"ASSOCIATION OF ALBUMIN LEVELS IN URINE WITH SEVERITY OF DIABETIC RETINOPATHY IN TYPE II DIABETES MELLITUS –A ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL."

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This is to certify that the dissertation entitled **"ASSOCIATION OF ALBUMIN LEVELS IN URINE WITH SEVERITY OF DIABETIC RETINOPATHY IN TYPE II DIABETES MELLITUS –A ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL."** is a bonafide research work done by **Registration No. : BK0111001**

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LIST OF ABBREVIATIONS

BMI	-	Body Mass Index
CURES	-	Chennai Urban Rural Epidemiology Eye Study
DRS	-	Diabetic Retinopathy Study
ETDRS	-	Early Treatment Diabetic Retinopathy Study
DME	-	Diabetic Macular Edema
CSME	-	Clinically Significant Macular Edema
NEI	-	National Eye Institute
DRVS	-	Diabetic Retinopathy Vitrectomy Study
DCCT	-	Diabetes Control and Complication Trial
UKPDS	-	United Kingdom Prospective Diabetic Study
UAE	-	Urine Albumin Excretion
PKC	-	Phosphokinase C
RAS	-	Renin Angiotensin System
VEGF	-	Vascular Endothelial Growth Factor
IGF	-	Insulin like Growth Factor
NPDR	-	Non Proliferative Diabetic Retinopathy
PDR	-	Proliferative Diabetic Retinopathy
DAG	-	Di Acyl Glycerol
PDGF	-	Platelet Derived Growth Factor
IRMA	-	Intra Retinal Microvascular Abnormality
NVD	-	Neovascularisation of the Disc
NVE	-	Neovascularisation Elsewhere
Hb	-	Hemoglobin
HbA1c	-	Glycosylated Hemoglobin
MAP	-	Mean Arterial Pressure
AAO	-	American Academy of Ophthalmology
WESDR	-	Wisconsin Epidemiological Study of Diabetic Retinopathy
RENAAL	-	**R**eduction of **E**ndpoints in **N**on-insulin dependent diabetes mellitus with the **A**ngiotensin II **A**ntagonist **L**osartan.

ABSTRACT

Background and Objective

Diabetic retinopathy is a leading cause of blindness in the world that increases the chances of losing sight about 25 times higher compared to normal individuals. Early detection and identification of the risk for the development of diabetic complications are important strategies to minimize the human suffering and costs incurred from the disease.

The objective of the study is to correlate the severity of diabetic retinopathy with levels of albumin in urine.

Methods

The study comprised a total of 100 patients with type II diabetes mellitus, examined at the Department of Ophthalmology, KLES DR.Prabhakar kore hospital, over a period of one year.

Ophthalmoscopic and biomicroscopic examination of ocular fundus was done. Grading of the severity of retinopathy was done according to the ETDRS classification. Urine albumin levels were estimated by random spot urine collection method. Patients were grouped into three groups {normoalbuminuria, microalbuminuria, macroalbuminuria} depending on their urine albumin excretion.

Results

There was a significant association between macroalbuminuria and PDR (p=0.00). Microalbuminuria was found to be associated with all grades of diabetic retinopathy with skewing towards the lesser grades.

Interpretation and Conclusions

Microalbuminuria, as a parameter for early predictor of diabetic retinopathy is not conclusively accurate. However, the presence of overt albuminuria in diabetic patients may signify an advanced grade of diabetic retinopathy.

Key words : Diabetic retinopathy ; urine albumin excretion ; microalbuminuria

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INTRODUCTION

Diabetes Mellitus can affect the eye in many ways. It is a major cause of avoidable blindness in both, the developing and the developed countries[1]. It increases the chances of loosing vision about 25 times higher compared to normal individual[2] Diabetic retinopathy is the most common and perhaps the most serious of all ocular complications[3]. It is considered as the hallmark of generalized microangiopathy occurring in a diabetic patient.

While there are multiple risk factors involved in the development and progression of retinopathy, the duration of the disease and the age of onset of diabetes are said to be the strongest predictors. Other risk factors like hypertension, pregnancy, blood glucose level control and presence of nephropathy are shown to have a strong association.

Microalbuminuria, dyslipidemia, BMI and smoking are some of the factors whose role as predictors of diabetic retinopathy is not well established.

Increased permeability related to endothelial dysfunction is described as one of the mechanisms in the pathology of diabetic retinopathy and nephropathy. Studies have been conducted to correlate diabetic retinopathy and urine albumin excretion, regarding them as two entities representing retinal and renal vasculature respectively. However it is not absolutely certain that whether the increased permeability is limited to retinal vessels or part of generalized microangiopathy affecting all the vessels of the body.

Around 246 million people in the world have diabetes[4]. Apart from its vision threatening effects, the presence of diabetic retinopathy increases risk of life threatening systemic vascular complications. Early diagnosis and intervention remains the key for preventing development and progression of diabetic retinopathy and other systemic complications. Microalbuminuria is the earliest clinical manifestation of nephropathy[5]

The prevalence of diabetic retinopathy in Chennai Urban Rural Epidemiology Eye Study (CURES) in South India was 17.6%, significantly lower than age matched western counterparts[6].

There is a growing concern for Asia being the region for diabetic epidemic. Despite this the epidemiological data for diabetic retinopathy is scarce[7,8]. The present study is undertaken to ascertain the correlation and to quantitatively assess the urine albumin excretion with the grade/severity of diabetic retinopathy.

OBJECTIVES

- To study the relationship between diabetic retinopathy and quantitative albuminuria.
- To correlate the urinary albumin excretion with the severity and grade of diabetic retinopathy.

REVIEW OF LITERATURE

History

Although diabetes was a well known disease since 2nd century A.D, no clinician attempted to link it with an eye pathology before the middle of 19th century. In pre-Christian era, the "Honey urine" was described by Susruta in Hindu medicine and was known in classical times and referred to as a "melting down of the flesh and limbs to urine" by Aretaues of Cappadocia. In 1846, French ophthalmologist Apollinaire Bouchardat (1806-1886) reported development of visual loss in absence of cataract in diabetics[9]. The implication of a macular disease in diabetics remained tentative until the invention of ophthalmoscope by Hermann von Helmholtz in 1851. It was Edward Jaguar (1818-1884) who first observed diabetic macular changes in 1855[7]. The association of diabetes and retinal complication was met with scepticism by other workers like von Graefe for more than a decade.

In 1885,Jaeger described changes in the retina in diabetic patients with albuminuria.The work of Arthur James Ballantyne (1876-1954) of Glasgow suggested that diabetic retinopathy represents a unique form of vasculopathy involving capillary wall alterations.

Gaudissart (1912) and Chabanier et all (1924) associated the retinal changes with hypercholesterolemia

Dejean (1932) and Villard and his colleagues (1933) considered an increase in toxic polypeptides in the blood as an aetiological factor

Wagner (1945) established a close relationship between the nephropathy and retinopathy in diabetes

The 1950s and 1960s were the periods of active research into the pathology of diabetic retinopathy. Though the primary emphasis of the majority of studies was on the abnormal vessel wall, metabolic parameters affecting the disease process were given due attention.

In 1968, more than 50 specialists from around the world met at a conference centre called the. "Airlie House" where a symposium was held to provide standard descriptive techniques as well as comparable formats for reporting the results of diabetic retinopathy. The main aims of the symposium were

1. Classification of diabetic retinopathy.
2. Natural history and visual progress evaluation.
3. Relation of retinopathy to metabolic control.
4. Pituitary ablation.
5. Photocoagulation.

The 'Diabetic Retinopathy Study' (DRS) from 1971-1976 sponsored by the National Eye Institute (NEI) evaluated the effectiveness of photocoagulation (Xenon/Argon) for treating diabetic retinopathy [10]. Another important study sponsored by the National Eye Institute was the ETDRS (Early Treatment of Diabetic Retinopathy Study). The study was concerned with the management of diabetic retinopathy and diabetic macular edema with photocoagulation and evaluation of effectiveness of aspirin for preventing diabetic retinopathy progression. ETDRS also

defined clinically significant macular edema (CSME) and recommended treatment modalities with photocoagulation[11].

Another sponsored study from NEI was 'Diabetic Retinopathy Vitrectomy Study' (DRVS) which was carried out from (1976-1985). It advocated the guidelines for early vitrectomy in diabetic retinopathy[12].

Diabetic retinopathy study (DRS) the Early Treatment Diabetic Retinopathy Study (ETDRS) and the Diabetic Vitrectomy Study (DRVS) have developed highly specific recommendations for the appropriate management of diabetic retinopathy

The 'Diabetes control and complication trial' (DCCT) showed that the intensive control reduced the risk of developing retinopathy by 76% and slowed progression of retinopathy by 54%. It also reduced the risk of albuminuria by 54%[13]. The 'United Kingdom Prospective Diabetic Study' (UKPDS) showed that the intensive glycemic control is associated with a reduced risk of newly diagnosed retinopathy and is also associated with reduced progression of existing retinopathy[13].

Decrease in glycosylated hemoglobin level was associated with a significant decrease in the progression of diabetic retinopathy as well as the incidence of proliferative diabetic retinopathy[14]

Literature Survey

In a study conducted by Nielsen et al, patients with median duration of disease of 21 years were taken. Classification of retinopathy was based on direct ophthalmoscopy and colour fundus photography. Advanced retinopathy was found in 11 patients of 36 with normal urine protein excretion rate. Advanced retinopathy was also found in 13 of 33 patients with tubular proteinuria only. In contrast, five of nine patients with above normal urinary excretion values of albumin had advanced retinopathy[15].

Erasmus et al conducted a study in 113 Nigerian diabetics and observed a high prevalence of microalbuminuria (≤ 30mg/24hrs) in both male (54%) and female diabetics (59%). 16% of the patients had diabetic retinopathy. Among 49 patients with normoalbuminuria (<30mg/24hrs), six (12%) had retinopathy compared to 12 (18%) in the microalbuminuria group. These observations suggested that there is a high prevalence of microalbuminuria amongst Nigerian diabetics and it may not predict retinopathy[16].

Brocco et al showed in his study that in renal biopsies revealing many interstitial changes (33%), only 31% had diabetic retinopathy. In patients with obvious diabetic glomerulopathy (26%) all had diabetic retinopathy[17].

Fiorette et al described a heterogeneity in renal structure of diabetics and 50% of these patients had no retinopathy. The same group of investigators extended this data in a cohort study of 53 patients with type II diabetes mellitus with microalbuminuria. Out of these patients, 41% had normal renal biopsies and in 59% of these patients, no retinopathy was seen[18].

Al Futaisi et al did a study on 261 patients with mean age of 50 years. 27% of the patients had microalbuminuria. The logistic regression model indicated that the odds of developing microalbuminuria increased with increase in HbA_{1C}, increase in log creatinine values and in those with hypertension[19] .

Klein R et al did a population based study in Wisconsin to examine relationship between gross proteinuria and proliferative diabetic retinopathy. They concluded that gross proteinuria is a risk indicator of proliferative retinopathy in younger patients with early onset of diabetes[20].

Serter et al studied 20 type II diabetes mellitus patients in early stages of diabetic complications. In these patients 'overnight albumin excretion rate' showed the best correlation to retinopathy. Those patients with 'overnight albumin excretion rate' of >7g/min revealed microaneurysms before microalbuminuria developed. Their study concluded that there is a good correlation between diabetic retinopathy and proteinuria and that albuminuria is an important indicator for early diabetic microangiopathy[21].

Savage et al conducted a study in which 947 type II diabetes mellitus patients were taken and categorized based on their urine albumin excretion [UAE]. The levels of UAE were compared with retinopathy as assessed by stereoscopic fundus photography. Chi Square test revealed UAE was significantly associated with presence of retinopathy ($p<0.001$). In multiple logistic regression analyses, UAE has a strong independent association with retinopathy[16].

Cruckshanks K J et al investigated the relationship between microalbuminuria and the presence and severity of diabetic retinopathy in a population based cohort

study. It was concluded that microalbuminuria was cross sectionally associated with presence of retinopathy in persons with diabetes[22].

Klein Ronald et al did a population based cohort study on 996 diabetic patients. The incidence and progression of diabetic retinopathy were determined by masked grading of stereoscopic color fundus photographs using modified ETDRS scale. Gross proteinuria was determined using dipstick. The results of these prospective data suggested that glycosylated Hb level was strongly related to incidence and/or progression of diabetic retinopathy and incidence of gross proteinuria[23].

Condonnier et al investigated 26 type II diabetes mellitus patients with albuminuria ranging from 70 to 4210 mg/24 hours and reported nonspecific vascular and glomerular damage in 15% of the patients. All patients with diabetic retinopathy had diabetic glomerulopathy and two patients with non diabetic glomerulopathy lacked diabetic retinopathy[24].

Kalk et al observed the ethnic difference in clinical and laboratory associations with retinopathy in type II diabetes mellitus patients. 547 patients of African, European and Indian origin were screened by mydriatic 60^{o} retinal photography. Severe degree of retinopathy was associated with the duration of the disease, macroalbuminuria and people of African origin ($P = 0.002$). But the association of severe retinopathy with microalbuminuria was not significant ($P = 0.01$)[25].

Parvanova et al did a cross sectional study of 115 type II diabetes mellitus patients. In this study, one progressive diabetic retinopathy case, one reference case and two controls without retinopathy were evaluated by indirect ophthalmoscopy and

retinal photography. It was found that those with progressive diabetic retinopathy had higher levels of albuminuria (P>0.05) compared to patients without retinopathy[26].

Kondavati Suresh Babu et al study between Glycated albumin and glycated hemoglobin (Hba1c) concluded that there is strong association between glycated albumin ,microalbuminuria with diabetic retinopathy [27]

Rani et al study concluded that individuals with microalbuminuria were around 2 times as likely to have Diabetic retinopathy as those without microalbuminuria , and this risk became almost 6 times in the presence of macroalbuminuria[28]

PATHOLOGY OF DIABETIC RETINOPATHY

The pathogenesis of diabetic retinopathy can be described in the following ways:

1. Retinal vascular changes.
2. Biochemical pathways.

1. RETINAL VASCULAR CHANGES

It includes

Capillary basement membrane thickening

Loss of microvascular intramural pericytes

Break down of Blood Retinal Barrier

Capillary basement membrane thickening

- Basement membrane serves as filtration barrier for molecules of various size and electrical charges. Alteration in the amount or degree of sulfation or in the anatomic distribution of highly negatively charged heparan sulfate proteoglycan molecules within the basement membrane can affect their permeability properties to various ions[29].

- Basement membrane collagen is extensively glycated which may be either qualitatively or quantitatively altered by enzymatic or non enzymatic processes.

- Another function of basement membrane is to regulate cell proliferation and differentiation[29]. The role of proteolytic enzymes that degrade basement membrane components is thought to be important in blood vessel growth, whether in normal development, in tissue repair or in the pathological neovasularisation of the disease.

Loss of microvascular intramural pericytes

- The mechanism by which pericytes are specifically lost early in diabetic retinopathy is unknown.

- It may be related to the action of sorbitol pathway since they find aldose reductase specifically in retinal capillary pericytes but not in endothelial cells.

- Another study postulates that the alterations of PDGF – B (platelet derived growth factor) secretion or function produced by prolonged hyperglycemia or

galactosemia, may selectively affect the pericyte viability leading to their loss by apoptosis[30].

Microaneurysms

- The earliest clinically observable lesion of diabetic retinopathy are the microaneurysms.

- Retinal capillary microaneurysms may represent focal regions of endothelial cell proliferation, where the antiproliferative effect of pericytes has been lost. This explains the development of cellular microaneurysms but not acellular ones.

- It is assumed that all microaneurysms are initially cellular, but some become acellular as a result of extensive apoptosis involving endothelial cells as well as pericyte nuclei.

- Another explanation of microaneurysms formation is that they may arise from weak points in the capillary wall following loss of pericytes.

- The point against this reasoning for microaneurysm development is the presence of microaneurysm in diseases wherein pericyte loss has not been observed[29].

Break down of Blood Retinal Barrier

- One possible cause of blood retinal barrier breakdown is opening of tight junctions (zonulae occludentes) between adjacent microvascular endothelial processes.

- Several proteins are closely involved with tight junction formation and function. The most widely studied of these are Zo-1 and occludin.

- In experimental diabetes, investigators found a reduced expression and anatomic distribution of occludin[29].

- Another vascular endothelial cell abnormality that may contribute to the breakdown of the blood – retinal – barrier is fenestration of endothelial cell cytoplasm. Fenestrae are normally absent in the thick endothelium of retinal capillaries, but they have been observed in subjects with retinal neovascularisation in which blood retinal barrier has broken down.

