PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI- HOSPITAL BASED STUDY

DISSERTATION SUBMITTED TO THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

M.D. BRANCH – I

(GENERAL MEDICINE)

Registration No.: 201711359

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 627011

MAY-2020

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI- HOSPITAL BASED STUDY"** submitted by **Dr.S.KATHIRVEL**, to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. Degree Branch – I (General Medicine) is a bonafide research work carried out by him under direct supervision & guidance.

Professor & Head of the Department,
Department of General Medicine
Tirunelveli Medical College,
Tirunelveli.

Unit Chief,
Department of General Medicine
Tirunelveli Medical College,
Tirunelveli.

CERTIFICATE BY THE DEAN

I hereby certify that this dissertation entitled "**PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI- HOSPITAL BASED STUDY"** is a record of work done by **Dr.S.KATHIRVEL**, in the Department of General Medicine, Tirunelveli Medical College, Tirunelveli, during his postgraduate degree course period from 2017- 2020. This work has not formed the basis for previous award of any degree.

Date :
Place : TIRUNELVELI

The DEAN
Tirunelveli Medical College,
Tirunelveli - 627011.

DECLARATION

I solemnly declare that the dissertation entitled **"PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI- HOSPITAL BASED STUDY"** is done by me at Tirunelveli Medical College Hospital, Tirunelveli Under the guidance and supervision of **Prof. Dr. L. RAJAGOPALA MARTHANDAM M.D,** the dissertation is submitted to The TamilnaduDr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

Place: Tirunelveli
Date:

Dr.S.KATHIRVEL
Registration No.: 201711359
Postgraduate Student,
M.D General Medicine,
Department of General Medicine,
Tirunelveli Medical College
Tirunelveli.

CERTIFICATE – II

This is to certify that this dissertation work entitled **"PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI- HOSPITAL BASED STUDY"** of the candidate **Dr.S.KATHIRVEL** with registration Number **201711359** for the award of **M.D.**Degreein the branch of **GENERAL MEDICINE (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **22 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

URKUND

Urkund Analysis Result

Analysed Document:	Prevalence of retinopathy and nephropathy in newly diagnosed type 2 DM patients atttedning tertiary care hospital, tirunelveli - hospital based study.pdf (D57295186)
Submitted:	10/20/2019 7:16:00 AM
Submitted By:	kathirvels1165@gmail.com
Significance:	22 %

Sources included in the report:

https://bacsinoitru.vn/f26/benh-than-dai-thao-duong-136.html
https://archive.org/stream/APracticalGuideToDiabetesMellitus7thEdgnv64/A%20Practical%20Guide%20to%20Diabetes%20Mellitus%20%25287th%20Ed%2529%2528gnv64%2529_djvu.txt
https://docplayer.net/121171337-Incidence-of-retinopathy-changes-in-new-cases-of-diabetes-mellitus-type-2.html
https://www.slideshare.net/fahmidahoque1/diabetic-retinopathy-76256485

Instances where selected sources appear:

27

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572738-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 1210/GM/2017

PROTOCOL TITLE: PREVALENCE OF RETINOPATHY AND NEPHROPATHY IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS ATTENDING TERTIARY CARE HOSPITAL, TIRUNELVELI-HOSPITAL BASED STUDY
PRINCIPAL INVESTIGATOR: Dr. S. KATHIRVEL, MBBS.
DESIGNATION OF PRINCIPAL INVESTIGATOR: PG STUDENT
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear, Dr. S.KATHIRVEL, MBBS, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 15.12.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantarama, MD
Registrar, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Dr.J.Suresh Durai, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

ACKNOWLEDGEMENT

I wish to express my heartfelt gratitude to our Dean Prof.Dr. S. M. .Kannan M.S., MCh., Tirunelveli Medical College for allowing me to do the study in this institution.

I would like to express my humble thanks to our professor & Head of the Department **Prof. Dr. M.Ravichandran M.D**., Department of General Medicine.

I express my sincere thanks to my renowned teacher and my guide **Prof. Dr. L. RAJAGOPALA MARTHANDAM M.D,** Professor, Department of General Medicine, Tirunelveli Medical College for his guidance, valuable suggestions and constant encouragement throughout the study.

I express my sincere thanks to my former professors **Dr.S.Arumugapandian @ Mohan,M.D.,** for their constant support, encouragement and suggestions which helped me greatly to expedite this dissertation .

I am greatly obliged **to Dr.V.Ramasubramanian M.D., D.M., (Nephrology), Dr. P.K. Senthil Kumar M.D., D.M., (Nephrology), V.Ramalakshmi M.S., (Ophthalmology)., Dr. J. Bharath M.D., Dr. M.Veerapandian M.D., Dr.P. Meena Kumari M.D., Dr. P. Ganesh Kumar M.D., Dr. A. Vinoj M.D.,** Assistant Professors, Dept. of General Medicine for their valuable suggestions in preparing this dissertation.

CONTENT

ABBREVIATIONS

BMI – Body Mass Index

LDL – Low Density Lipoprotein

VLDL – Very Low Density Lipoprotein

HDL – High Density Lipoprotein

TG – Triglycerides

NPDR – Non Proliferative Diabetic Retinopathy

PDR – Proliferative Diabetic Retinopathy

CSME – Clinically Significant Macular Edema

INTRODUCTION

Diabetes mellitus describes a group of metabolic disease in which the person has increased blood glucose values, either because insulin production is insufficient or because the body's cells do not respond properly to insulin or both

There is no defined diabetes cause. Because the causes of diabetes mellitus vary depending on the individual and type. Diabetes mellitus is a common disorder with an annual prevalence of 8.2%. Type 2diabetes mellitus is the most common form (90%).

There is usually an asymptomatic period between the actual onset of hyperglycemia and clinical diagnosis. This asymptomatic phase is estimated to last for 4-7 years [(1)]. Because of this undiagnosed and untreated chronic hyperglycemia is responsible for the increased prevalence of microvascular complications in newly diagnosed diabetes mellitus.

Coronary artery disease, peripheral arterial disease and cerebrovascular disease are the macrovascular complications of diabetes mellitus. Microvascular complication in diabetes include

- Diabetic retinopathy
- Diabetic nephropathy
- Diabetic neuropathy

Some studies have shown diabetic retinopathy is common in newly detected diabetes mellitus[2]. It is seen in 39% of men and 35% of women ,with marked retinopathy present in 4% of women and 8% of men at the time of diagnosis of diabetes mellitus[3]. It remains the leading cause of blindness in most of the diabetic population. With appropriate medical care visual loss can be prevented.

Incidence of diabetic nephropathy in newly detected type2 diabetes mellitus is as high as 17.34%. Poor glycaemic control, high HbA1C, Increased BMI, dyslipidemia, hyperuricemia male sex may play a significant role in diabetic nephropathy. Presence of microvascular complication in newly detected diabetes mellitus are showing increasing trends in India. Early detection of microvascular complications like diabetic retinopathy, diabetic nephropathy and its treatment prevent the progression of these complications and hence morbidity and mortality among patients.

