 11yrs | Regular | 6.9 | 112/216 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 18.9 | 15.9 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Mild NPDR+CSME | Mild NPDR | Mild NPDR+CSME | Med mag | PRP |
| 176 | Paravathi | 60/F | 28492 | 7yrs | irregular | 10.7 | 200/366 | Nil | 6/6 | HM | 6/6 | NI | 15.9 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Pseudo P | Brownish NSII | Mild NPDR | Glow absent | Pseudo P + Mod NPDR | NSII cataract | Med mag | Med Mag |
| 177 | Ranganna | 48/M | 313286 | 1.5yrs | irregular | 6.4 | 108/168 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 13.2 | 14.4 | normal | Normal | Pterigium | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Pterygium | Normal | Med mag | Med Mag |
| 178 | Neelkanth | 66/M | 304791 | 26yrs | Regular | 7.4 | 209/253 | 2+ | 6/12 | 6/12 | 6/9 | 6/9 | 17.9 | 15.9 | normal | normal | normal | normal | normal | normal | normal | PSC+ NSII | PSC+NSII Cataract | Mod NPDR | Mod NPDR+CSME | PSC+NSII+Mod NPDR | PSC+NSII+Mod NPDR+CSME | Med mag | Focal Laser |
| 179 | Siddappa | 45/M | 17582 | New | | 8.4 | 190/296 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 12.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Dry ARMD | Dry ARMD | Dry ARMD | Dry ARMD | Med mag | Med Mag | |
| 180 | Bangaraw | 80/F | 18635 | 9yrs | irregular | 6.3 | 187/260 | 1+ | CF-3M | CF-1m | CF-1M | NI | 14.2 | 12.2 | Pterigium | Normal | Pterigium | normal | normal | normal | normal | NS-III Cataract | NS III cataract | Mild NPDR | Mild NPDR | Pterigium+NS III +Mild NPDR | NS III +Mild NPDR | Med mag | Med Mag |
| 181 | Hanu Chav | 62/M | 18898 | 4yrs | irregular | 6.9 | 180/280 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 15.9 | 14.2 | Xanthelasma | Xanthelasma | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Xanthelasma | Xanthelasma | Med mag | Med Mag |
| 182 | Gangamma Terdal | 223496 | 4yrs | Regular | 6.8 | 140/240 | Nil | 6/24 | 6/24 | 6/18 | 6/24 | 10.3 | 12.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Early ARMD | Early ARMD | Early ARMD | Early ARMD | Med mag | Med Mag | |
| 183 | Ramappa | 54/M | 223029 | 4yrs | Regular | 7 | 90/175 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.2 | 15.9 | Stye | normal | normal | normal | normal | ulcer | normal | Normal | Normal | Normal | Normal | Stye | Cor ulcer | Med mag | Med Mag |
| 184 | Huvanna A | 72/M | 234456 | 1.5yrs | Regular | 6.6 | 128/178 | Nil | 6/9 | 6/9 | 6/6 | 6/6 | 18.9 | 15.9 | Blepharitis | | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Blepharitis | Normal | Med mag | Med Mag |
| 185 | Arjun Vand | 58/M | 10818 | 6 yrs | Regular | 6.4 | 106/160 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 13.2 | 14.2 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | |
| 186 | Pandappa | 60/M | 7859 | 9yrs | irregular | 7.2 | 118/178 | Nil | CF-3M | CF-3M | 6/60 | 6/60 | 15.9 | 10.3 | normal | normal | normal | normal | normal | normal | normal | Cortical+NSII | Subluxated -inj | Mod NPDR | Mod NPDR | Cortical+NSII + Mod NPDR | Subluxated lens+Mod NPDR | Med mag | Med Mag |
| 187 | G K Birada | 72/M | 81444 | 2yrs | Regular | 7 | 102/156 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 10.3 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | |
| 188 | Vishal Bida | 50/M | 43543 | 5.5yrs | Regular | 7 | 114/166 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | |
| 189 | Hanumant | 61/M | 37324 | 4.5yrs | Regular | 10.2 | 166/256 | 1+ | 6/60 | CF-1M | 6/24 | NI | 14.2 | 12.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Cortical cataract | Sev NPDR | Sev NPDR | Sev NPDR | Cortical+PSC + Sev NPDR | PRP | PRP |
| 190 | Ravindrana | 50/M | 153274 | 8yrs | Regular | 6.4 | 180/280 | Nil | 6/9 | 6/6 | 6/6 | 6/6 | 15.9 | 14.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | |
| 191 | Shankar Hi | 60/M | 153356 | 6yrs | irregular | 7.8 | 260/340 | Nil | 6/24 | 6/24 | 6/9 | 6/9 | 10.3 | 12.2 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag |
| 192 | Rajendra K | 51/M | 10995 | 6.5yrs | Regular | 6.4 | 96/146 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag | |
| 193 | Ashok Nar | 45/M | 156767 | 6mon | Regular | 6.4 | 130/200 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 15.9 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | BRAO | Normal | Normal | Med mag | Med Mag | |