- Another possible explanation is an increase in transport by endocytic vesicles.

2. BIOCHEMICAL PATHWAYS

Even though the cellular mechanisms through which hyperglycemia acts is unclear, various mechanisms have been proposed in relation to biochemical pathways underlying diabetic retinopathy.

i) Non enzymatic binding of sugars to proteins leads to formation of glycated products with very long cellular lifetimes. These undergo a series of additional reactions leading to inter and intrachain cross linking with considerable alteration of protein function.

ii) Accelerated oxidative stress in the cells leads to formation of excess of 'toxic end products of oxidation' $\rightarrow$ peroxides, superoxides, nitric oxide and oxygen free radicals which may remain elevated due to chronic changes in metabolic pathway[31].

iii) Changes in the enzymatic pathways due to prolonged hyperglycemia.

The following are the important biochemical factors implicated in the pathogenesis.

1. Vascular endothelial growth factor (VEGF)
   - Stimulates angiogenesis and increases capillary permeability leading to neovascularisation and retinal edema respectively.
2. Renin – Angiotensin
   - Upregulation of renin-angiotensin system occurs in diabetes mellitus.
   - Angiotensin II might stimulate VEGF expression in retinal vascular endothelial cells.

3. Erythropoetin
    - Expressed primarily due to retinal ischaemia and possibly due to hyperglycemia, oxidative stress, inflammatory cytokines.
    - Can promote VEGF – independent angiogenic activity in retinal vascular endothelial cells.
4. Diacylglycerol (DAG) and Protein Kinase C (PKC)
    - Elevated DAG and PKC activity in the retina correlates with decreased blood flow rate; also implicated in renal abnormalities like increased albumin excretion and GFR (Glomerular filtration rate).
    - PKC in retinal cells gets activated due to hyperglycemia leading to increased expression of matrix proteins and vasoactive mediators with adverse structural and functional retinal vascular changes.
5. Sorbitol
    - Hyperglycemia increases glucose flux through the polyol pathway.
    - Aldose reductase converts aldose sugars (glucose) to their respective sugar alcohols (sorbitol).
    - Sorbitol possibly induces osmotic damage to retinal endothelial cells and pericytes.
6. Growth hormone and insulin growth factor (IGF)
    - These modulate the function of retinal endothelial precursor cells and drive retinal angiogenesis in response to hypoxia.
    - IGF-1 can also disrupt the blood-retinal barrier and increase retinal vascular permeability.
7. Carbonic anhydrase
    - Intraocular carbonic anhydrase is increased in diabetic retinopathy.

- Extracellular carbonic anhydrase increases retinal vascular permeability by increasing pH leading to Kallikrien – mediated proteolytic activation of Kinin.

CLINICAL FEATURES

1. MICROANEURYSMS

- Retinal arteriolar dilation might be an early physiological indicator of microvasular dysfunction which signifies impaired arteriolar auto regulation[32].

- This retinal arteriolar dilation according to the laws of Laplace and Starling increases retinal capillary pressure leading to capillary wall dilation → microaneurysms[33].

- These can occur at any level between superficial and deeper retinal capillary networks or even from choroidal circulation[34].

- The size usually ranges from 12-100 μm in diameter. But only those larger than 30μm are visible clinically[35]. An upper limit of 125 μm with sharp margins, smooth borders, round shape and central light reflex are considered if a lesion is to be distinguished as a microaneurysm[36].

- The aneurysms reflect:

  (i) Out pouching from capillary wall due to loss of pericyte support.

  (ii) Active cellular response to retinal hypoxic insult

2. INTRARETINAL HEMORRHAGES

- The intraretinal hemorrhages appear secondary to ruptured microaneurysms, capillaries or venules.
- The shape of the hemorrhage is dependent on the location of the hemorrhage within the retinal layers.
- Commonly the intraretinal hemorrhages are of two types:
  i) Superficial
  ii) Deep

i) Superficial

- These are usually flame shaped and occur in the nerve fibre layer of the retina.
- The shape is due to
  - Tighter organization of the cells in the nerve fibre layer.
  - Relative paucity of extracellular space due to compact nerve fibre arrangement.

ii) Deep

- They are also referred to as 'Dot and Blot' hemorrhages.
- Dot hemorrhages have very distinct borders and blot hemorrhages have fuzzier borders.
- The looser arrangement of the cells in the deeper layers and ample extracellular space allows the hemorrhage to take a larger form/shape.

- Some hemorrhages may have a white centre which probably represents auto occlusion.
- Hemorrhage in the retina block fluorescence and appear as areas of hypofluorescence unlike microaneurysms.
- The intraretinal hemorrhages resolve within 6 to 12 weeks.

- Their usual site is the posterior pole, however they can occur anywhere in the fundus.
- They usually do not cause visual obscuration unless they are situated at the fovea.

3. HARD EXUDATES

- These refer to the serum lipoproteins and lipid laden macrophages located within the outer plexiform layer. These accumulations occur due to the leakage of abnormal permeable vessels.
- The pattern is usually in the form of streaks or clusters or in circinate arrangement around microaneurysms.
- Ophthalmoscopically, they appear glistening, yellowish-white and waxy, scattered along the posterior pole with an affinity to macula.
- Hard exudates may either resolve spontaneously or may organize into hard plaques, eventually forming a disciform scar.

4. SOFT EXUDATES/COTTON WOOL SPOTS

- Extensive arteriolar closure in severe and advanced NPDR leads to infarcts of the nerve fibre layer. These are referred to as cotton wool spots/soft exudates.
- Pathologically, occlusion (or) decreased flow of an arteriole leads to axoplasmic stasis and retinal tissue swelling in the nerve fibre layer.
- These appear as bright, fluffy, whitish-yellow lesions with fuzzy margins.
- They usually resolve in 2-3 months but may take much longer.

- Residual nerve fibre layer atrophy and ganglion cell atrophy at the site where a cotton wool spot existed is referred to as 'depression sign of Goldmann'.

- 'Strict' or 'Rapid' metabolic control in patients with diabetes leads to increase in the number of cotton wool spots.

5. VENOUS BEADING

- Focal areas of venous dilatation with thinning of venous wall → venous beading.

- These changes are associated with capillary non-perfusion and retinal ischaemia.

- Histopathologically, the walls of beaded veins become thickened later and undergo hyaline degeneration.

- Venous beading is correlated with increased probability of progression to proliferative retinopathy.

- It is the most powerful individual predictor for future disease.

6. IRMA (INTRARETINAL MICROVASCULAR ABNORMALITIES)

- This refers to dilated, tortuous telangiectatic channels that occur between arterioles and venules.
- These represent intraretinal neovascularisation.
- These channels have minimal dye leakage compared to surface neovascularised fronds.
- It is associated with increased risk of developing proliferate retinopathy.
-

7. NEOVASCULARISATION

- It is the hallmark of PDR. It refers to new vessels that arise from retina or optic disc and proliferate along retinal surface or into vitreous with or without a fibrous component[37].

- It is most commonly associated with mid-peripheral capillary non-perfusion.

- It is most commonly located posteriorly within 45^{o} of the optic disc and/or on the optic disc itself.

NVD

- New vessels located on or within one disc diameter of the optic disc.
- Appear as fine wisps of blood vessels looping across other disc vessels.
- They create an advancing edge as they branch earlier forming proximal loops.
- It is best appreciated by Goldmann contact lens or non contact lens like Hruby lens or 78D lens.

NVE

- Neovascularisation anywhere in the retina apart from NVD is termed NVE.
- Seen as wheel like network of fine vessels from retinal veins/capillaries crossing between arterial and venous sides.
- Well made out by indirect and direct ophthalmoscopy.
- New vessels originate intraretinally but eventually break through the retinal internal limiting membrane and proliferate.

CLASSIFICATIONS

Various terminologies and clinical classifications have been used as a clinical scale to distinguish various grades of diabetic retinopathy. A few of them are enlisted here.

1. Fukuda classification.
2. Davis classification.
3. Scotts classification.
4. O'Hare classification.
5. Airlie House classification.
6. Modified Airlie house classification.
7. Early Treatment Diabetic Retinopathy Study (ETDRS).
8. Diabetic Retinopathy Study (DRS).
9. AAO 2002 classification[38].

Apart from these, many simplified versions of classification are used to grade the severity of the disease. One of the important classifications commonly used is the ETDRS classification[34]. This has been described below:

ETDRS CLASSIFICATION[34]

In this there are two major divisions:

1. Non Proliferative Diabetic Retinopathy (NPDR).
2. Proliferative Diabetic Retinopathy (PDR).

NPDR has again been subdivided into:

a) Mild to Moderate NPDR

- Microaneurysms.
- Intraretinal hemorrhages < 4 quadrants.
- Hard exudates.
- Foveal avascular zone abnormalities.

b) Moderate to severe NPDR

- Cotton wool spots.
- Intraretinal hemorrhages in 4 quadrants.
- Venous beading.
- Intraretinal microvascular abnormalities.

c) Severe NPDR

Any one of the following

- Severe intraretinal hemorrhages in 4 quadrants.
- Venous beading in two quadrants.
- Moderately severe IRMA in one quadrant.

d) Very severe NPDR

Any two of the following

- Severe intraretinal hemorrhages in 4 quadrants.
- Venous beading in two quadrants.
- Moderately severe IRMA in one quadrant.

PDR can be categorised as:

1. Early proliferative retinopathy (without DRS high risk characteristics)*

2. High risk proliferative retinopathy (with DRS high risk characteristics)*

* DRS high risk characterstics constitutes any three of the following:

1. Presence of new vessels

2. Location of new vessels on the disc or within one disc diameter of the optic disc (NVD)

3. Severity of the new vessels

If NVD ≥ one quarter or one third disc area.

If NVE ≥ one half disc area.

(If both NVD and NVE are present then the severity of NVD is taken into account)

4. Presence of preretinal or vitreous hemorrhage.

The ETDRS also defined clinically significant macular edema (CSME) and recommended treatment with focal laser photocoagulation for the following

1. Retinal edema located at or within 500μ of the centre of the macula.
2. Hard exudates at or within 500μ of the centre, if associated with thickening of adjacent retina.
3. Zone of thickening larger than 1 disc area if located within 1 disc diameter of the centre of the macula.

There have been numerous studies in the past reporting a relationship between microalbuminuria (or) proteinuria and diabetic retinopathy.

Retinopathy and nephropathy are both related to endothelial dysfunction mediated microvascular complications of diabetes mellitus[39].

The prevalence of PDR was much higher in patients with persistent microalbuminuria[40]

The severity (or) the presence / absence of lesions at one site may predict the status of the other[41,42]. Diabetic retinopathy, as measured by albuminuria, proteinuria and renal failure is found to be a risk factor (for nephropathy) associated in some but not all studies[43].

The presence of microalbuminuria is taken as an index of microangiopathic and macroangiopathic morbidity and motality[44]

The relationship between the two is complex and is frequently characterised by the presence of certain common predisposing factors. Chronic hyperglycemia, elevation of HbA_{1c}, duration of the disease and associated hypertension are risk factors for both retinopathy and microalbuminuria.

Hypertension as well as the duration of diabetes can also confound some of the effects of renal disease on diabetic retinopathy.

Assessment of microalbuminuria can be performed by three methods -

1. Measurement of albumin to creatinine ratio in a random spot collection of urine.
2. 24 hour urine collection with creatinine, allowing simultaneous measurement of creatinine clearance.
3. Timed collection (eg. 4 hour or overnight collection).

The first method is often found to be the easiest and generally provides accurate information.

DEFINITIONS OF ABNORMALITIES IN ALBUMIN EXCRETION[45]

Category	24hr collection (mg/24hr)	Timed collection («g/min)	Spot collection («g/mg creatinine)
Normal	< 30	< 20	< 30
Microalbuminuria	30-299	20-199	30-299
Macroalbuminuria	≥ 300	≥ 200	≥ 300

DIAGNOSIS

The aim of diagnostic modalities should be to detect

i) Serious ocular manifestations of diabetes mellitus.

ii) In absence of serious manifestations, (eg. Vitreous hemorrhage, Retinal detachment, maculopathy) assess the risk of progression to vision threatening disease.

The methods of diagnosis / monitoring / screening include

- Direct ophthalmoscopy
  - Permits an adequate but incomplete assessment.
  - Value enhanced by slit lamp biomicroscopy with condensing lens.
- Retinal photography
  - High sensitivity and specificity for detection of diabetic retinopathy if read by trained interpreters[46,47].
  - It can guide appropriate ophthalmic referral[47].

- However, accessibility / affordability is difficult.

- Fluorescein angiography
  - Aids in clinical assessment of diabetic retinopathy.
- Optical coherence tomography
  - Optical biopsy of the retina.
  - Gives high resolution, three dimensional images which approximate the histology of the retina.
  - Gives precise and reproducible measurements.
  - Useful for detection of structural changes requiring surgical intervention.
  - Helps to monitor the progression and response to treatment in macular edema.

MONITORING / SCREENING

- In practice, timing and frequency of eye examination in people with diabetes are often individualized.

- Regular eye examinations are effective for detection and monitoring of asymptomatic vision threatening retinopathy.

- Liverpool diabetic eye study suggests a three year screening interval for patients without evidence of retinopathy as a safe method[48].

- Preferred Practice Pattern committee, retinal panel, AAO (2003) suggests the following timetable based on retinopathy findings[49].

Retinal abnormality	Suggested follow up
Normal	Annual
Mild NPDR	Every 9 months
Moderate NPDR	Every 6 months
Severe NPDR	Every 2-4 months
CSME	Every 2-4 months
PDR	Every 2-3 months

The importance of screening is highlighted by the fact that in the WESDR study, 33% of patients with type II diabetes mellitus developed diabetic retinopathy within 5 years of diagnosis of the disease.

Diabetic retinopathy screening should be done at the diagnosis of diabetes mellitus and either yearly or every second year thereafter.

TREATMENT MODALITIES

The treatment of diabetic retinopathy should have a holistic approach. The treatment consideration should include:

i) General measures.

ii) Specific treatment of diabetic retinopathy.

I. GENERAL MEASURES

These are directed towards modifiable risk factors.

a. Glycemic Control

As hyperglycemia instigate the cascade of events leading to diabetic retinopathy, tight control of glycaemia reduces the risk of development and progression of diabetic retinopathy.

b. Blood Pressure Control

Hypertension is a common co-existing condition in diabetics and it exacerbates diabetic retinopathy by mechanical damage of vascular endothelial cells and stimulating VEGF release.

Lowering of blood pressure by using lisinopril can reduce the risk of retinopathy progression by 50% and proliferative retinopathy by 80% while using candesartan increased the regression of retinopathy by 34%[50].

c. Lipid Lowering Therapy

Studies have shown that lipid modifying agents reduce the need for laser treatment of vision threatening diabetic retinopathy by 31% in patients with type II diabetes mellitus[51].

d. Regular exercise with diet modification

II. SPECIFIC OCULAR THERAPY FOR DIABETIC RETINOPATHY

a. Laser Photocoagulation

- Mainstay for vision threatening diabetic retinopathy.
- However, even with adequate laser therapy reversal of visual loss is uncommon.
- The two basic types of laser therapy are :
    a. Pan retinal photocoagulation for PDR.
    b. Focal or grid photocoagulation for diabetic macular edema.
- Pan retinal photocoagulation reduces the risk of severe visual loss by 50% over 5 years in PDR.
- Macular laser reduces the risk of moderate visual loss from CSME by half[52].

b. Surgical intervention

- Vitrectomy is the mainstay surgical treatment for advanced retinopathy with persistent vitreous hemorrhage and tractional retinal detachment.
- It reduces the risk of retinal neovascularisation and macular edema, while increasing the risk of iris neovascularisation and cataract formation[53].
- It has also been suggested as a treatment option for diabetic macular edema refractory to laser.

NEWER TREATMENT OPTIONS

1. Anti VEGF agents

VEGF is a therapeutic target for diabetic retinopathy as it is a mediator of abnormal retinal vessel growth and leakage[54].

Anti VEGF therapy is delivered by injection directly into the vitreous, thus theoretically ensuring local efficacy.

However the long term safety of anti VEGF therapy in the management of diabetic retinopathy has not yet been established[55].

2. Intraocular Steroids

- These are used intravitreally for treatment of diabetic macular edema.
- They cause modest and short term vision improvement[54].
- They have an adjunctive role to laser in diabetic retinopathy management.