Our study is to detect microvascular complications like diabetic retinopathy and diabetic nephropathy at the time of diagnosis of diabetes mellitus.

REVIEW OF LITERATURE

Diabetes mellitus is a group of metabolic disease characterized by hyperglycemia resulting from defects in

1.insulin secretion

2.insulin action

3.or combination of both.

HISTORICAL REVIEW

DIABETES MELLITUS

The knowledge of diabetes dates back to centuries before Christ. Polyuric disease resembling diabetes was described as early as 150 BC in ancient Egyptiyan records discovered by George Beers. Celsius (30 BC – 50 AD) had recognized the disease.

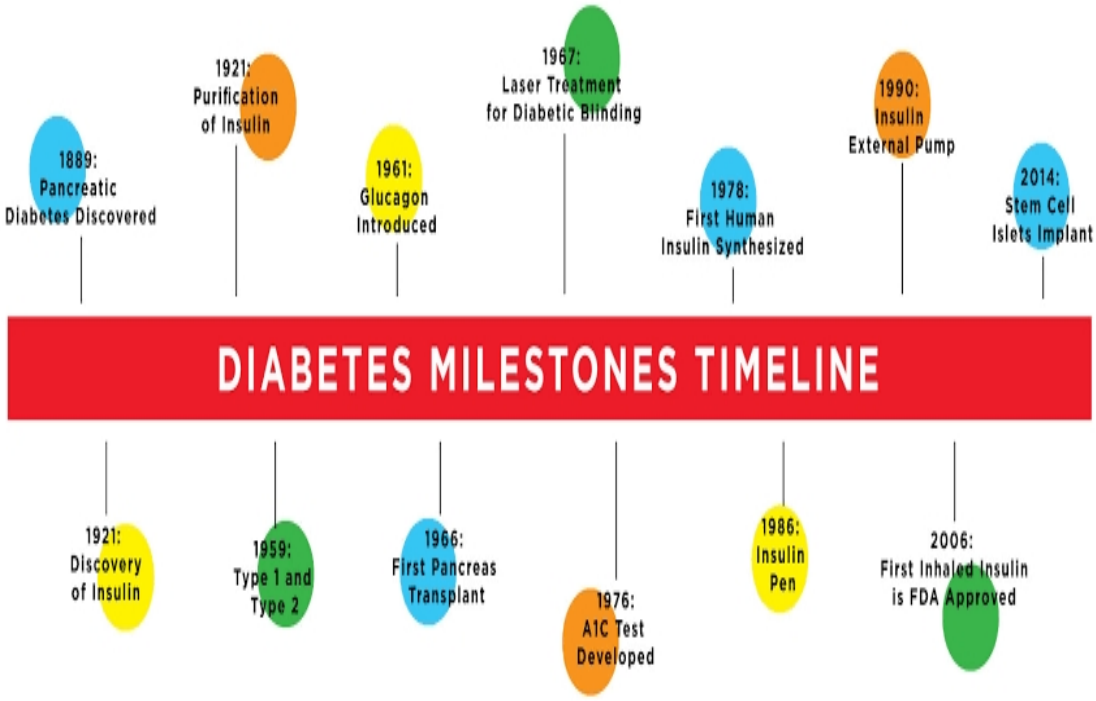
Diabetes, a Greek term which literally means to 'run thru' or a 'siphon' was initially used by Aretaeus in 1ST century AD for the generic description of a condition causing increased urine output[4]. Roman physicians thought of diabetes as a wonderful affection, not very frequent among men, being melted down of flesh and limbs into urine. The patient never stopped making water , but the flow is incessant as if from a opening of aqueducts – Aretaeus , the Cappadocian[5,6]. The association of polyuria

with a sweet tasting substance in the urine was first reported in Sanskrit literature dating from 5th to 6th centuries AD at the time of two noted Indian physicians Susruth and Charaka.

It was in the 17th century that Thomas Willis (1621-1675) made the observation “as if imbibed with honey and sugar about the diabetic urine “ A century after Willis , Mathew Dobson (1735-1784) demonstrated that the sweetness of urine was indeed due to sugars. It was John Rollo who was one of the first to use the adjective mellitus (mellitus-honey) to distinguish it from other polyuric states in which the urine was unsavory (Greek – insipitus). Over the centuries , gradually the causes and the complications of this disease were recognized.

Effective treatment for diabetes was not developed until the early part of the twentieth Century. Diabetes mellitus, colloquially referred to as diabetes is a multi-system metabolic disease in which there is high plasma sugar values.

Diabetes is due to reduced level of production of insulin by the pancreatic islets or due to receptor level resistance to insulin despite adequate circulating insulin levels .



ETIOLOGICAL CLASSIFICATION OF DIABETES

I. Type 1 diabetes mellitus

A. Immune mediated

B. Idiopathic

II. Type 2 diabetes mellitus

III. Other specific types

A. Genetic defects in beta cell function – MODY type 1 to type 6 mitochondrial diabetes

B. Genetic defects in Insulin action

- Type A Insulin resistance
- Lipoatrophic diabetes

C. Pancreatic diseases Fibrocalcific pancreatitis

Pancreatectomy

Cystic fibrosis

D. Endocrinopathies – Acromegaly

Cushing's syndrome

Pheochromocytoma

Hyperthyroidism

E. Drug induced – Glucocorticoids

Thyroid hormone

Diazoxide

Thiazides

Phenytoin

Vacor

Pentamidine

Olanzapine

Rifampicin

F. Infections –

- Congenital Rubella
- Cytomegalovirus
- Mumps

G. Uncommon forms of immune mediated diabetes – “Stiff-man” syndrome , Anti-insulin receptor antibodies

H. Genetic syndrome association

- Down’s syndrome
- Turner’s syndrome
- Klinefelter’s syndrome
- Myotonic dystrophy
- Prader-Willi syndrome

IV. Gestational Diabetes

CRITERIA FOR DIAGNOSIS OF DIABETES MELLITUS

- HbA1c ≥ 6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay

OR

- Fasting ≥ 126 mg/dL. Fasting is defined as no caloric intake for at least 8 hours

OR

- 2-hr plasma glucose ≥ 200 mg/dL during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water*

OR

- Symptoms of hyperglycemia and a casual plasma glucose ≥ 200 mg/dL. Casual is defined as any time of day without regard to time since last meal. The classic symptoms of hyperglycemia include polyuria, polydipsia, and unexplained weight loss

RISK FACTORS

Modifiable risk factors

- Over weight (obesity)
- Sedentary life style
- Previously identified glucose intolerance (IGT and / or IFG)
- Metabolic syndrome
- Dietary factors
- Intrauterine environment
- Smoking