| 194 | Shrishail | 70/M | 9406 | 11yrs | Regular | 6.4 | 92/158 | Nil | 6/60 | 6/60 | 6/24 | 6/24 | 18.9 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Mild NPDR | Mild NPDR | Mild NPDR | Med mag | Med Mag | |
| 195 | Laxman Hd | 57/M | 36565 | 1yr | Regular | 7.4 | 145/287 | Nil | 6/60 | 6/60 | 6/60 | 6/24 | 15.9 | 13.4 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag |
| 196 | S R Narale | 80/M | 35566 | 15yrs | irregular | 7.4 | 134/190 | 2+ | 6/18 | 6/24 | 6/18 | 6/18 | 17.3 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Pseudo P | Pseudo P | Normal | Normal | Pseudo P | Pseudo P | Med mag | Med Mag |
| 197 | Arjun Bida | 55/M | 34365 | 15yrs | irregular | 7.2 | 140/240 | 1+ | 6/6 | 6/6 | 6/6 | 6/6 | 17.3 | 13.4 | normal | normal | normal | normal | normal | normal | normal | Cruciform Cataract | Cruciform Cata | Mod NPDR | Mod NPDR | Cruciform cataract+ Mod NPDR | Cruciform cataract+ Mod NPDR | Vitrectomy | Vitrectomy |
| 198 | Mallikarju | 58/M | 231211 | 2yrs | Regular | 6.4 | 110/160 | Nil | 6/18 | 6/18 | 6/6 | 6/6 | 17.3 | 17.3 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Sev NPDR | Sev NPDR | Sev NPDR + Asteroid H | Sev NPDR + Asteroid H | Med mag | Med Mag |
| 199 | Chandappa | 60/M | 274670 | 5.5yrs | Regular | 8.8 | 80/128 | Nil | 6/36 | CF-3M | 6/12 | 6/60 | 14.6 | 17.3 | normal | normal | normal | normal | normal | normal | normal | Pseudo P | PSC + Nuclear I | Sev NPDR | Sev NPDR | Sev NPDR+PP | Sev NPDR+PSC | Med mag | Med Mag |
| 200 | N R Jadhav | 84/M | 300387 | 25yrs | irregular | 7 | 164/250 | 2+ | CF-3M | 6/24 | NI | 6/18 | 12.2 | 13.4 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Wet AMD | Dry ARMD | Wet ARMD | Wet ARMD | Med mag | Med Mag |
| 201 | Tarabai Ra | 62/F | 291778 | 1yr | Regular | 6.8 | 159/264 | Nil | 6/12 | CF-3m | 6/9 | NI | 10.3 | 14.6 | Normal | Normal | normal | normal | normal | normal | normal | Mature cataract | NSII cataract | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 202 | Shreedevi | 28/F | 291898 | 1mon | Regular | 8.2 | 139/380 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 14.6 | 15.9 | normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 203 | Vishnu Ga | 50/M | 26537 | 3yrs | Regular | 6.9 | 116/138 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 14.2 | normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 204 | Khajisab | 65/M | 25518 | 10yrs | irregular | 7.4 | 148/261 | 1+ | 6/60 | CF-3M | 6/36 | NI | 18.9 | 17.3 | Normal | Normal | normal | normal | normal | normal | normal | Normal | NS II Cataract | Normal | Normal | Normal | NSII cataract | Med mag | Med Mag |
| 205 | Mallawwa | 59/M | 29176 | 11yrs | irregular | 7.2 | 113/215 | 1+ | 6/18 | 6/9 | 6/9 | 6/6 | 12.2 | 14.2 | normal | normal | Pterigium | Pterigium | normal | normal | normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag |
| 206 | Subhas | 42/M | 21215 | 3yrs | Regular | 8 | 146/296 | Nil | 6/12 | 6/9 | 6/6 | 6/6 | 17.3 | 15.9 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Mod NPDR | Mod NPDR | Mod NPDR | Mod NPDR | Med mag | Med Mag |
| 207 | Mallapa | 66/M | 23990 | 6yrs | Regular | 6.8 | 118/168 | Nil | 6/24 | CF-3M | 6/18 | CF-2M | 18.9 | 17.3 | Normal | Normal | normal | normal | normal | normal | normal | NS II Cataract | NS III cataract | Normal | Normal | NS II Cataract | NS III cataract | Med mag | Med Mag |
| 208 | Gangamma | 50/F | 24236 | 2yrs | Regular | 8 | 170/270 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 10.3 | 10.3 | normal | normal | normal | normal | normal | normal | normal | Normal | Normal | mod with csme | mod with csme | Normal | Normal | prp | prp |
| 209 | Rajendra | 68/M | 21339 | 3yrs | irregular | 8.2 | 130/220 | Nil | CF-3M | CF-2M | 6/60 | 6/36 | 15.9 | 14.2 | Normal | Normal | normal | normal | normal | normal | normal | PSC | PSC | Normal | Normal | PSC | PSC | Med mag | Med Mag |
| 210 | Mahadevi | 50/F | 2014315 | 4yrs | Regular | 8.2 | 256/307 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 13.2 | 14.2 | Normal | Normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 211 | Mallamma | 52/F | 9856 | 2yrs | Regular | 6.6 | 96/120 | Nil | 6/6 | 6/6 | 6/6 | 6/6 | 11.3 | 17.3 | Normal | normal | normal | normal | normal | normal | normal | Normal | Normal | Normal | Normal | Normal | Normal | Med mag | Med Mag |
| 212 | Siddhamm | 72/F | 21044 | 3yrs | Regular | 6.4 | 108/239 | Nil | PL | | | | | | | | | | | | | | | | | | | | |