Potential newer molecules for management of diabetic retinopathy[56]

AGENT	DRUG / MOLECULE
Antiplatelet / Anticoagulant	Aspirin, Ticlopidine
Aldose reductase inhibitors	Ponalrestat / Tolerestat
Growth Hormone Supressors	Octreotide
Anti-Angiogenic agents	Curcumin
ACE inhibitors	Candesartan / Perinodopril
PKC Inhibitors	Ruboxistaurin*

*Ruboxistaurin (dose of 32 mg/day) has decreased the risk of vision loss, macular edema progression and laser treatment for macular edema[57,58].

MATERIALS AND METHODS

The cases for the study have been selected from the patients having type II Diabetes mellitus attending ophthalmic OPD and IPD and patients referred from,Either diabetic clinic or medicine clinic attached to KLES DR.Prabhakar kore hospital And MRC , Belgaum

INCLUSION CRITERIA

Participants diagnosed to have type II diabetes mellitus with retinopathy changes are included in the study.

EXCLUSION CRITERIA

1) Patients with any renal infection (i.e.non-diabetic nephropathy)

2) Pregnant women

3) Patients with accelerated hypertension

4) Patients with hazy ocular media

5) Participants not willing to give consent

All the subjects satisfying the inclusion criteria were enrolled in the study after obtaining informed consent .All enrolled subjects were asked preliminary questionnaires including age , education ,economic status , duration of diabetes ,treatment history . Then ocular examination including visual acuity (by means of snellens charts) fundoscopy (utilizing slit lamp, direct and indirect ophthalmoscope)

was done and subjects were classified according to ETDRS classification into mild ,moderate, severe and very severe non-proliferative diabetic retinopathy and proliferative diabetic retinopathy.

Subsequently albumin levels was determined by MICROALBUMIN-TURBILATEX METHOD on single urine sample collected in laboratory in KLES DR. Prabhakar kore hospital. If it is less than 30mg/l patients is classified as normoalbuminuric . Between 30-299mg/l were indicative of microalbuminuria and above 300mg/l were indicative of macroalbuminuria.

Statistical methods :

Fisher Exact test was used to determine the relationship between Albuminuria and severity of diabetic retinopathy in patients of type 2 DM.

Chi Square test was used to determine the relationship between severity of diabetic retinopathy with duration of diabetes and hypertension

All the calculations were done using SPSS statistical data package editor, version 12.0.

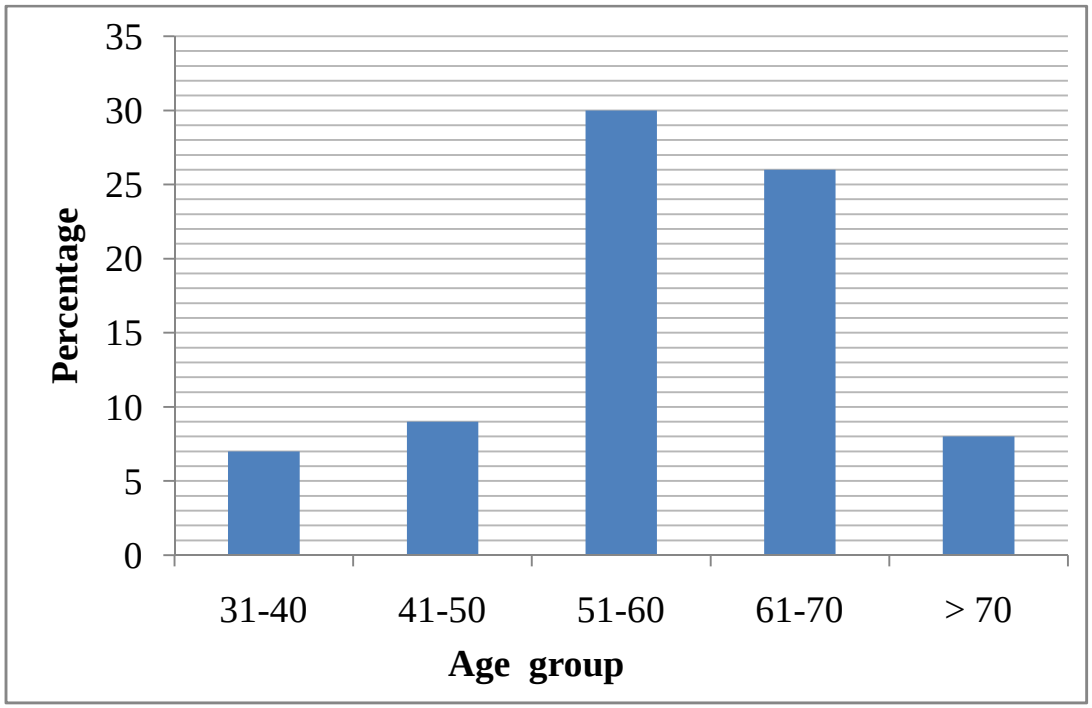
RESULTS

Data Analysis was done based on various statistical tests like descriptive statistics, contingency coefficients and Chi-square test and the results were as follows:

Table – 1 : Age Distribution

Age Group (in years)	Number of patients	Percentage
31-40	7	7%
41-50	9	9%
51-60	30	30%
61-70	26	26%
More than 70	8	8 %
Total	100	100 %

Figure 1 : Graph showing Age Distribution

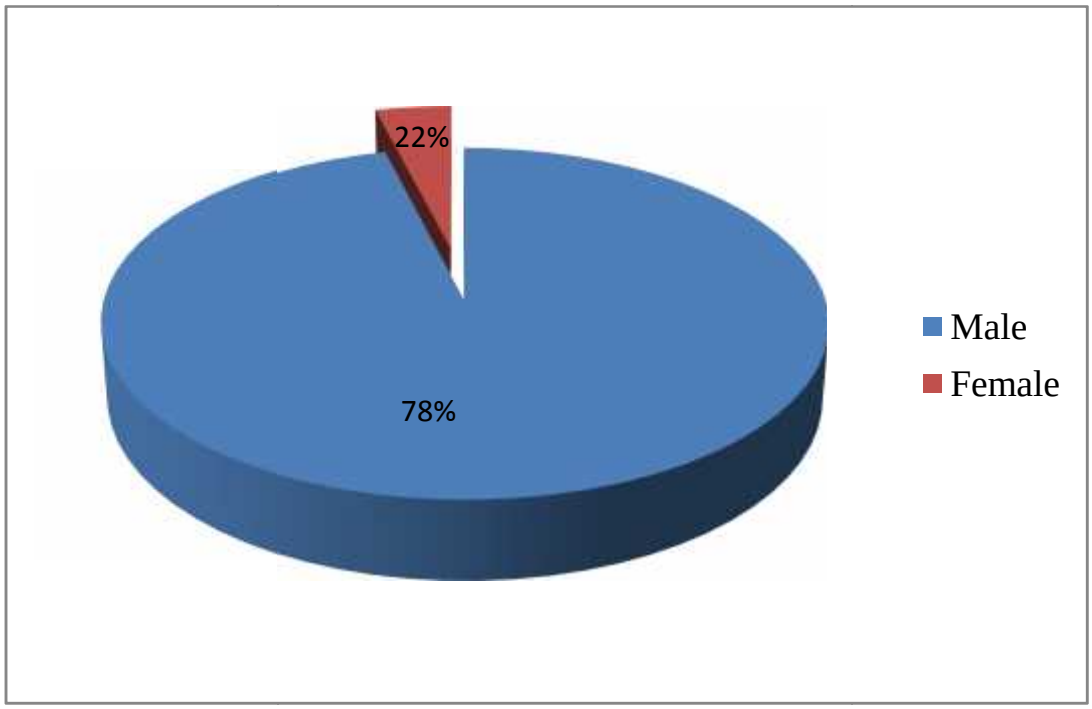


All patients were between 30 and 80 years of age.there were 7 patients in the 31-40 years group, 9 patients in the 41-50 years age group, 30 patients in 51-60 years age group, 26 patients in the 61-70 years age group and 8 patients in > 70 years age group.

Table – 2 : Sex Distribution

Sex	Number of patients	Percentage
Male	78	78%
Female	22	22%
Total	100	100 %

Figure 2 : Diagram showing sex distribution

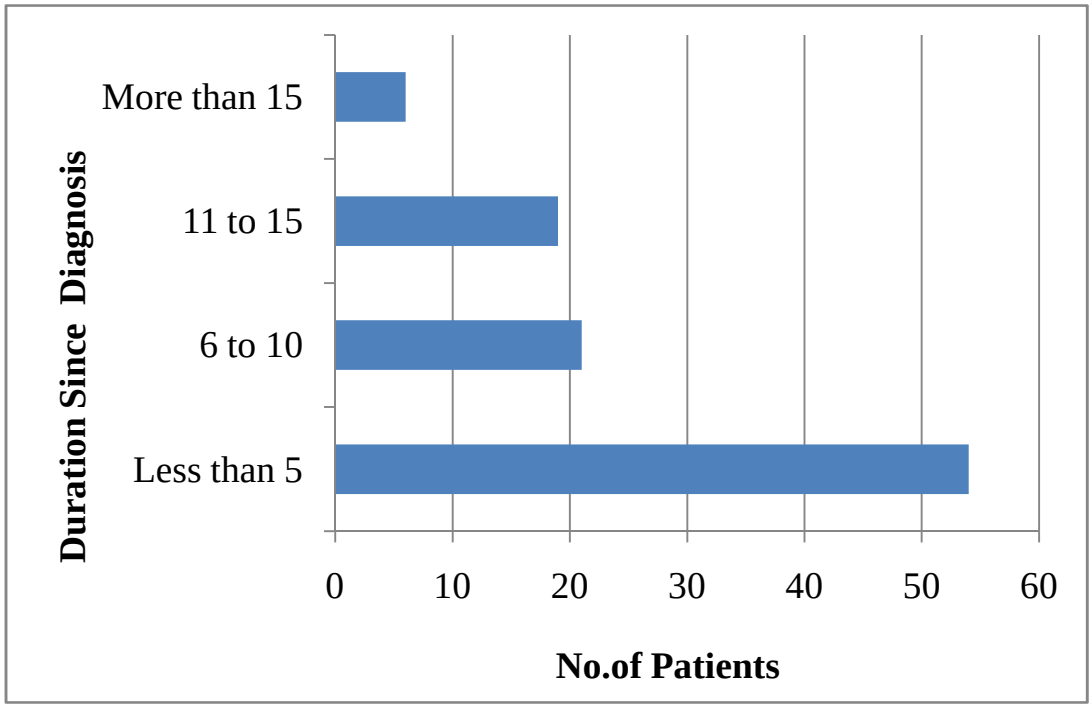


Out of the 100 patients, 78 (78%) were male and 22 (22%) female.

Table – 3 : Duration since diagnosis of diabetes mellitus

Duration since diagnosis (in years)	Number of patients	Percentage
Less than or equal to 5	54	54.0 %
6-10	21	21.0 %
11-15	19	19.0 %
More than 15	6	6.0 %
Total	100	100 %

Figure 3 : Graph showing distribution of diabetic age

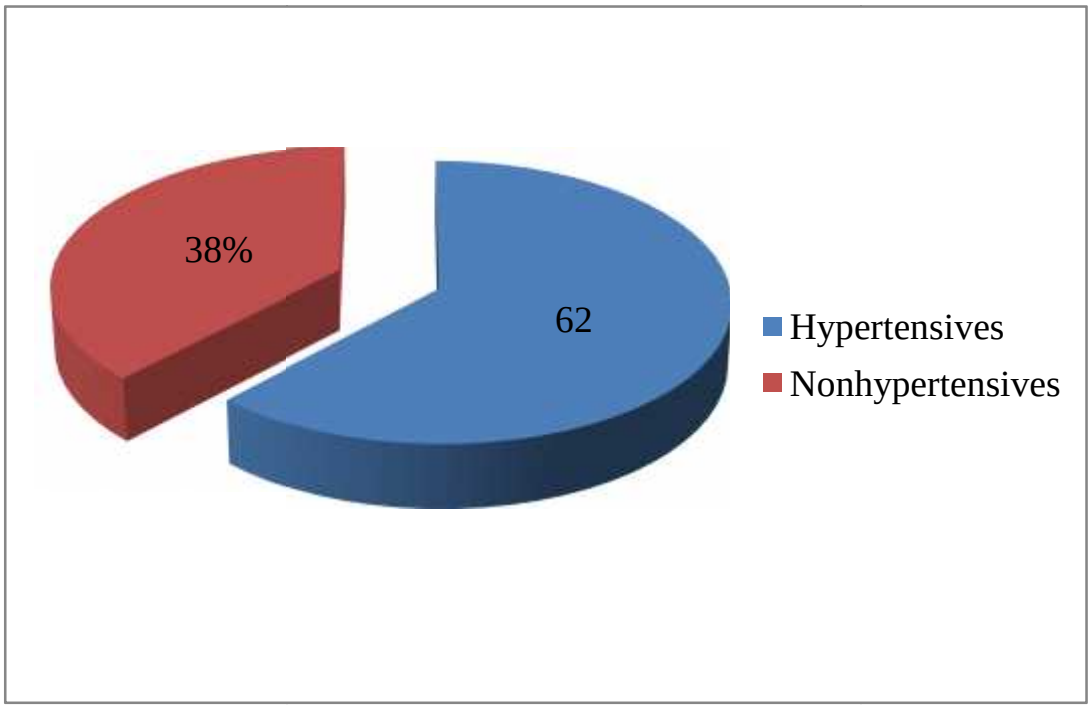


In the study patients, the duration since diagnosis of diabetes mellitus (diabetic age) ranged from 1-25 years. Among the 100 patients, 54 patients had diabetes mellitus for less than 5 years, 21 patients had it since 6-10 years, 19 patients had disease since 11-15 years and 6 patients had the condition for more than 15 years.

Table – 4 : Distribution of systemic hypertension

Association with systemic hypertension	Number of patients	Percentage
Hypertensives	62	62%
Non-Hypertensive	38	38%
Total	100	100 %

Figure 4 : Graph showing distribution of systemic hypertension

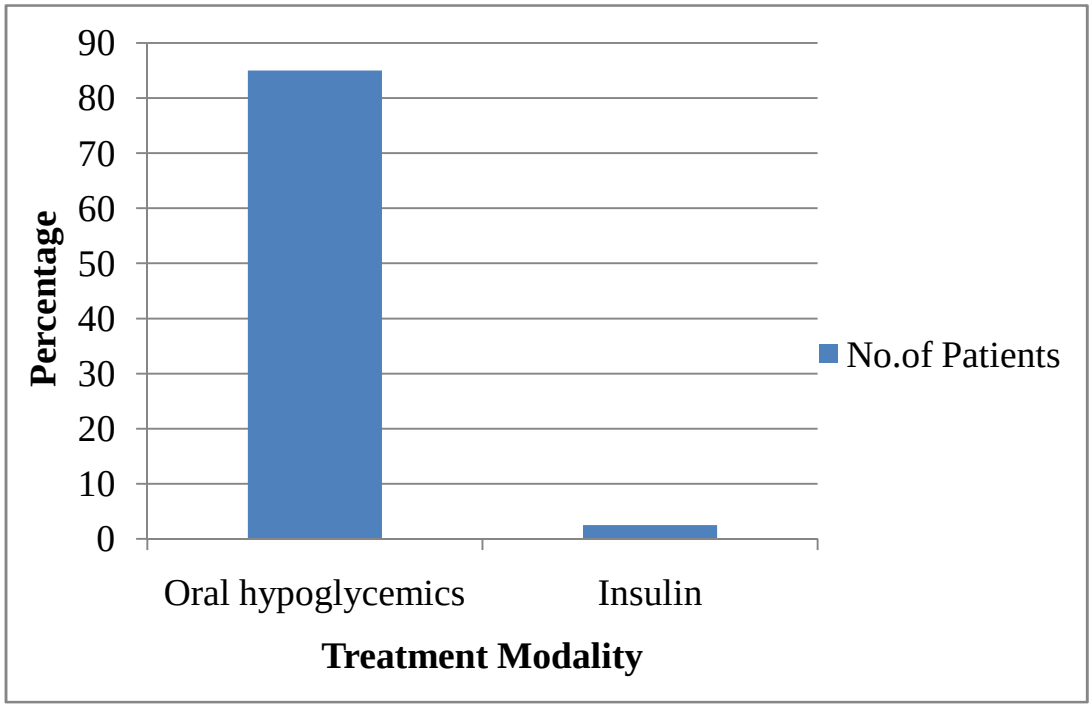


Among the 100 patients, 62 (62.0%) patients were hypertensives and 38(38.0%) patients were non-hypertensives.