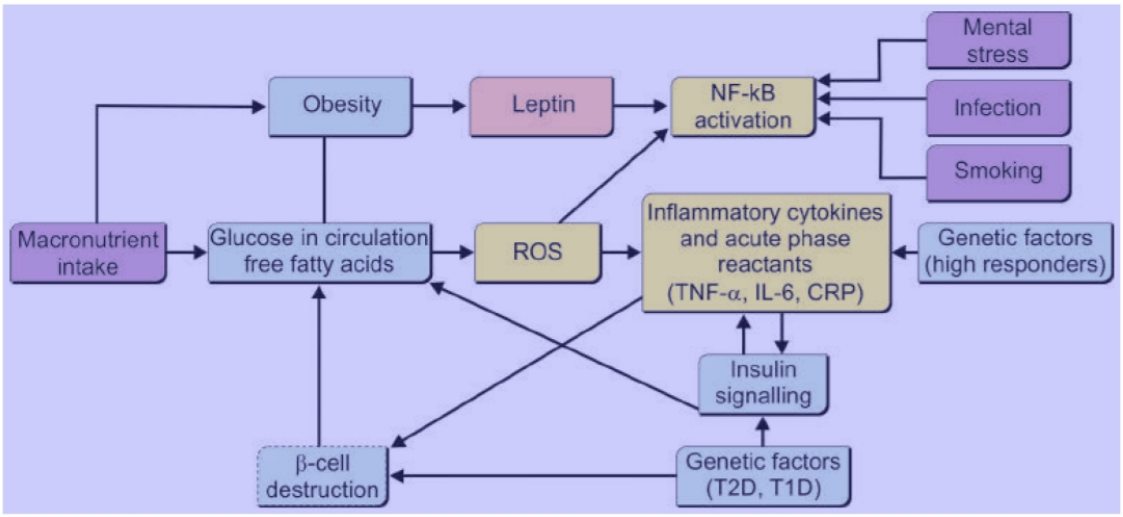
Non modifiable risk factors

- Ethinicity (African – American , native American or pacific islanders)
- Family history of type 2 diabetes mellitus
- Age
- Gender
- History of gestational diabetes
- Polycystic ovarian syndrome
- Inflammation

TYPE 2 DIABETES MELLITUS

This occurs due to insulin resistance or insensitivity of tissue to insulin and relative insulin deficiency.Over weight is a major risk factor for the disease. The insulin resistance seems to be caused by the toxic effects of dyslipidemia which interferes with insulin signalling processes between receptor activation and cellular effects. Some studies have shown that obese individuals have reduced number of insulin receptors in adipose tissue, muscle and liver. They usually show an improvement in glucose tolerance with exercise.

ROLE OF GENETIC FACTORS, OBESITY AND INFLAMMATION IN DIABETES

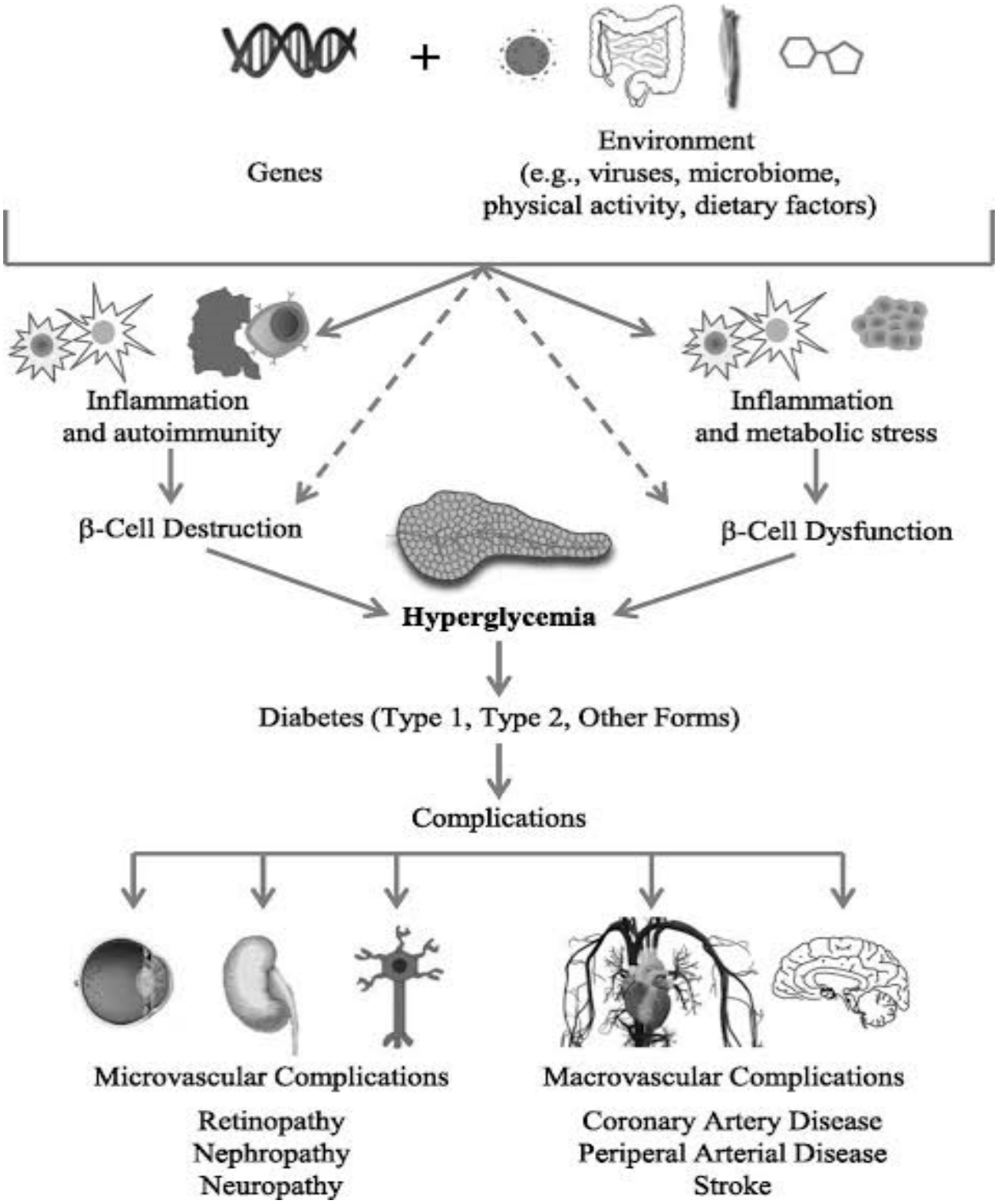


Source: Dandona P, Aljada A, Bandyopadhyay A. Inflammation: the link between insulin resistance, obesity and diabetes. Trends Immunol. 2004;25:4-7.
(ROS: Reactive oxygen species; NF-κB: Nuclear factor-kappaB; CRP: C-reactive protein; TNF: Tumor necrosis factor; IL: Interleukin; T1D: Type 1 diabetes; T2D: Type 2 diabetes).