Table – 5 : Distribution of the Treatment modality

Treatment Modality	Number of patients	Percentage
Oral Hypoglycemics	85	85 %
Insulin	15	15 %
Total	100	100 %

Figure 5: Graph showing distribution of Treatment modality

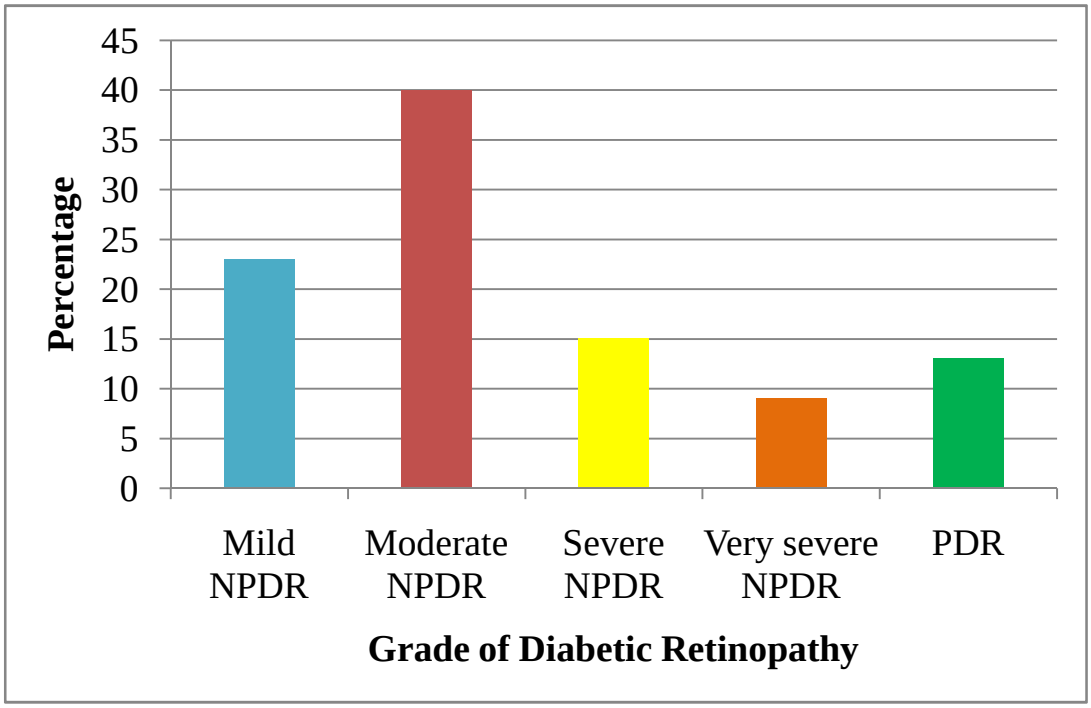


Among the 100 patients, 85 (85.0%) patients were on oral hypoglycemic. 15 (15.0%) patients were on insulin .

Table – 6 : Distribution of severity of diabetic retinopathy

Grade of Diabetic Retinopathy	Number of patients	Percentage
Mild NPDR	23	23.0 %
Moderate NPDR	40	40.0 %
Severe NPDR	15	15.0%
Very severe NPDR	9	9.0 %
PDR	13	13.0 %
Total	100	100 %

Figure 6 : Graph showing distribution of severity of diabetic retinopathy

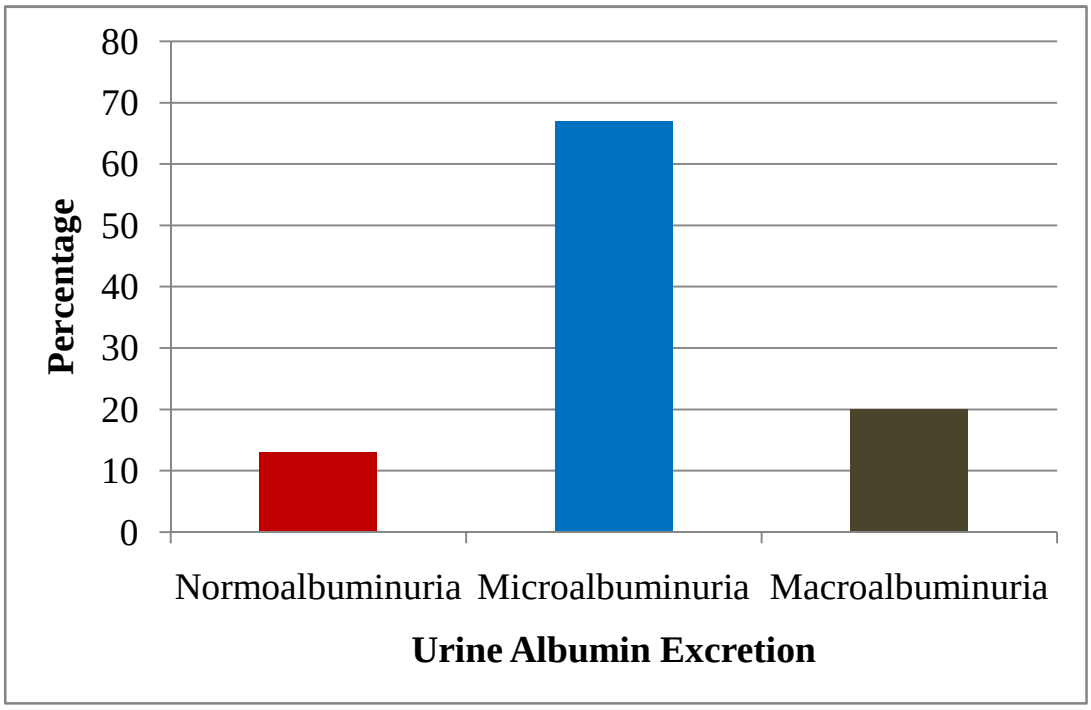


Among the 100 patients, 23 (23.0%) patients had mild NPDR, 40 (40.0%) patients had moderate NPDR, 15 (15.0%) patients had severe NPDR, 9 (9.0%) patients had very severe NPDR and 13(13.0%) patients had PDR.

Table – 7: Distribution of urine albumin excretion

Urine Albumin Excretion	Number of patients	Percentage
Normoalbuminuria	13	13.0 %
Microalbuminuria	67	67.0 %
Macroalbuminuria	20	20.0 %
Total	100	100 %

Figure 7: Graph showing Distribution of urine albumin excretion

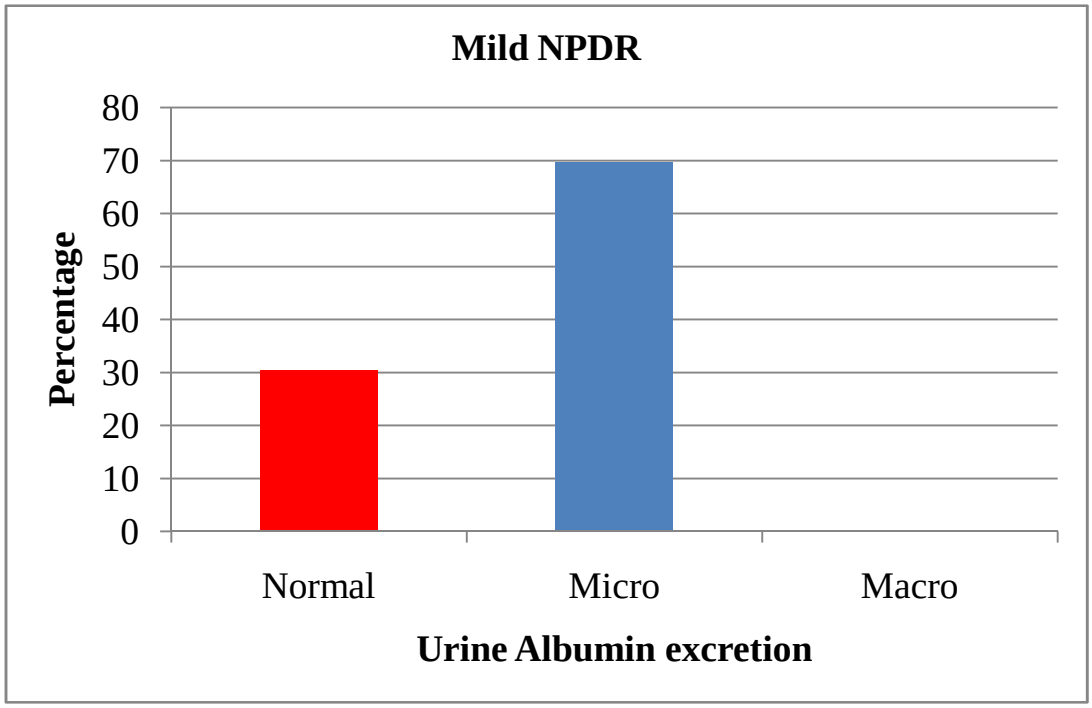


Among the 100 patients, 13 (13.0%) had normoalbuminuria, 67 (67.0%) had microalbuminuria and 16 (16.0%) patients had macroalbuminuria.

Table – 8 : Distribution of urine albumin excretion in the Mild NPDR Group

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
Mild NPDR	No.	7	16	0	23
	%	30.4%	69.6.%	.0%	100.0%

Figure 8 : Graph showing distribution of urine albumin excretion in the Mild NPDR Group

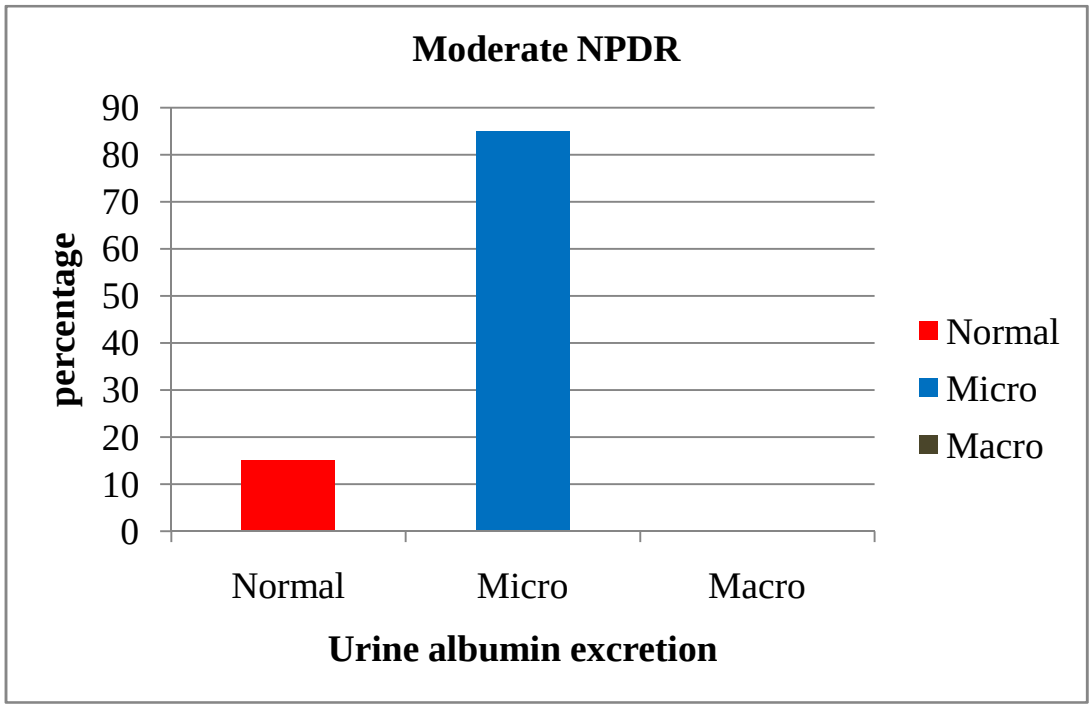


Among the 23 mild NPDR patients, 7 patients (30.4%) had normoalbuminuria and 16 patients (69.6%) had microalbuminuria.

Table – 9: Distribution of urine albumin excretion in the Moderate NPDR Group

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
Moderate NPDR	No.	6	34	0	40
	%	15.0%	85.0%	.0%	100.0%

Figure 9: Graph Showing Distribution of urine albumin excretion in the Moderate NPDR Group

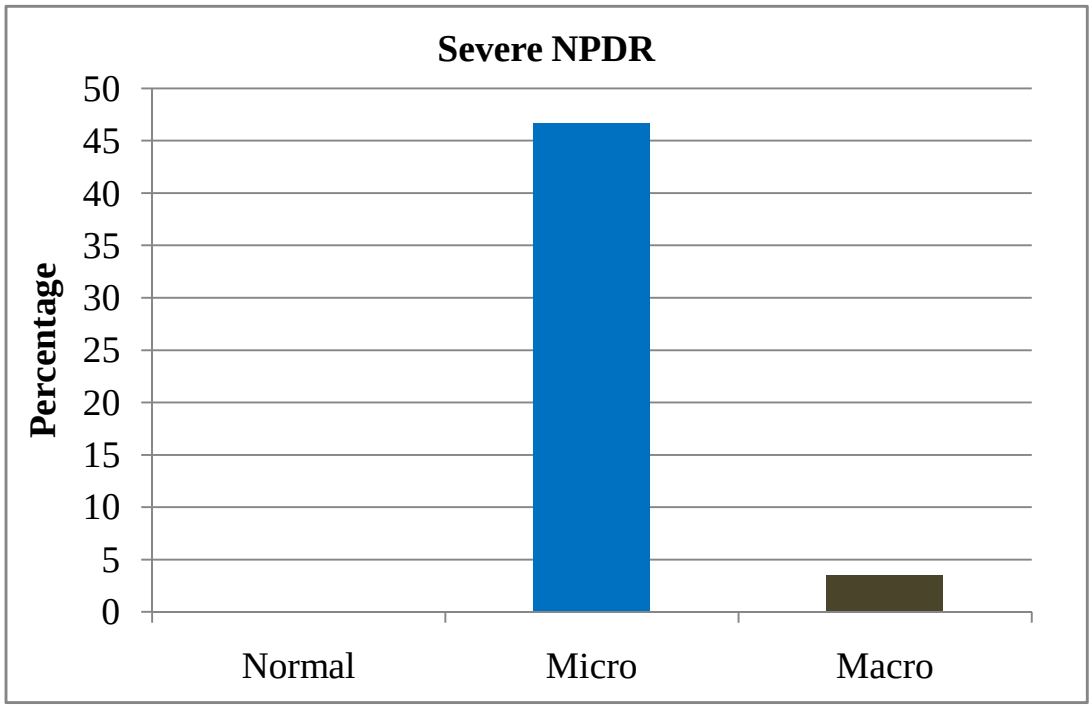


In 40 patients with moderate NPDR, 6 patients (6%) had normoalbuminuria and 34 patients (85.0%) had microalbuminuria.

Table – 10: Distribution of urine albumin excretion in the Severe NPDR Grou

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
Severe NPDR	No.	0	7	8	15
	%	.0%	46.7%	53.3%	100.0%

Figure 10: Graph showing distribution of urine albumin excretion in the severe NPDR Group

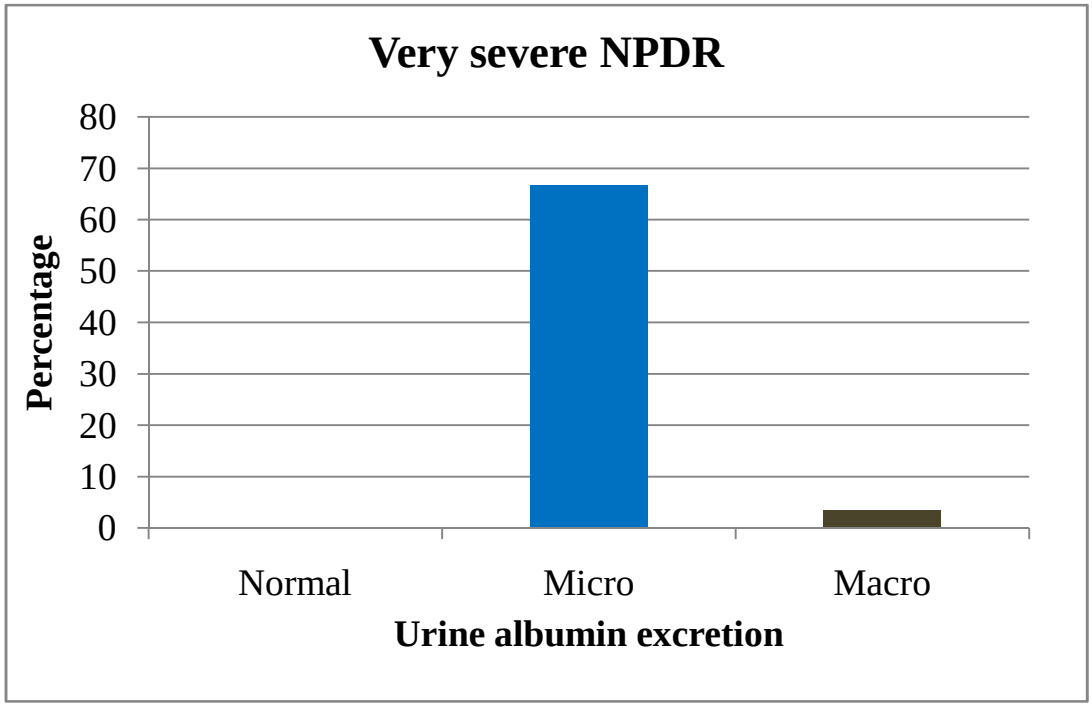


In the 4 severe NPDR patients, 7 patients (46.7%) had microalbuminuria and 8 patients (53.3%) had macroalbuminuria.