CONSEQUENCES OF DISTURBED METABOLISM IN DIABETES

Metabolic defect	*Chemical abnormality*	*Clinical abnormalities*
Carbohydrate metabolism		
• ↓↓ Glucose uptake by tissues (muscle, adipose tissue, liver) • ↑↑↑ Glucose production 2° to Glycogenolysis and glyconeogenesis in liver	Hyperglycemia ↓ Glycosuria ↓ Osmotic diuresis	• Polyuria, Polydipsia, Polyphagia • Blurred vision • Diminished mental alertness • Dehydration (→ death)
Protein metabolism		
• ↓ Uptake of amino acids • ↓ Protein synthesis • ↑ Proteolysis	• Negative nitrogen balance • ↑ Levels of branch chain amino acid • ↑ Blood urea nitrogen level • ↑ K^+ level	• Weakness • Poor resistance to infections • Muscle wasting
Lipid metabolism		
• ↑ Lipolysis • ↓ Lipogenesis • ↑ Triglycerides, FFA production • ↑ Ketone production • ↓ Ketone excretion • ↑ Production of LDL and VLDL	• ↑ Plasma FFA • ↑ Plasma glycerol • Hypertriglyceridemia • ↑ Plasma and urine ketone • ↑ Plasma LDL and VLDL	• Loss of adipose tissue → Weight loss • Pancreatitis • Metabolic acidosis → Hyperventilation → Kussmaul breathing → death • Atherosclerotic vascular disease

PATHOGENESIS OF DIABETES MELLITUS



SIGNS AND SYMPTOMS

The classical symptoms of hyperglycemia are

- Weight loss
- Polyuria
- Polyphagia
- Polydipsia
- Several signs and symptoms are signal the onset of diabetes although they are not specific for the disease they include
- Blurred vision
- Thirst
- Fatigue
- Slow healing of wounds
- Headache
- Itchy skin
- Hunger
- Vaginal infection
- Frequent urination
- Sudden weight loss
- Tingling sensation of hands and legs

COMPLICATIONS OF DIABETES

Acute

- Diabetic ketoacidosis
- Hyperglycemic hyperosmolar state
- Hypoglycemia

Long term

MICROVASCULAR COMPLICATIONS

- Retinopathy
- Neuropathy
- Nephropathy

MACROVASCULAR COMPLICATION

- Ischemic heart disease
- Cerebrovascular disease
- Peripheral vascular disease

Others

- Infections
    - UTI
    - Tuberculosis
    - Candidiasis – Oral / Vulvovaginal

- Mucormycosis

- Necrotising fasciitis
- Periodontitis

DIABETIC RETINOPATHY

Diabetic retinopathy is the most common cause of legal blindness in 20 - 70 years. Blindness usually due to non-resolving vitreous hemorrhage, tractional retinal detachment or diabetic macular oedema. However, the 5-year risk of severe visual loss can be reduced if a person with proliferative DR undergoes laser photocoagulation. DR is usually asymptomatic in its most treatable stages; so early detection through regularly scheduled ocular examination is important[7] .

Epidemiology

The prevalence of retinopathy at the time of diagnosis is much greater in type 2 (6.7–30.2%) than type 1 (0–3%), because the former is more likely to remain undiagnosed for longer periods of time. Approximately one fourth of diabetic patients is unaware of their disease. Prevalence of proliferative DR is more in long standing type 1 diabetes mellitus. However, in type 2 DM patients on insulin, risk is equally high (25%) possibly owing to chronic hyperglycemia.

Risk Factors

Duration of diabetes

- Most important factor
- Longer the duration higher the incidence
- Rare before puberty.

Poor control of diabetes

- Worsens the progression
- Tight control does not guarantee prevention, but delays the onset and slows progression.

Pregnancy

- Associated with rapid progression of pre-existent retinopathy
- Postpartum reversal of retinopathy may occur
- It is rare for women without retinopathy to develop it during pregnancy
- De novo gestational diabetes has no risk of retinopathy.

Hypertension is associated with worsening of Retinopathy

Nephropathy

- Associated with worsening worsening of Retinopathy
- Treatment as with renal transplantation leads to improvement.

Obesity

Hyperlipidemia

Smoking.

CLASSIFICATION

- Non proliferative diabetic retinopathy
- proliferative diabetic retinopathy
- clinically significant macular edema

Non proliferative diabetic retinopathy is further classified into

- Mild
- Moderate
- Severe
- Very severe

NONPROLIFERATIVE DIABETIC RETINOPATHY

Mild NPDR

- one microaneurysm earliest clinically detectable lesion
- Retinal hemorrhages and hard or soft exudates may be present.

Moderate NPDR

- Microaneurysms + dot and blot hemorrhages in at least one quadrant
- Soft exudates (Cotton wool spots)
- Venous beading or intraretinal microvascular abnormalities (IRMA).

Severe NPDR

- Microaneurysms and intraretinal hemorrhages in all four quadrants
- Venous beading in two or more quadrants
- Moderate IRMA in at least one quadrant
- Known as the 4-2-1 rule.

Very Severe NPDR

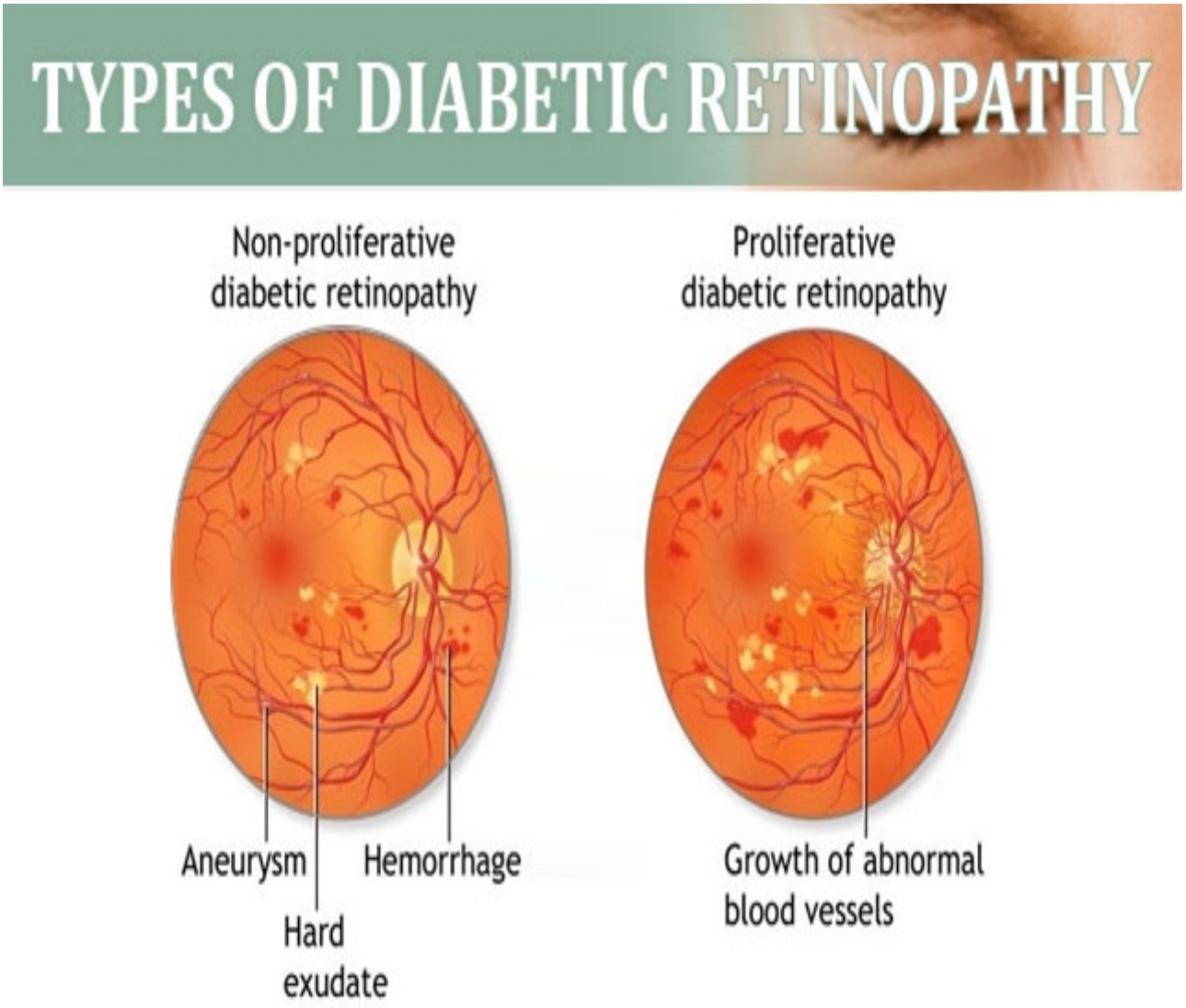
- any two of the features of the 4-2-1 rule