Table – 11: Distribution of urine albumin excretion in the Very severe NPDR Group

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
Very severe NPDR	No.	0	6	3	9
	%	.0%	66.7%	33.3%	100.0%

Figure 11: Graph showing distribution of urine albumin excretion in the very severe NPDR Group

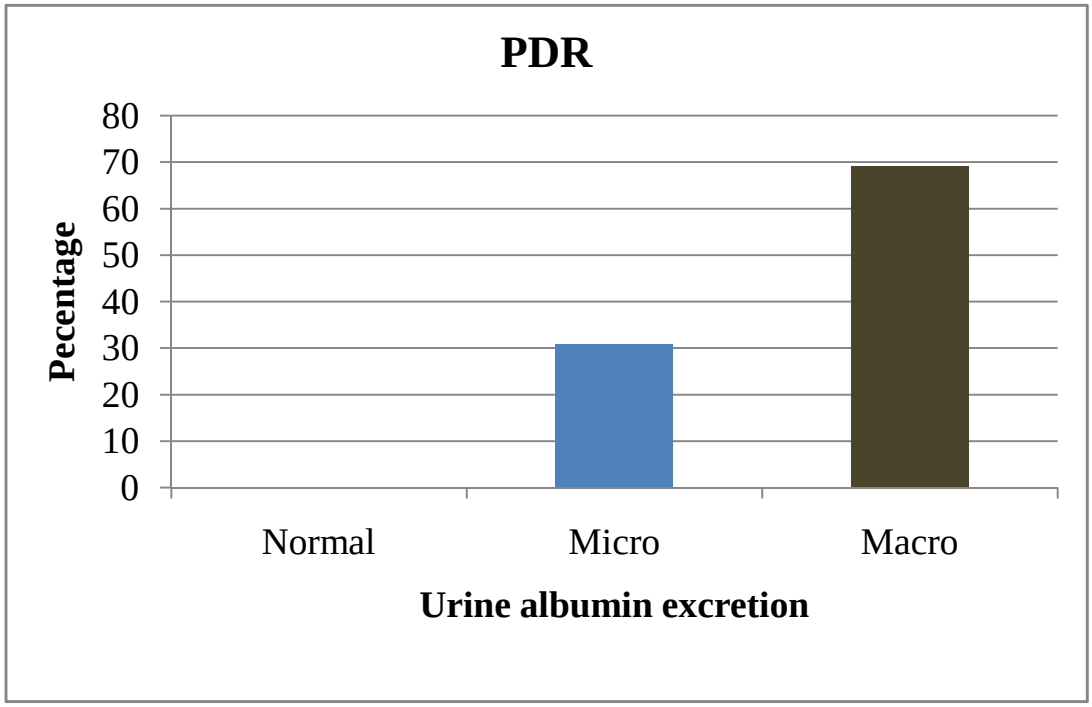


In the very severe NPDR, 6 patients (66.7%) had microalbuminuria and 3 patients (33.3%)had macroalbuminuria.

Table – 12: Distribution of urine albumin excretion in the PDR Group

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
PDR	No.	0	4	9	13
	%	.0%	30.8%	69.2%	100.0%

Figure 12: Graph showing distribution of urine albumin excretion in the PDR Group

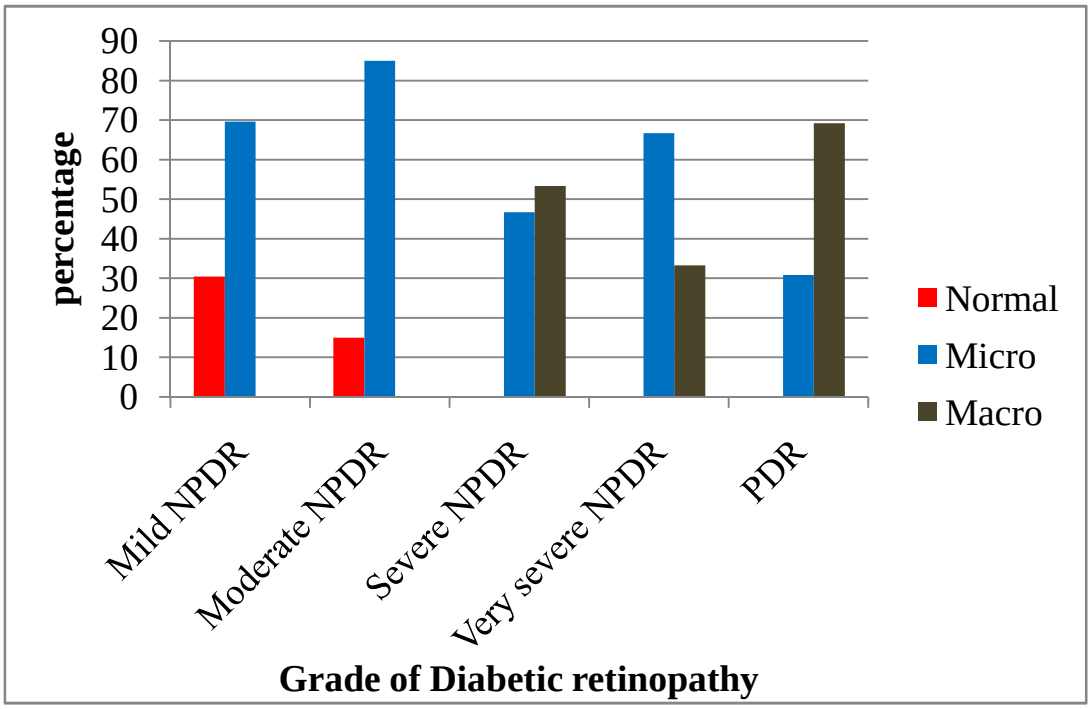


In 13 PDR patients, 4 patients (30.8%) had microalbuminuria and 9 patients (69.2%) had macroalbuminuria.

Table – 13: Summary of Association between grade of diabetic retinopathy and urine albumin excretion.

Grade of Diabetic Retinopathy		Urine albumin Excretion			Total
		Normal	Micro	Macro	
Mild NPDR	No.	7	16	0	23
	%	30.4%	69.6%	.0%	100.0%
Moderate NPDR	No.	6	34	0	40
	%	15.0%	85.0%	.0%	100.0%
Severe NPDR	No.	0	7	8	15
	%	.0%	46.7%	53.3%	100.0%
Very severe NPDR	No.	0	6	3	9
	%	.0%	66.7%	33.3%	100.0%
PDR	No.	0	4	9	13
	%	.0%	30.8%	69.2%	100.0%
Total	No.	13	67	20	100
	%	13.0%	67..0%	20.0%	100.0%

Figure 13: Graph showing summary of Association between grade of diabetic retinopathy and urine albumin excretion.



DISCUSSION

In the present study, 100 patients having type II diabetes mellitus with diabetic retinopathy of age group ranging from 30 to 80 years were studied and were subcategorised depending on the severity / grade of retinopathy.

It has been shown in other studies that in most diabetics, diabetic retinopathy develops before clinical nephropathy. It has also been shown that microalbuminuria is a risk indicator for development of proliferative retinopathy[21]. Urine albumin excretion [UAE] was estimated in all the patients in the present study. UAE was then correlated with the status of ocular fundus.

In the present study, the duration since diagnosis of diabetes (diabetic age) ranged from 1 – 25 years. Patients with diabetic age more than 10 years accounted to 25.0% in the present study. The association between grade of diabetic retinopathy and duration since diagnosis was significant ($p = 0.001$). In the study conducted by Savage et al, there was an independent association between the severity of diabetic retinopathy and the duration of diabetes greater than 10 years ($p=0.0001$)[59]. In the study conducted by Kalk et al, it was found that among Africans, severity of diabetic retinopathy was significantly associated with the duration of diabetes ($p<0.0001$)[25]. These two studies are in agreement with the present study. However, in the study conducted by Serter et al[21], no significant correlation between the duration of diabetes and retinopathy was found unlike the present study.

In the present study, the distribution of cases with respect to gender was uneven with a male to female ratio [M : F] of 78 : 22. In the study conducted by

Savage et al[59] the M : F ratio was 61 : 39. In the study conducted by Parving et al[60] the M : F ratio was 62 : 38. These two studies are in agreement with this study.

The age group in the present study ranged from 30 – 80 years. In the study conducted by Serter et al the patients were in the age group of 35 – 55 years[21]. Hirvela et al conducted the study in which the age group ranged between 30 – 70 years. In the study conducted by Savage et al the mean age of the cases was 58.8 ± 0.8 years[59]. The present study is in concordance with the other studies with respect to the age distribution of cases. The difference with respect to the sex distribution was not statistically significant.

Hypertension commonly co-exists in diabetics and is considered an important confounding factor in the vascular complications of diabetes. In the present study, 62.0% of the cases were hypertensives and the association between severity of diabetic retinopathy and the co existence of hypertension was not significant .

While in the study conducted by Kalk, there was a significant association of diabetic retinopathy with high systolic BP ($p = 0.03$)[25]. Susan et al reported a significant association of diabetic retinopathy with high systolic BP ($p = 0.0073$) and diastolic BP ($p = 0.0141$)[59]. The present study does not support the observation that diabetic retinopathy commonly coexists with hypertension.

In the present study, In the mild NPDR group, 30.4% of the cases had normoalbuminuria and 69.6% of the cases had microalbuminuria. In the moderate NPDR group, 15.0% of the cases had normoalbuminuria and 85.0% of the cases had microalbuminuria. None of the patients in the above mentioned two groups had

macroalbuminuria. However, both the mild NPDR and moderate NPDR group had a large proportion of cases with microalbuminuria.

In the severe NPDR group, one half of the cases had microalbuminuria while the other half had macroalbuminuria. In the very severe NPDR group, 66.7% of the cases had microalbuminuria and 33.3% of the cases had macroalbuminuria. In the PDR group, only 30.8 % of the cases had microalbuminuria and the rest 69.2% of the cases had macroalbuminuria. None of the patients in the above mentioned three groups had normal albumin excretion. However, it can be observed that macroalbuminuria is seen in all the three groups. Also, major proportion of the PDR cases had macroalbuminuria.

Nielsen et al observed a statistically significant correlation between the urinary excretion levels of albumin and the degree of retinopathy. The study reported that the prevalence of diabetic retinopathy was more with macroalbuminuria (58%). The study also reported that the prevalence of retinopathy in cases normoalbuminuria was 12% while in the microalbuminuria group, 28% of the cases had diabetic retinopathy[23]. The findings of the above mentioned studies are in agreement with the present study .

Parvanova et al demonstrated that relative to patients without diabetic retinopathy, those with proliferative diabetic retinopathy had significant association with albuminuria ($p < 0.05$)[26]. In the study conducted by Parving et al, it was observed that there is a positive correlation between degree of retinopathy and proteinuria in type II diabetes mellitus. It also demonstrated that any degree of diabetic retinopathy was associated with more proteinuria, higher risk of end stage renal disease and death[60].

In the present study, majority of the patients with non proliferative diabetic retinopathy had microalbuminuria. Most of the patients who had proliferative retinopathy (69.2%) were found to have macroalbuminuria while the remaining 30.8% had microalbuminuria.

As in various other studies, hypertension is an important confounding factor in the study with respect to both diabetic retinopathy and urine albumin excretion. Also, the referral of uncontrolled diabetics to the tertiary centre for further management allows the possibility of selection bias to creep into the study. This gives future scope for a population based study rather than a hospital based study.

CONCLUSION

Despite the high prevalence of type II diabetes mellitus, the prevalence of diabetic retinopathy is relatively low, particularly the advanced grades of retinopathy. With the projected aging of the population in India, the prevalence of type II diabetes mellitus and correspondingly the prevalence of diabetic retinopathy will certainly increase in the future.

The natural history of type II diabetes mellitus is difficult to delineate as the disease could have been present for many years before it is diagnosed. Thus early predictors of diabetic retinopathy may aid in the prevention, delaying the progression and management of the disease. Assessment of urine albumin excretion [UAE] has been considered one of the probable early predictors of diabetic retinopathy. However, the association between diabetic retinopathy and urine albumin excretion in various studies have been discordant.

This study demonstrates that macroalbuminuria was associated only with those patients who had either severe NPDR, very severe NPDR or PDR. It also shows that none of these three grades of diabetic retinopathy had normal levels of urine albumin excretion. Microalbuminuria was a finding associated with all grades of retinopathy with skewing towards the lower grades of diabetic retinopathy .Normal albumin levels in urine was an occasional finding in mild and moderate NPDR and was not at all found in the higher grades of diabetic retinopathy.

There is a strong association of UAE with proliferative diabetic retinopathy. However, the association between lower grades of diabetic retinopathy and UAE is not very well defined.

SUMMARY

This cross sectional study was done in the department of Ophthalmology, KLES DR.Prabhakar kore hospital. A total of 100 patients with diabetic retinopathy were studied.

A major proportion of the cases were in the 51 to 60 years age group. Out of the 100 patients, 78 were males and 22 were females. Estimation of albumin levels in the urine was done by the Microalbumin turbilatex method. The patients were categorized into three groups based on the levels of albumin excretion [normoalbuminuric, microalbuminuric, macroalbuminuric]. A thorough fundus examination and documentation was done in all cases. Subjects were divided into the grades of diabetic retinopathy according to ETDRS classification.

None of the patients with normal albumin levels had a grade of diabetic retinopathy more than moderate NPDR. None of the patients with macroalbuminuria had a grade less than severe NPDR. Microalbuminuria was however distributed in all grades of diabetic retinopathy with skewing towards less severe grades.

Whether microalbuminuria can be used as an early predictor of diabetic retinopathy remains inconclusive. However, it was found that the occurrence of macroalbuminuria is significantly higher in severe NPDR, very severe NPDR and PDR. Thus, this study reinforces the observation that there is a strong association between macroalbuminuria and advanced grades of diabetic retinopathy.

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ANNEXURE – I

ASSOCIATION OF ALBUMIN LEVELS IN URINE WITH SEVERITY OF DIABETIC RETINOPATHY IN TYPE II DIABETES MELLITUS –A ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL.

DATA COLLECTION INSTRUMENT

I.D.NO

NAME
(First name)(Surname)

AGE (in yrs.)

SEX 1=Male 2 = Female

RELIGION
1-Hindu 2-Muslim 3-Christian 4-Others

ADDRESS

OPD NO.

IPD NO.

PROVISIONAL DIAGNOSIS: _____

1- Type I diabetes mellitus.

2- Type II diabetes mellitus.

3- Maturity onset diabetes of the young (MODY).

INCLUSION CRITERIA

1- met 2- not met

Criteria of inclusion : diagnosed to have type II diabetes mellitus with retinopathy changes in the fundus.

INFORMED CONSENT

1-taken 2- not taken ☐

Approval of the guide: _____

CHIEF COMPLAINTS:

1= YES 2= NO

	RE	**LE**	**BE**
1.) Diminution of vision	☐	☐	☐
2.) Pain	☐	☐	☐
3.) Flashes	☐	☐	☐
4.) Floaters	☐	☐	☐
5.) Metamorphopsia	☐	☐	☐
6.) Polyuria	☐	☐	☐
7.) Polydypsia	☐	☐	☐
8.) Polyphagia	☐	☐	☐
9.) ANY OTHER COMPLAINTS:	☐	☐	☐

IF YES:_____

PAST HISTORY:

1= YES, 2= NO a= >0-5 yrs b= >5-10 yrs c= >10-15 yrs d= >15-20 yrs e= >20 yrs.