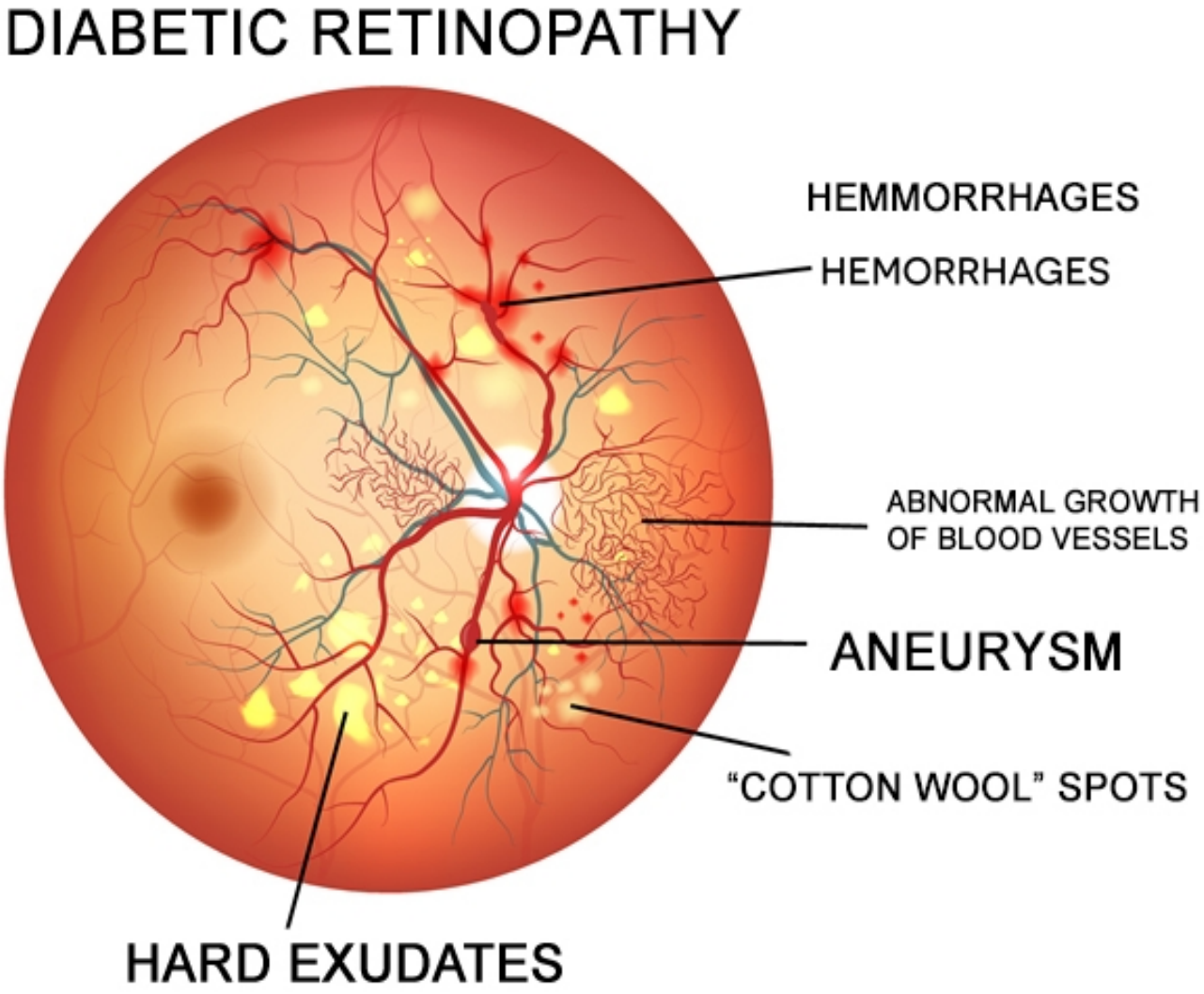
Proliferative Diabetic Retinopathy

- Proliferation of new vessels, usually from the veins.
- New vessels on the optic disk (NVD)
- New vessels elsewhere on the retina, along the course of the retinal vessels (NVE).

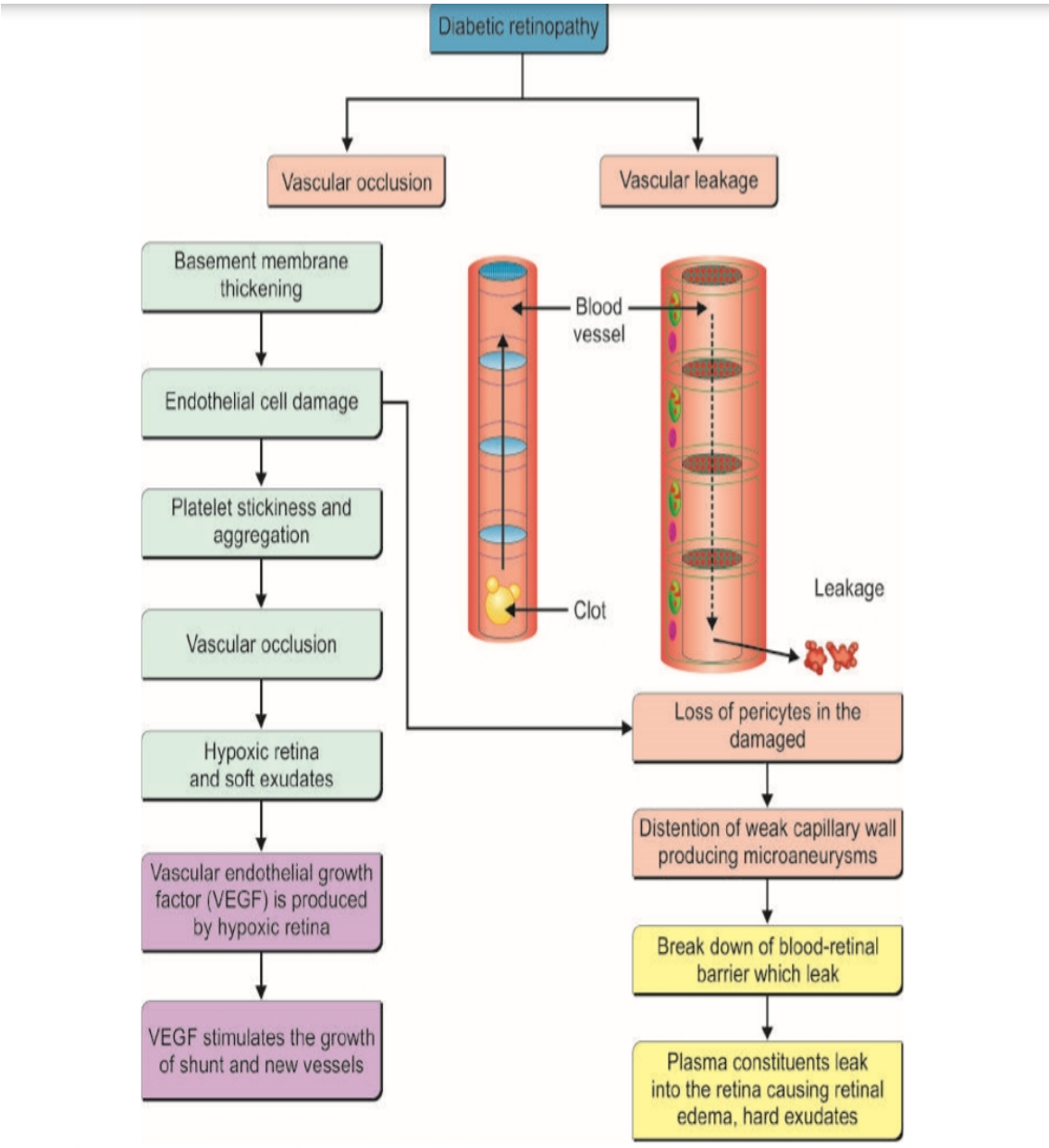
Clinically Significant Macular Edema

- Presents with dimness of vision
- Retinal edema close to fovea
- Hard exudates close to fovea associated with adjacent retinal edema.


TYPES OF DIABETIC RETINOPATHY
Non-proliferative diabetic retinopathy
Proliferative diabetic retinopathy
Aneurysm
Hemorrhage
Hard exudate
Growth of abnormal blood vessels


DIABETIC RETINOPATHY
HEMMORRHAGES
HEMORRHAGES
ABNORMAL GROWTH
OF BLOOD VESSELS
ANEURYSM
"COTTON WOOL" SPOTS
HARD EXUDATES

PATHOGENISIS OF DIABETIC RETINOPATHY



It is a microangiopathy caused by effect of hyperglycemia on small blood vessels leading to

- retinal capillary occlusion
- retinal capillary leakage.

It is caused by

- Sorbitol accumulation (glucose $\xrightarrow{\text{aldose reductase}}$ sorbitol)
- Free radical mediated oxidative stress
- Accumulation of advanced glycation end products
- Excessive activation of protein kinase C and diacyl glycerol

FUNDUS FINDINGS OF DIABETIC RETINOPATHY

Microaneurysms:

- Earliest sign of diabetic retinopathy
- appears as tiny red dots
- saccular outpouchings of damaged retinal capillaries
- appears first temporal to the fovea

Dot and Blot Hemorrhages

- Due to rupture of wall of microaneurysm
- If the hemorrhage is deep, it is round or oval

- Vitreous haemorrhage – bleeding into the vitreous

Flame shaped haemorrhage

- Arises from superficial pericapillary arterioles
- If the haemorrhage is in the nerve fiber layer , it takes a flame shape
- They follow the architecture of the nerve fiber layer

Exudates

Hard exudates

- lipid deposits due to chronic leakage from damaged vessel
- waxy, glistening lesions with distinct margins
- appears as a ring around leaking micro aneurysms (circinate retinopathy)
- it can be reabsorbed spontaneously
- or can be resolved following laser photo coagulations

Cotton wool spots (soft exudates)

- white fluffy lesion in nerve fiber layer
- localized nerve fiber layer infarcts
- indicates worsening of NPDR

Venous beading

- focal areas of venous dilatations
- intra retinal microvascular abnormalities
- dilated tortuous channels between diseased arterioles and venules
- recognized only by slit lamp microscope

Neo vascularization

- new vessels on the disc
- new vessels elsewhere on the retina
- network of fine wisps and strands
- flat or elevated
- associated with fibrosis

Tractional retinal detachment

- due to contraction of the fibro vascular mass
- mass is adherent to the vitreous
- it exerts traction on the retina

SCREENING PROTOCOL FOR DIABETIC RETINOPATHY

DIABETES TYPE	RECOMMENDED TIME FOR FIRST EXAMINATION	RECOMMENDED FOLLOW UP
TYPE I	3-5 years after diagnosis	yearly
TYPE II	At the time of diagnosis	yearly

Symptoms of diabetic retinopathy

- Blurred vision
- Floaters and flashes
- Fluctuating vision
- Dark areas in the vision
- Distorted vision
- Impaired colour vision
- Partial or total loss of vision
- Poor night vision