10.) H/O WEARING GLASSES ☐ If yes, duration ☐

11.) H/O HYPERTENSION ☐ If yes, duration ☐

12.) H/O DIABETES MELLITUS ☐ If yes, duration ☐

13.) H/O ANY OCULAR SURGERY ☐ If 1, specify _____

14.) H/O AUTOIMMUNE DISEASE ☐

15.) H/O DRUG ALLERGY ☐ If yes, specify _____

16.) ANY OTHER ☐ IF YES: _____

17.) FAMILY HISTORY:

1-Significant 2- Not significant ☐

18.) Life style:

1= YES 2 = NO ☐

Active ☐

Sedentary ☐

Personal history:

1-yes 2-no a= >0-5 yrs b= >5-10 yrs c= >10-15 yrs d= >15-20 yrs e= >20 yrs.

19.) Alcohol ☐ If yes, duration ☐

20.) Smoking ☐ If yes, duration ☐

21.) Diet : Veg/Mixed ☐ (1 = veg. , 2 = mixed) ☐

DIABETIC HISTORY :

22.) Age of onset of diabetes _____years

23.) Duration of diabetes _____years

24.) Any significant events _____years

TREATMENT HISTORY : 1=yes 2=no

a= >0-5 yrs b= >5-10 yrs c= >10-15 yrs d= >15-20 yrs e= >20 yrs.

25.) Oral hypoglycaemic: Single ☐ If yes, duration ☐

Multiple ☐ If yes, duration ☐

Groups of drug used: a. 1st generation sulfonylureas

b. 1st generation sulfonylureas

c. Biguanides

d. Miglitinide analogues.

e. Thiazolidinediones.

f. Glucosidase inhibitor.

g. Others. Specify : _____

26.) Insulin injections : If yes, duration

27.) Laser : RE : If yes, duration

LE : If yes, duration

28.) Any other :

GENERAL PHYSICAL EXAMINATION:

29.) PULSE RATE(per min.) | | | |

30.) RESPIRATORY RATE(per min.) | | | |

BLOOD PRESSURE(in mm Hg)

31.) Systolic | | | |

32.) Diastolic | | | |

33.) TEMPERATURE(in deg F) | | | |

SYSTEMIC EXAMINATION : 1= Normal 2= Abnormal

34.) CVS: ☐ If abnormal_____

35.) R/S: ☐ If abnormal_____

36.) P/A: ☐ If abnormal_____

37.) CNS: ☐ If abnormal _____

38.) Renal: ☐ If abnormal_____

39.) Skin: ☐ If abnormal_____

40.) Foot: ☐ If abnormal _____

OCULAR EXAMINATION

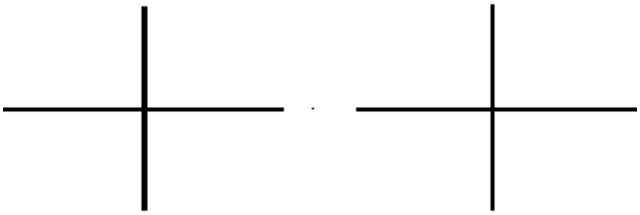
41.) HEAD POSTURE: ☐ 1= ERECT 2=TILTED

42.) VISUAL AXIS: ☐ 1=PARALLEL 2= DEVIATED

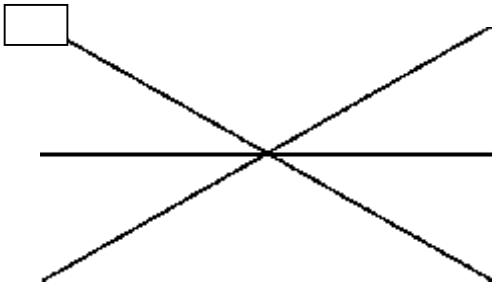
43.) FACIAL SYMMETRY: ☐ 1=SYMMETRICAL 2=DEVIATED

EXTRAOCULAR MOVEMENTS: ☐ NORMAL 2=RESTRICTED

44.) UNIOCULAR: RE ☐ LE ☐



45.) BINOCULAR



Ocular examination : **Right eye** **Left eye**

46.) <u>VISUAL ACUITY</u> 1: 6/6 – 6/12 2: 6/18 – 6/36 3:≤ 6/60 Unaided vision : With pinhole : With spectacles :	☐	☐
47.) <u>ADNEXA</u> 1- Normal 2- Abnormal If 2,specify_____	☐	☐
48.) <u>Lids</u> 1-Normal 2-Abnormal If 2,specify_____	☐	☐
49.) **<u>CONJUNCTIVA</u>** 1 – Normal 2- Congested 3 – Other If 3,Specify : _____	☐	☐
50.) <u>Cornea</u> 1- Clear 2- Hazy 3- other If 3,Specify : _____	☐	☐
<u>ANTERIOR CHAMBER:</u> 51.) **DEPTH** 1- Normal 2- Shallow 3- Deep	☐	☐
52.) **<u>IRIS:</u>** COLOUR AND PATTERN 1- Normal 2- Atrophic Patches	☐	☐

53.) **PUPIL:** SIZE 1-normal 2- Constricted 3- Dilated	☐	☐
54.) **REACTION:** DIRECT INDIRECT 1- Present 2- Absent 3- Sluggish	☐ ☐	☐ ☐
55.) PUPILLARY MARGIN 1-Normal 2- Abnormal If 2,specify_____	☐	☐
56.) POSTERIOR SYNECHIAE 1- Present 2- Absent	☐	☐
57.) ACCOMODATION 1-Present 2- Absent	☐	☐
58.) LENS: 1- Clear 2- Cataractous 3-Pseudophakia 4-Aphakia.	☐ ☐	☐ ☐
59.) If Cataractous: 1- Immature 2- Mature 3- Hypermature	☐ ☐	☐

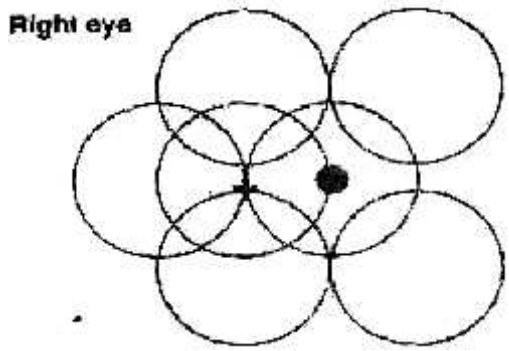
Observations :

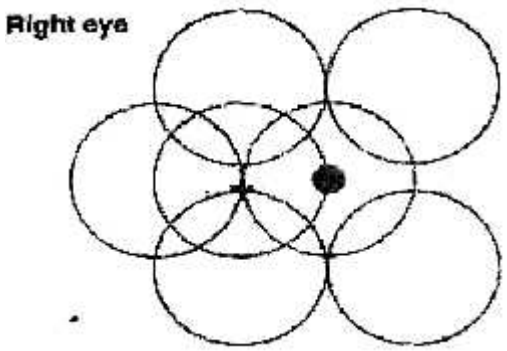
60.) Urine Albumin		
61.) FBS		
Fundus examination	**OD**	**OS**
62.) Glow 1=present, 2=faint, 3=absent		
63.) Media 1=clear, 2=hazy Corneal Lenticular Vitreous		
64.) Disc 1= normal 2 = Pallor 3 = NVD other, specify_____		
65.) Cup : disc ratio 1=normal (0.3) 2=abnormal If 2,specify_____		
66.) Vessels 1=normal 2 = Venous Beading 3 = Venous dilatation 4 = Venous looping 5 = Venous tortuosity Other, specify_____		
67.) Background 1=normal, 2=abnormal If 2,specify_____		

68.) **Haemorrhages** 1=present , 2=absent If 1 :- (specify quadrants in brackets) (a) = superionasal (b) = superiotemporal (c) = inferionasal (d) = inferiotemporal		
69.) Types of h'rge 1=Dot haemorrhages 2=Blot haemorrhages 3=Dark blot haemorrhages 4=Flame shaped haemorrhages 5=pre-retinal haemorrhages 6=Vitreous haemorrhages		
70.) Hard exudates 1=present , 2=absent If 1, specify quadrants as above.		
71.) A= NVE B= collaterals C= IRMAs 1 = present, 2 = absent. If 1, specify quadrants as above.		
72.) Soft exudates. 1 = present, 2 = absent. If 1, specify quadrants as above.		
73.) Fibrovascular proliferation: 1 = present, 2 = absent. If 1, specify quadrants as above.		
74.) **Others**		

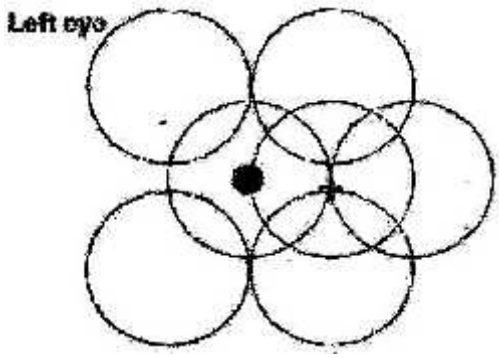
75.) Macula 1= normal 2 = microaneurysms 3 = haemorrhare 4 = hard exudates 5 = thickening in 500um or less from center of macula. 6 = hard exudates in 500um or less from center of macula. 7 = thickening >/= 1 disc area, any portion of which is </= 1 disc diameter from center of macula. Others, specify _____		

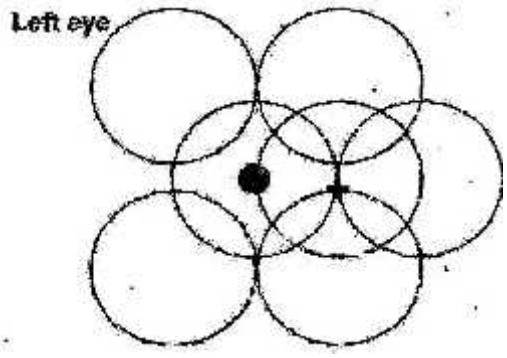
Right eye:Fundus diagram FFA diagram





Left eye: Fundus diagram FFA diagram





Funduscopic Diagnosis :

Angiographic diagnosis :

ANNEXURE – II

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Ms_____

You are invited to participate in our research study titled **"ASSOCIATION OF ALBUMIN LEVELS IN URINE WITH SEVERITY OF DIABETIC RETINOPATHY IN TYPE II DIABETES MELLITUS –A ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL."** conducted by Dr. _____, Post Graduate in M.S. Ophthalmology under the guidance of Dr. _____, Professor in the Department of Ophthalmology, J .N. Medical College, Belgaum.

Respected Sir/Madam we request you to enroll yourself to participate in our study as you are eligible for participating in the study. Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

Purpose of the Study: The purpose of research is TO DETERMINE THE CORRELATION OF ALBUMIN LEVELS IN URINE WITH SEVERITY OF DIABETIC RETINOPATHY IN TYPE II DIABETES MELLITUS.

Procedure Involved : If you agree to enroll yourself in this study, I will ask your present, past and family history. You will be clinically examined and relevant investigations will be accessed. Then you will be subjected to direct and indirect ophthalmoscopy, slit lamp biomicroscopy, fundus photography and fundus fluorescein angiography if required. The hence obtained data will be monitored, documented and reproduced as required.

Risks and Benefits : There are no major risks involved in the above mentioned procedures however some discomfort may occur, for which all precautions will be taken.

Your participation may benefit you and others suffering from the same ailment in future, by helping us learn more about the disease process and better treatment modalities.

Alternatives : If you are not willing to participate you will be treated according to the existing protocol & it will not affect your relationship with this hospital.

Costs for participating in this research: The participant will have to pay for the investigations which are the part of the existing management protocol for this ailment.

Privacy and Confidentiality: No information about you or information provided by you during the research will be disclosed to others without your written permission.

Authorization to Publish Results: When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity.

Compensation: In the event of injury related to the study, treatment will be made available through KLES Prabhakar Kore Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. The doctors and the staff will provide facilities and medical attention to you.

Questions:

If you have any questions about the research you may please contact:

1) Chief investigator, **Dr. _____**, P.G., Department of Ophthalmology, JNMC,Belgaum. Contact No. _____.
2) **Dr. _____**, Professor, Guide, Department of Ophthalmology, JNMC, Belgaum. Ph: _____
3) **Dr.A.S.GODHI** ,Principal, JNMC, Belgaum and chairman of Institutional Ethics Committee. Ph. 0831-2471350

CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

I, Mr./Ms./Mrs _____ voluntarily agree for the participation as a subject of this study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in my own vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name:_____

Signature of Witness:_____

Investigators Name: _____

Signature of Investigator : _____

Date : _____

Place : _____

KEY TO MASTER CHART

M= male

F=female.

IP=In-patient

OP=Out-patient

OHA=Oral hypoglycemic agents

M = mild NPDR.

Md = moderate NPDR.

RE = right eye.

LE= left eye

S = severe

VS= very severe

M= microanerysm

NV=neovascularization

FB=fibrovascular band

FBS= fasting blood sugar

P=present

A=absent

N=normal

AB=abnormal

NFL=nerve fibre layer

PLPR=projection of light and perception of rays

HMCF=hand movement close to face.