COMPLICATIONS OF DIABETIC RETINOPATHY

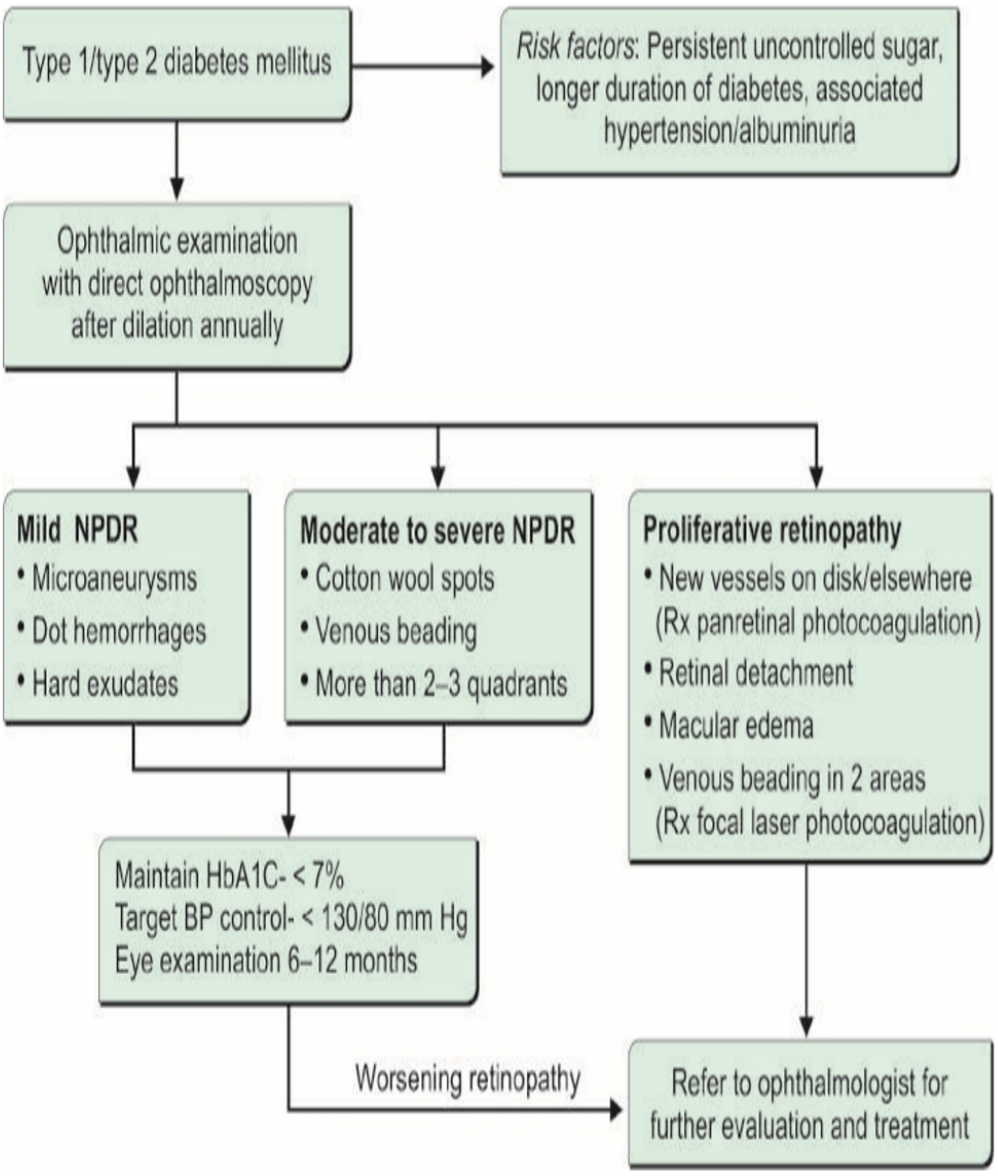
All are essentially complications of neovascularization

- Vitreous haemorrhage
- Tractional retinal detachment
- Rubeosis iridis
- Glaucoma

All complications are preventable if appropriate treatment is instituted early enough.

TREATMENT

Algorithm for management of retinopathy



(NPDR: Nonproliferative diabetic retinopathy)

Indications of treatment

- NVD > 1/3rd disc in area
- Less extensive NVD + haemorrhage
- NVI >1/2 disc in area + haemorrhage

Treatment of underlying disorders

- Glycaemic control
- Insulin, oral hypoglycaemic agents
- Blood pressure control – anti hypertensive medications
- Cholesterol control – statins, fibrates
- Support renal function- ACE inhibitors, ARB
- Life style modifications
- Smoking cessation
- Alcohol cessation
- Exercise
- Weight control

LASER PHOTOCOAGULATION

- Focal
- Laser therapy to seal leaking blood vessels(focal lasers)
- Clinically significant macular edema (grid pattern)

Panretinal photocoagulation

- Laser therapy to reduce the oxygen demand on retina

Vitrectomy

- Surgical treatment to remove extensive vitreous fibro vascular strands
- Because these fibro vascular strands causing retinal traction

Intra retinal anti-VEGF

- Bevacizumab
- Ranibizumab

Intra retinal steroids

- Triamcinolone acetonide

Pars plana vitrectomy

DIABETIC NEPHROPATHY

Diabetic nephropathy is a clinical syndrome defined by the persistent proteinuria greater than 500mg/24hours in a person with diabetic retinopathy without other renal disease. Albuminuria >300mg/24hours is equivalent to a proteinuria >500mg/24hours[8].

Epidemiology

Approximately 40% of patients with type1diabetes or type2 diabetes develop nephropathy. But due to the higher prevalence of type 2 diabetes (90%) compare to type1 (10%), the majority of patients with diabetic nephropathy have type 2 diabetes. Microalbuminuria appears 5-10 years after the onset of diabetes.

RISK FACTORS

- ✓ Elevated arterial blood pressure
- ✓ poor glycemic control-most important
- ✓ uncontrolled hypertension
- ✓ HbA1C >12%

OTHER CONTRIBUTING FACTORS

- ✓ Hyperglycemia induced glomerular hyperfiltration
- ✓ Obesity

- ✓ Smoking
- ✓ Dyslipidemia
- ✓ Degree of proteinuria at diagnosis
- ✓ Dietary factors - amount and type of ingested protein and fat
- ✓ Family history of diabetes and kidney disease

CLASSIFICATION

MOGENSEN'S CLASSIFICATION[8]

Stage I

- ✓ Increased glomerular filtration rate (GFR>90ml/min)
- ✓ Result of concomitant renal hypertrophy (glomerular and tubular)
- ✓ Contributing factors
    - o Intra renal hemodynamic abnormalities
    - o TGF beta
    - o Increased salt absorption

Stage II-The Silent stage or the stage of apparent normalcy

- ✓ This stage is clinically silent
- ✓ It lasts for 5-15years
- ✓ GFR has returned to normal (GFR 60-89ml/min) and no evidence of albuminuria

- ✓ Glomerular damage – basement membrane thickening and mesangial expansion

Stage III

- ✓ Stage of moderately increased albuminuria / microalbuminuria / incipient nephropathy
- ✓ This stage occurs 6-15years after the diagnosis of diabetes
- ✓ Urine albumin excretion has increased up to 30 to 300mg/24hours
- ✓ Renal function could be normal or reduced
- ✓ 30-50% of patients may show reversal of microalbuminuria
- ✓ Persistent microalbuminuria if left untreated will progress to ESRD
- ✓ So all diabetes patients should be screened for microalbuminuria

Stage IV

- ✓ Macroalbuminuria /overt nephropathy/ established nephropathy
- ✓ This stage occurs 15-18 years after the diagnosis of diabetes
- ✓ Urine albumin excretion rate >300mg/24hours
- ✓ Most patients at this stage have hypertension
- ✓ This stage is associated with dyslipidemia and asymptomatic myocardial ischemia
- ✓ If left untreated it will progress to ESRD