CF= counting finger

					HISTORY					OCULAR EXAMINATION																												INVESTIGATION		DIAGNOSIS	
	Patient Details				Ocular	betes mellitu		HTN		BCVA						FUNDUS																									
																							EXUDATES				HAEMORHAGE															
Sr.n	Name	Ip/op no.	Age	Sex	Visual los		OHA	HTN	B.P	Distantvision		Near		Ant segmen		Media		Disc		Arteries		Veins		Soft		Hard		Deep		NFL		Preretinal		Vitreous	MA	NV	FB	Macula		FBS	Albumin	BDR	Maculopathy
										RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					RE	LE				
1	ASR	2325956	60	M	P	7yr	P	P	138/9o	6I36	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	136mg/dl	100mg/l	M.NPDR	A
2	DSR	2353768	40	M	P	1yr	P	A	148/68	6I12	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	P	P	A	P	P	P	N	N	160mg/dl	410mg/l	PDR	A
3	SST	2429464	45	M	P	13yr	P	P	144/90	PL PR	HMCF	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	A	P	A	P	A	P	A	A	P	P	P	P	N	AB	149mg/dl	162mg/l	PDR	P
4	SRB	774933	63	M	P	14yr	P	A	150/80	6I36	6I60	N12	N12	N	N	N	N	N	N	N	N	N	N	A	A	A	P	P	P	P	P	A	A	A	P	A	A	N	AB	180mg/dl	60mg/l	MdNPDI	P
5	SSR	915632	45	M	P	4yr	P	A	136/90	6I24	6I24	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	A	A	P	P	A	A	N	N	180mg/dl	44mg/l	M.NPDR	A
6	ASP	2377249	46	F	P	5yr	P	A	14O/80	6I24	6I36	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	A	A	A	A	A	P	A	A	AB	AB	160mg/dl	56mg/l	MdNPDI	P
7	LPH	926956	79	M	P	5yr	P	A	143/86	6I18	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	A	P	P	A	A	A	A	A	P	A	A	N	N	142mg/dl	64mg/l	MdNPDI	A
8	AJK	2381249	48	M	P	3yr	P	P	140/80	6I9	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	P	A	A	A	A	A	P	A	A	N	N	142mg/dl	120mg/l	M.NPDR	A
9	BUD	2370735	49	M	P	3yr	P	P	144/88	6I18	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	A	A	A	A	A	P	A	A	N	N	126mg/dl	202mg/l	MdNPDI	A
10	SHC	1685051	53	M	P	11yr	P	P	148/68	6I18	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	P	P	P	A	A	A	P	P	A	A	A	P	A	A	N	N	188mg/dl	120mg/l	MdNPDI	A
11	CDH	1504342	70	M	P	10yr	P	P	146/80	6I18	6I24	N6	N6	N	N	N	N	N	N	N	N	N	N	P	P	P	P	P	P	A	A	A	A	A	P	A	A	AB	AB	220mg/dl	140mg/l	MdNPDI	P
12	SAM	2405195	60	M	P	17yr	P	P	130/80	6I18	HMCF	N6	>N36	N	N	N	N	N	N	N	N	N	N	P	A	A	A	A	A	A	P	A	A	P	P	A	A	N	N	168mg/dl	320mg/l	PDR	A
13	KJB	2151716	65	F	P	3yr	P	P	140/90	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	144mg/dl	100mg/l	MNPDR	A
14	**MIJ**	**512685**	**46**	**M**	**P**	**6yr**	**P**	**P**	**130/90**	**6I18**	**6I24**	**N6**	**N6**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**A**	**A**	**P**	**P**	**P**	**P**	**P**	**P**	**A**	**A**	**A**	**P**	**A**	**A**	**AB**	**AB**	**168mg/dl**	**241mg/l**	**MdNPDI**	**P**
15	SYB	570890	60	M	P	5yr	P	P	138/90	6I18	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	A	P	A	A	A	P	A	A	N	N	168mg/dl	120mg/l	MdNPDI	A
16	GGM	1855833	60	F	P	4yr	P	A	140/90	6I24	6I18	N12	N12	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	140mg/dl	124mg/l	MdNPDI	A
17	MKP	512560	55	M	P	4yr	P	P	120/70	6I24	6I36	N12	N12	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	AB	AB	144mg/dl	288mg/l	VSNPDI	P
18	SJP	2425091	52	M	P	7yr	P	P	140/70	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	AB	AB	168mg/dl	110mg/l	SNPDR	P
19	MAG	2428513	45	F	P	6yr	p	P	148/92	6I24	6I60	N12	N12	N	N	N	N	N	N	N	N	N	N	A	A	P	A	P	A	A	P	A	A	A	P	A	A	AB	AB	160mg/dl	339mg/l	SNPDR	P
20	STB	917849	55	M	P	5yr	P	P	130/70	6I24	6I36	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	A	P	P	A	A	A	A	A	P	A	A	AB	N	160mg/dl	114mg/l	MdNPDI	P
21	AGG	2409699	49	M	P	9yr	P	P	140/80	6I60	6I60	N12	N12	N	N	N	N	N	N	N	N	N	N	A	A	P	A	P	P	P	P	A	A	A	P	A	A	N	AB	124mg/dl	10mg/l	MdNPDI	P
22	SAA	484742	57	M	P	12yr	P	A	160/80	6I12	6I12	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	AB	AB	150mg/dl	500mg/l	VSNPDI	P
23	BIR	486847	72	M	P	4yr	P	P	136/88	6I9	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	P	P	P	P	A	A	A	A	P	P	A	P	A	A	N	N	134mg/dl	110mg/l	MNPDR	A
24	ERA	486473	66	M	P	14yr	P	P	140/90	6I12	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	P	P	A	A	A	P	A	A	N	N	188mg/dl	738mg/l	SNPDR	A
25	SPK	478278	41	M	A	11yr	P	P	130/60	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	P	A	P	P	P	P	P	P	A	A	A	P	A	A	N	N	144mg/dl	500mg/l	SNPDR	A
26	AKT	480433	38	M	A	6yr	P	P	138/60	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	133mg/dl	13mg/l	MNPDR	A
27	APS	488411	56	M	P	5yr	P	A	130/78	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	P	P	P	P	P	A	A	P	A	A	A	P	A	A	N	N	180mg/dl	260mg/l	SNPDR	A
28	BJP	492297	67	M	A	12yr	P	A	122/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	A	P	A	A	A	P	A	A	N	N	166mg/dl	15mg/l	MdNPDI	A
29	VDH	2585597	43	F	P	2Yr	P	A	120/78	6I60	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	A	A	A	N	N	144mg/dl	56mg/l	MNPDR	A
30	KPS	448176	65	M	P	7yr	P	A	128/78	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	N	N	240mg/dl	160mg/l	MdNPDI	A
31	YKB	1887365	62	M	A	5yr	P	P	130/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	188mg/dl	210mg/l	MNPDR	A
32	CGB	592849	54	M	A	5yr	P	A	124/60	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	124mg/dl	25mg/l	MNPDR	A
33	ASS	489073	59	F	A	9yr	P	P	148/88	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	208mg/dl	16mg/l	MNPDR	A
34	SDT	2176274	51	M	A	4yr	P	A	140/90	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	N	N	136mg/dl	85mg/l	MdNPDI	A
35	FHT	486423	55	M	P	14yr	P	P	148/64	6I12	6I12	N8	N8	N	N	N	N	N	N	N	N	N	N	P	P	P	P	A	A	P	P	A	A	A	P	A	A	N	N	200mg/dl	400mg/l	VSNPDI	A
36	RMP	2174232	59	M	A	4yr	P	P	130/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	139mg/dl	2mg/l	MdNPDI	A
37	KTS	2160513	42	M	A	3yr	P	A	140/90	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	181mg/dl	6mg/l	MdNPDI	A
38	AKS	495197	37	M	A	1yr	P	P	140/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	119mg/dl	20mg/l	MNPDR	A
39	DBD	484298	49	M	A	7yr	P	A	126/78	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	160mg/dl	5mg/l	MNPDR	A
40	RMP	2174232	59	F	A	3yr	P	P	146/82	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	110mg/dl	2mg/l	MNPDR	A
41	MDD	1240297	64	M	P	13yr	P	A	134/82	6I18	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	A	A	A	A	A	P	P	A	AB	AB	163mg/dl	210mg/l	PDR	P
42	RAS	2337128	65	M	P	19YR	P	P	140/90	6I24	CF-2M	N12	N36	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	P	A	P	A	P	A	P	AB	AB	136mg/dl	510mg/l	PDR	P
43	BAA	2326254	77	M	P	15YR	P	P	142/86	6I24	6I36	N8	N8	N	N	N	N	N	N	N	N	N	N	A	A	A	A	P	P	A	A	A	A	A	P	P	A	N	N	102mg/dl	340mg/l	PDR	A
44	AAD	2348504	50	M	A	5YR	P	P	148/90	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	AB	116mg/dl	180mg/l	MNPDR	P
45	BAM	1418757	38	M	P	8YR	P	A	130/86	6I18	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	P	A	A	A	A	A	A	A	A	A	A	A	A	A	AB	N	131mg/dl	110mg/l	SNPDR	P
46	SLA	265472	57	M	P	17YR	P	P	124/80	6I12	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	P	P	P	P	P	A	A	P	A	A	A	P	A	A	N	N	113mg/dl	500mg/l	VSNPDI	A
47	EHA	265975	66	F	P	19YR	P	A	130/80	6I12	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	P	P	P	P	A	A	A	A	A	A	A	P	A	A	AB	AB	240mg/dl	738mg/l	SNPDR	P
48	BIR	486847	72	M	A	10yr	P	A	130/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	96mg/dl	104mg/l	MNPDR	A
49	SBP	3283290	55	F	P	4yr	P	A	120/90	6I24	6I24	N12	N8	N	N	N	N	N	N	N	N	N	N	A	P	A	A	P	P	P	P	A	A	A	P	A	A	AB	AB	159mg/dl	320mg/l	SNPDR	P
50	KIA	452707	45	M	A	5YR	P	A	148/96	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	140mg/dl	15mg/l	MdNPDI	A

51	PIT	115139	68	M	P	5yr	P	P	130/60	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	P	P	A	A	A	A	P	P	A	A	A	P	A	A	N	N	242mg/dl	12mg/l	MdNPDI	A
52	SIG	1961679	60	M	P	15YR	P	A	134/72	6I18	6I24	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	N	N	101mg/dl	146mg/l	VSNPDI	A
53	NIT	101113	67	M	A	4yr	P	A	126/86	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	111mg/dl	140mg/l	MNPDR	A
54	CSS	495449	62	M	P	4yr	P	A	130/74	6I9	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	N	P	P	A	A	A	A	P	P	A	A	A	P	A	A	N	N	140mg/dl	1200mg/l	SNPDR	A
55	KSS	2160513	71	M	P	3yr	P	P	130/76	6I12	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	170mg/dl	6mg/l	MdNPDI	A
56	PGN	452412	68	M	A	8YR	P	A	13O/78	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	151mg/dl	12mg/l	MdNPDI	A
57	MBB	1819721	62	M	A	22Yr	P	P	140/90	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	92mg/dl	106mg/l	MNPDR	A
58	NAN	1866809	50	M	P	5yr	P	A	130/80	6I12	6I18	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	P	P	P	P	A	A	A	P	A	A	N	N	107mg/dl	105mg/l	MdNPDI	A
59	FGP	1921004	72	M	P	25Yr	P	A	146/84	6I24	6I60	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	P	P	A	A	A	A	P	A	A	AB	AB	136mg/dl	150mg/l	VSNPDI	P
60	EPF	2179049	46	F	A	5yr	P	P	120/80	6I18	6I24	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	170mg/dl	55mg/l	MNPDR	A
61	KGN	452707	46	M	A	4yr	P	A	140/80	6I36	6I24	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	130mg/dl	155mg/l	MdNPDI	A
62	GUM	2198735	68	M	P	13yr	P	P	140/80	6I36	6I36	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	A	A	A	A	A	A	P	A	P	A	P	N	AB	100mg/dl	420mg/l	PDR	P
63	SAP	176463	65	F	P	10yr	P	P	160/80	6I24	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	P	A	A	A	A	P	A	A	A	N	N	131mg/dl	150mg/l	VSNPDI	A
64	JSF	176466	65	F	A	8YR	P	P	140/68	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	N	N	103mg/dl	70mg/l	MdNPDI	A
65	YSS	17854	65	M	P	3yr	P	P	148/80	6I18	6I24	N8	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	162mg/dl	30mg/l	MdNPDI	A
66	KAN	448176	56	M	P	4yr	P	A	130/80	6I18	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	P	P	A	A	A	P	A	A	N	N	180mg/dl	160mg/l	MdNPDI	A
67	YAP	1887365	47	M	P	4yr	P	A	140/90	6I18	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	N	N	146mg/dl	201mg/l	MdNPDI	A
68	PGR	448256	54	M	P	3yr	P	P	120/90	6I18	6I18	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	N	N	140mg/dl	312mg/l	SNPDR	A
69	PSP	452412	44	M	P	5yr	P	P	140/80	6I24	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	110mg/dl	12mg/l	MdNPDI	A
70	VAG	166695	59	M	A	12yr	P	A	13O/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	140mg/dl	121mg/l	MNPDR	A
71	MGB	456402	50	M	P	5yr	P	A	140/90	6I12	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	N	P	P	A	A	P	P	P	P	A	A	A	P	A	A	N	N	141mg/dl	166mg/l	MdNPDI	A
72	CKB	450370	67	M	P	11yr	P	P	148/90	6I24	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	AB	AB	148mg/dl	142mg/l	SNPDR	P
73	SSP	494000	49	M	P	8YR	P	P	130/60	6I18	6I24	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	N	N	160mg/dl	102mg/l	MdNPDI	A
74	AKK	1628730	54	M	P	11yr	P	A	130/74	6I24	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	P	P	A	A	A	A	P	P	A	A	A	P	A	A	N	N	170mg/dl	210mg/l	VSNPDI	A
75	SBI	147687	47	M	A	4yr	P	P	130/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	P	P	A	A	P	P	A	A	A	A	A	P	A	A	N	N	239mg/dl	146mg/l	MdNPDI	A
76	RMM	487427	49	M	A	4yr	P	A	130/76	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	128mg/dl	122mg/l	MdNPDI	A
77	KHS	482315	46	F	A	5YR	P	P	130/86	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	180mg/dl	30mg/l	MdNPDI	A
78	SIK	196167	64	M	P	5YR	P	A	136/80	6I18	6I24	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	N	N	128mg/dl	162mg/l	SNPDR	A
79	DKK	483002	56	M	P	11yr	P	P	140/80	6I12	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	N	P	P	P	P	A	A	P	P	A	A	A	P	A	A	N	N	240mg/dl	67mg/l	VSNPDI	A
80	SAA	484237	64	M	P	5yr	P	A	128/74	6I18	6I24	N12	N12	N	N	N	N	N	N	N	N	N	N	N	P	A	P	P	A	A	A	P	A	A	A	P	A	A	N	N	162mg/dl	212mg/l	MdNPDI	A
81	PKK	478278	41	M	P	9yr	P	A	141/82	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	P	P	P	P	P	P	P	P	A	A	A	P	A	A	AB	AB	201mg/dl	500mg/l	SNPDR	P
82	VGG	1666957	46	M	A	4yr	P	P	160/80	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	136mg/dl	124mg/l	MNPDR	A
83	NYA	138934	54	M	A	5yr	P	P	146/88	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	A	A	P	P	A	A	A	P	A	A	N	N	133mg/dl	130mg/l	MdNPDI	A
84	YSP	137454	38	M	A	2Yr	P	P	140/84	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	N	N	166mg/dl	130mg/l	MdNPDI	A
85	UMM	2284641	52	M	P	12yr	P	P	140/80	6I18	6I18	N12	N12	N	N	N	N	N	N	N	N	N	N	N	P	P	P	P	A	A	A	A	A	A	A	P	A	A	AB	AB	216mg/dl	130mg/l	SNPDR	P
86	MVP	482613	48	M	A	5YR	P	P	130/90	6I6	6I6	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	132mg/dl	122mg/l	MNPDR	A
87	YBB	518203	65	F	P	15YR	P	P	130/82	6I24	6I36	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	A	A	A	P	A	A	A	A	P	A	A	AB	N	122mg/dl	138mg/l	MdNPDI	P
88	JPP	2453625	31	M	A	2Yr	P	P	139/88	6I6	6I6	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	A	A	N	N	108Mg/dl	26mg/l	MdNPDI	A
89	STG	2459702	58	M	P	5yr	P	P	130/86	6I36	6I24	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	A	P	P	A	A	A	P	P	A	AB	AB	203mg/dl	160mg/l	PDR	P
90	MDK	246382	63	F	P	8YR	P	P	140/80	6I18	6I60	N18	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	P	A	A	A	P	A	A	N	N	132mg/dl	128mg/l	MdNPDI	A
91	SCP	1735095	60	F	P	5Yr	P	P	138/60	CF-4M	6I60	N36	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	P	A	P	A	A	A	A	P	A	A	AB	N	200mg/dl	261mg/l	MdNPDI	P
92	SAH	2361776	50	F	P	4YR	P	P	136/74	6I18	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	P	P	P	A	A	A	P	A	A	N	N	108Mg/dl	118mg/l	MdNPDI	A
93	MAB	2491709	59	M	P	9YR	P	P	130/89	6I36	6I24	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	P	P	A	A	A	P	A	A	AB	AB	108Mg/dl	136mg/l	SNPDR	P
94	GDS	2465820	75	M	P	13YR	P	P	160/90	6I36	6I36	N12	N12	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	P	A	P	P	A	A	A	P	P	A	N	N	210mg/dl	140mg/l	PDR	A
95	ACC	2478284	67	M	P	4yr	P	P	140/80	6I18	6I18	N8	N8	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	A	A	A	A	A	P	A	A	N	N	162mg/dl	24mg/l	MNPDR	A
96	MKJ	2584048	54	F	P	10yr	P	P	136/84	6I24	6I18	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	A	A	A	A	A	P	A	P	AB	N	144mg/dl	363mg/l	PDR	P
97	KGD	2573229	48	F	P	4yr	P	P	150/70	6I36	6I24	N8	N8	N	N	N	N	N	N	N	N	N	N	N	P	A	A	P	P	P	P	P	A	A	A	P	A	A	AB	AB	129mg/dl	401mg/l	PDR	P
98	AKK	795247	33	F	P	2Yr	P	P	140/80	6I9	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	A	A	N	N	140mg/dl	62mg/l	MNPDR	A
99	SMM	542272	67	F	P	5Yr	P	A	130/90	6I12	6I9	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	P	P	A	A	A	P	p	A	AB	AB	158mg/dl	311mg/l	PDR	P
1OC	MPP	2592252	72	F	P	1yr	p	P	160/80	6I18	6I12	N6	N6	N	N	N	N	N	N	N	N	N	N	N	A	A	P	P	P	P	A	A	A	A	A	P	P	A	N	N	162mg/dl	338mg/l	PDR	A

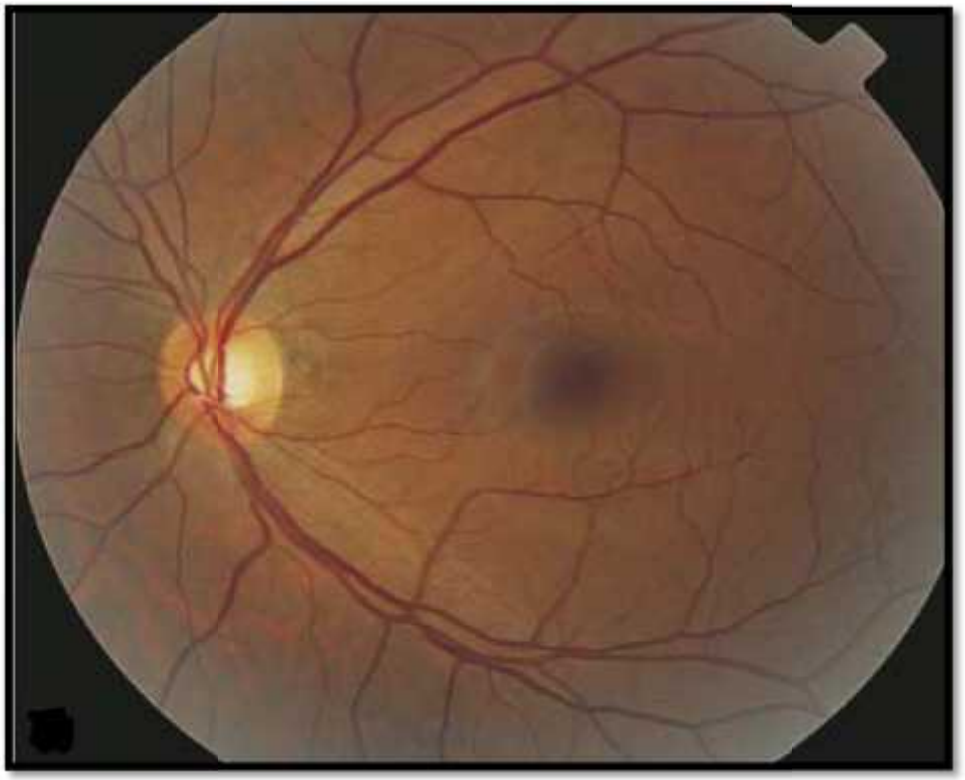


Figure no.14:Normal Fundus

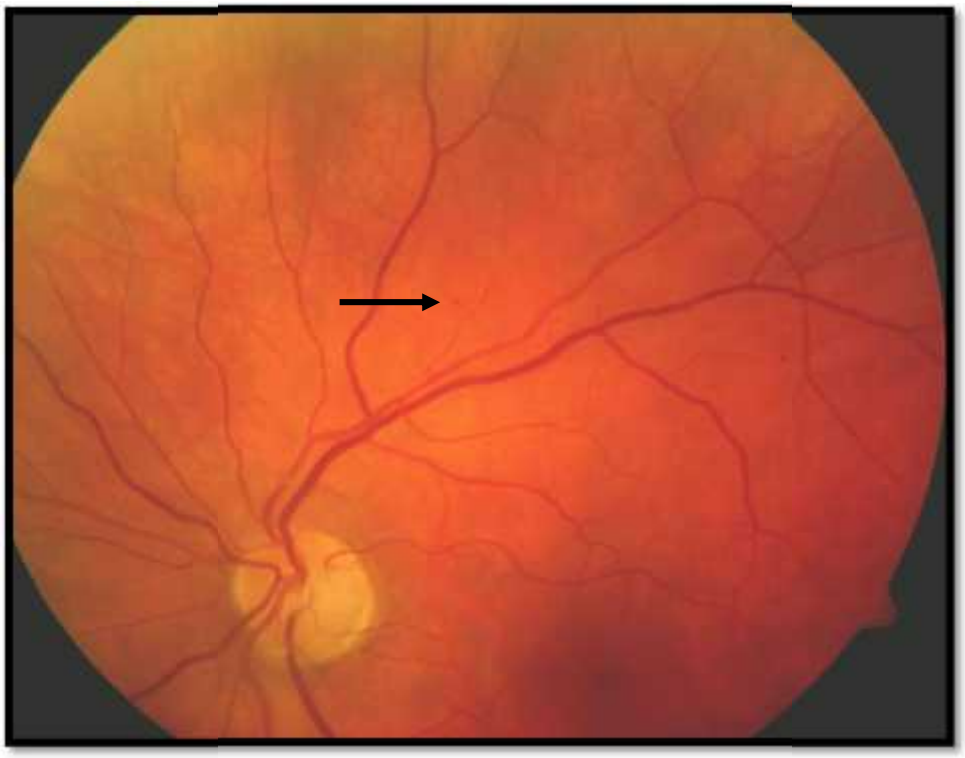


Figure 15 : Microaneurysms

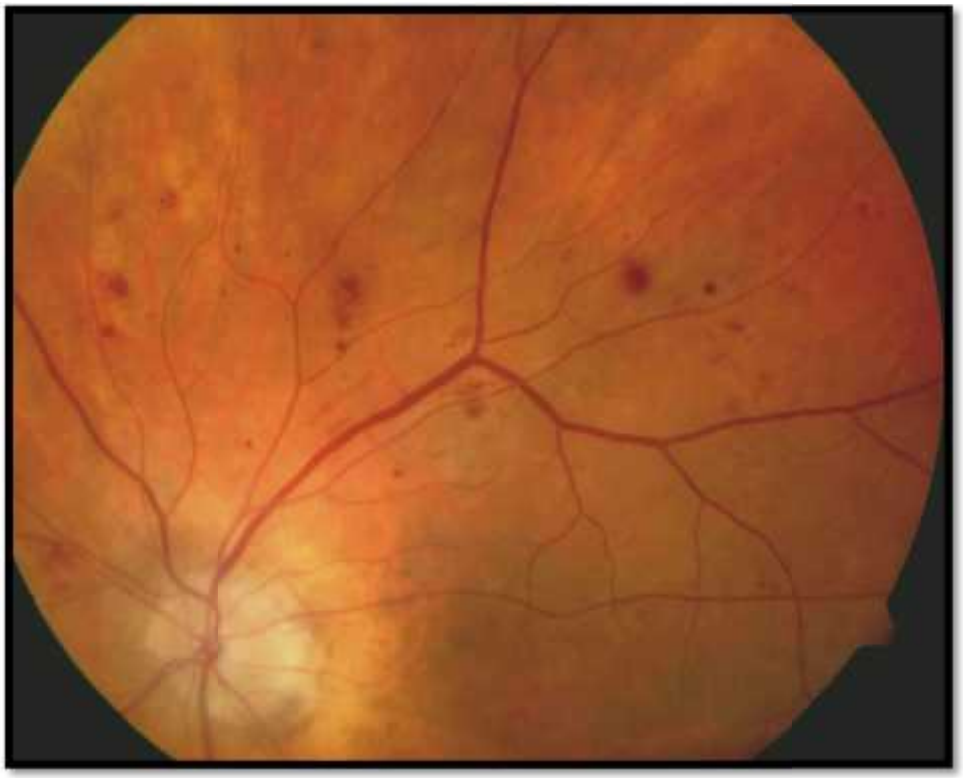


Figure16 : Intraretinal Hemorrhages

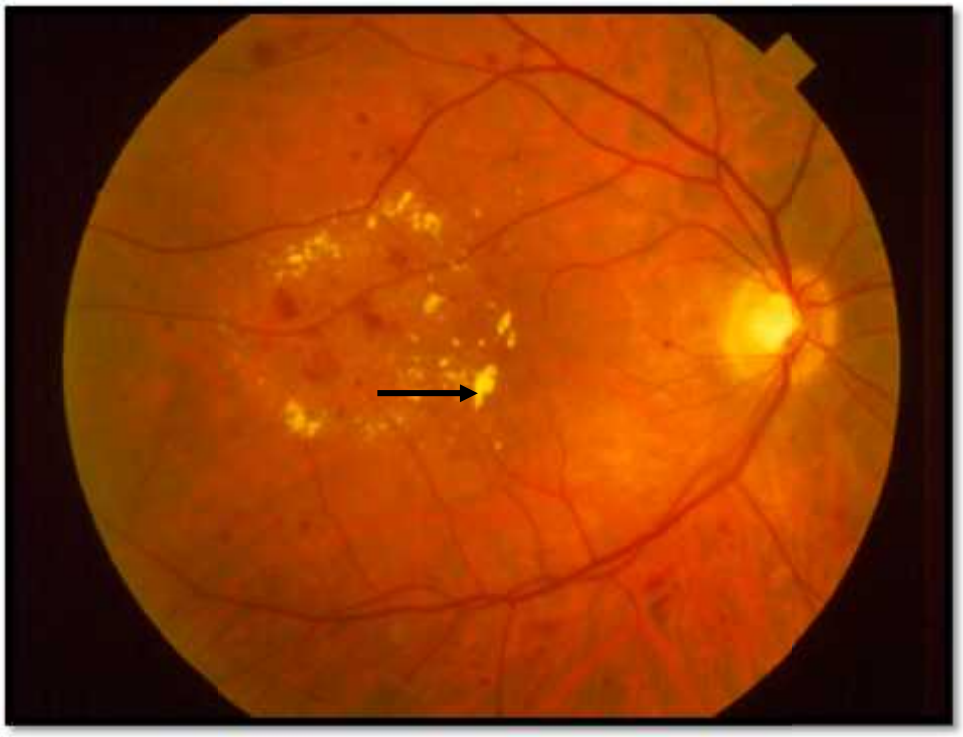


Figure 17 : Hard Exudates with CSME

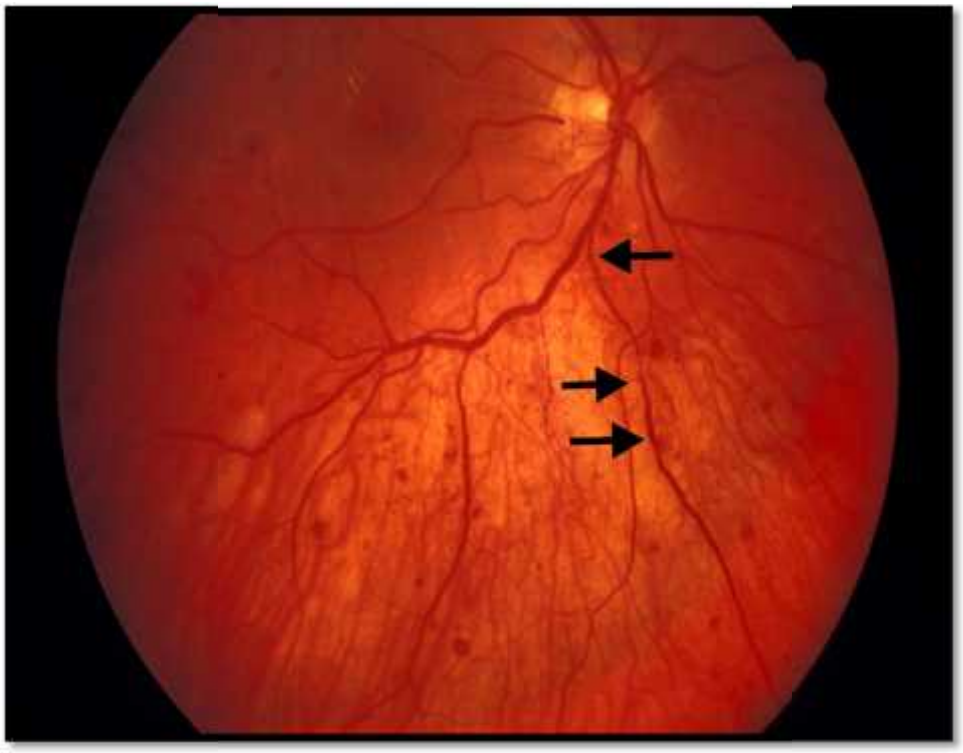


Figure 18 : Venous Beading

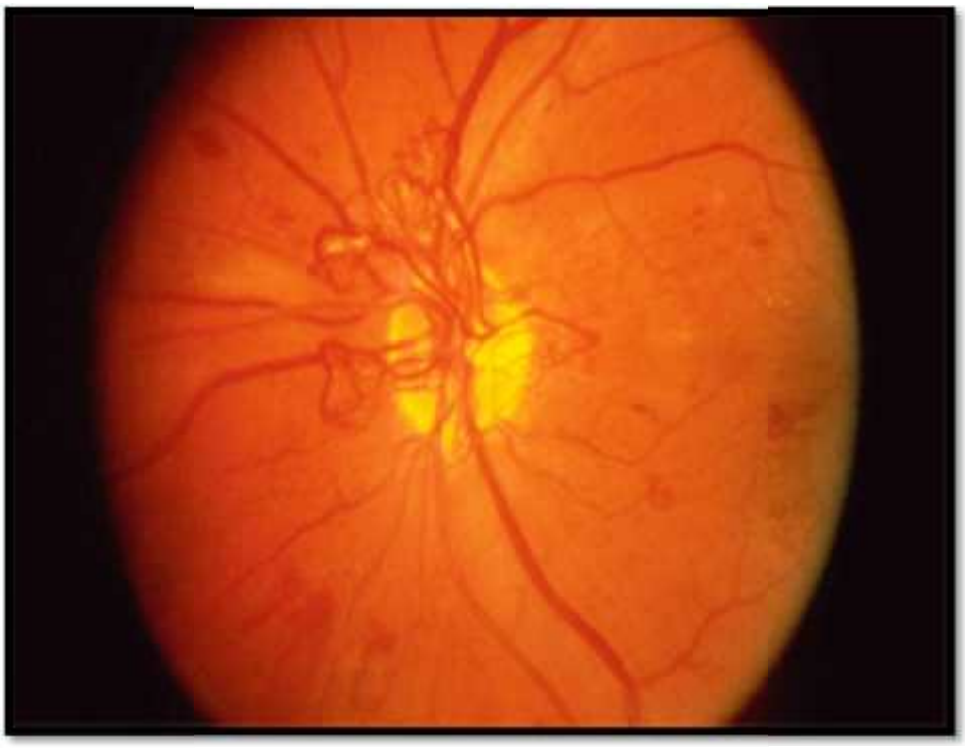
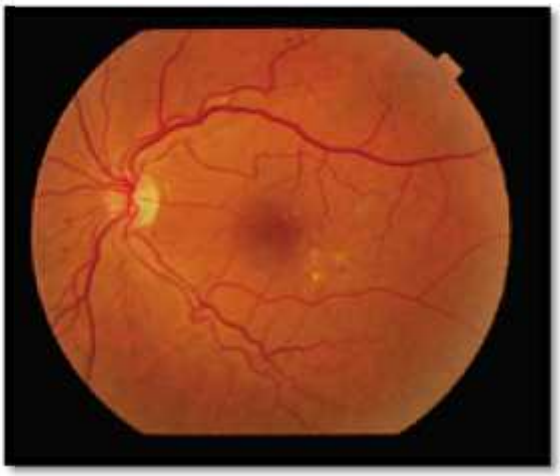


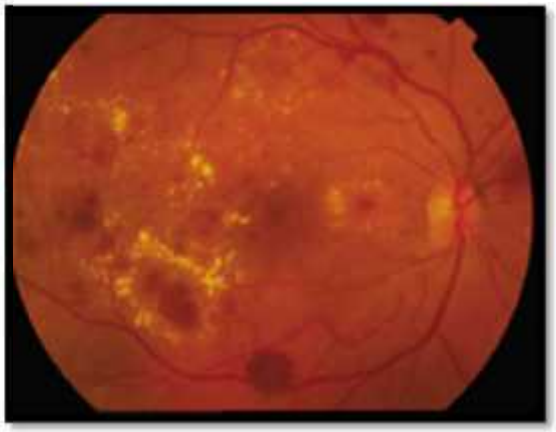
Figure 19 : Neovascularisation

Figure 20.Various Grades of Diabetic Retinopathy



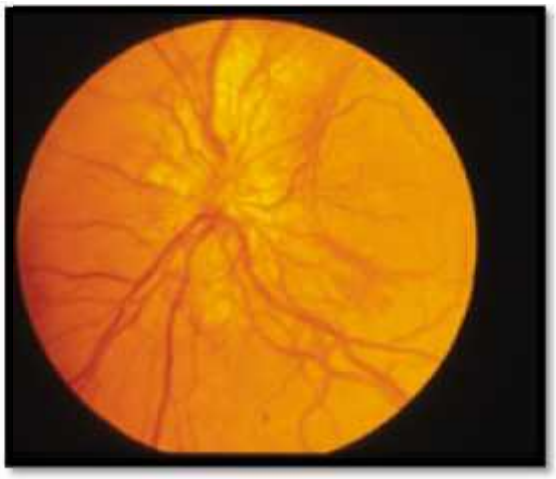
Mild NPDR

Moderate NPDR

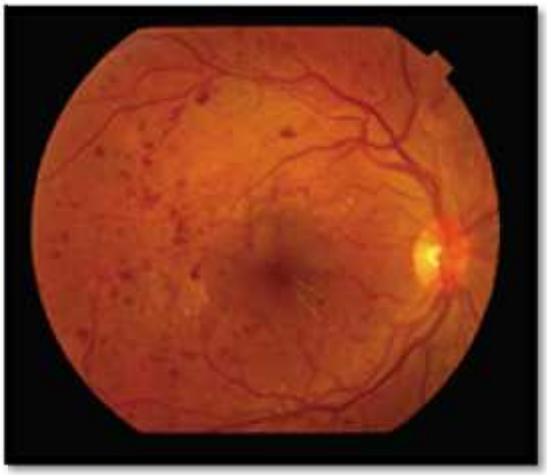


Severe NPDR

Very Severe NPDR



NVD{PDR}



NVE{PDR}

Figure21:CANON FUNDUS CAMERA USED FOR 7 FIELD PHOTOGRAPHY