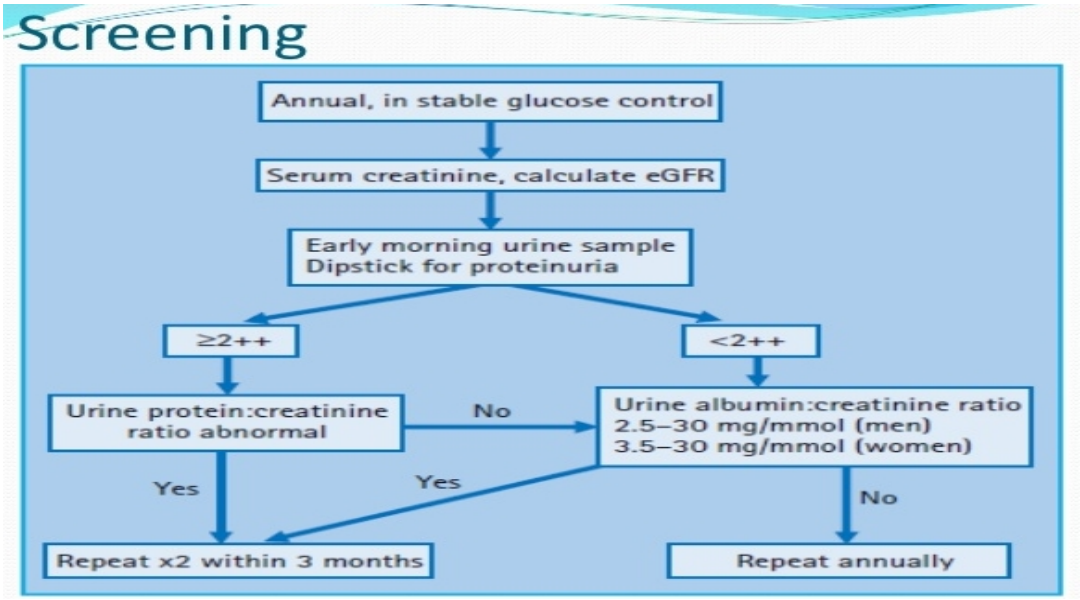
Stage V – uremia /ESRD

- ✓ GFR has fallen to <15ml/min
- ✓ Renal replacement therapy is needed at this stage (hemodialysis , peritoneal dialysis , kidney transplantation)

SCREENING

Screening can be done by 3 methods

- Random urine sample – albumin to creatinine ratio
  (Preferably Early morning sample)
- 24 hours urine protein collection
- Timed urine collection
- Initial screening may be done by using a standard urine dipstick
- Serum creatinine level also be measured annually
- Recently cystatin C has been suggested as an alternative to creatinine measurement.



PATHOGENESIS

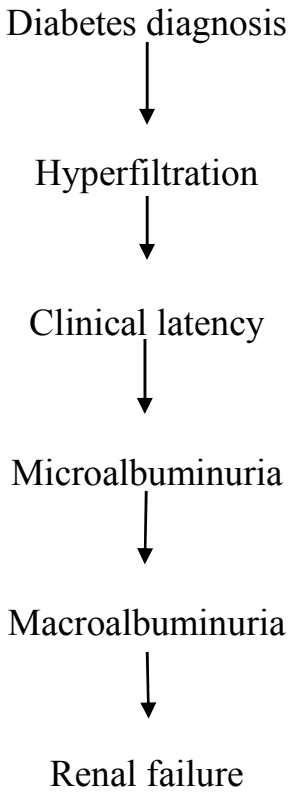
3 MAIN CHANGES IN DIABETIC NEPHROPATHY

I - Glomeruli – expansion of mesangium by increased production of matrix or by their glycosylation

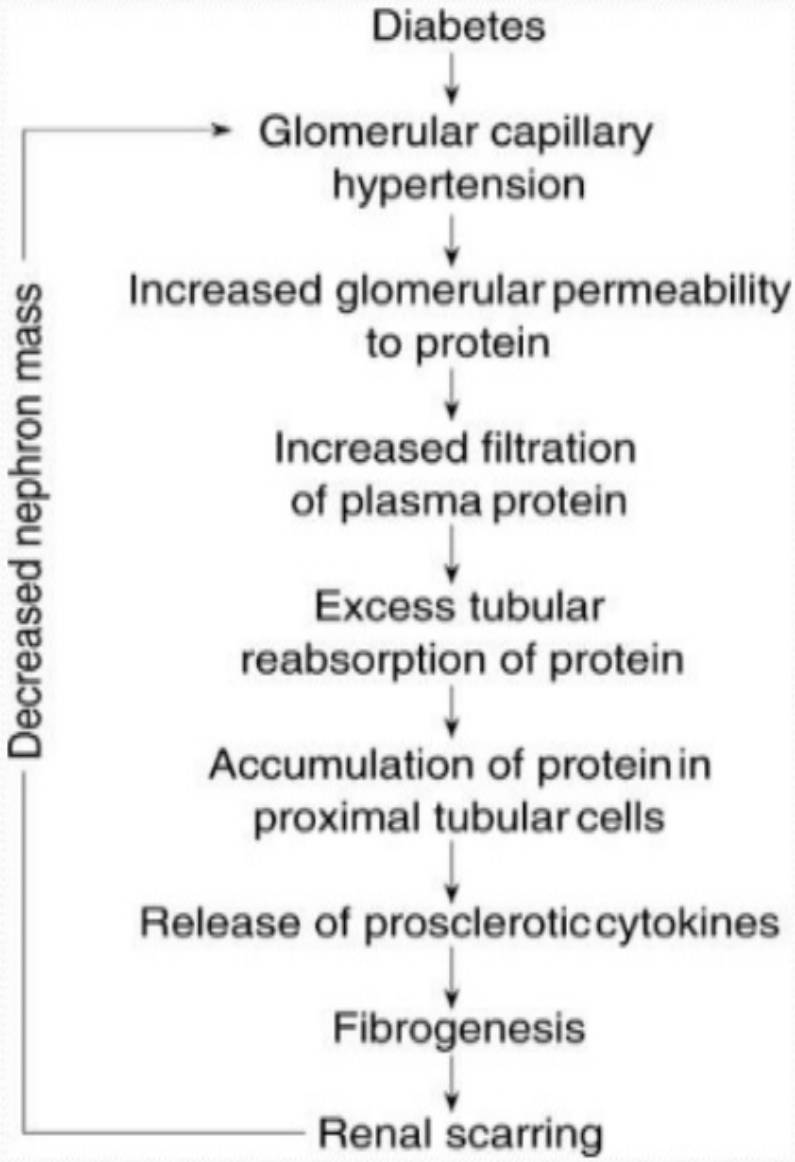
It results in formation of nodules called Kimmelstiel Wilson nodules

II – thickening of glomerular basement membrane

III – glomerular sclerosis caused by intraglomerular hypertension.


Diabetes diagnosis
Hyperfiltration
Clinical latency
Microalbuminuria
Macroalbuminuria
Renal failure

Proteinuria induced renal damage



CLINICAL FEATURES

Early in the course of disease –

- ✓ Asymptomatic

Established nephropathy

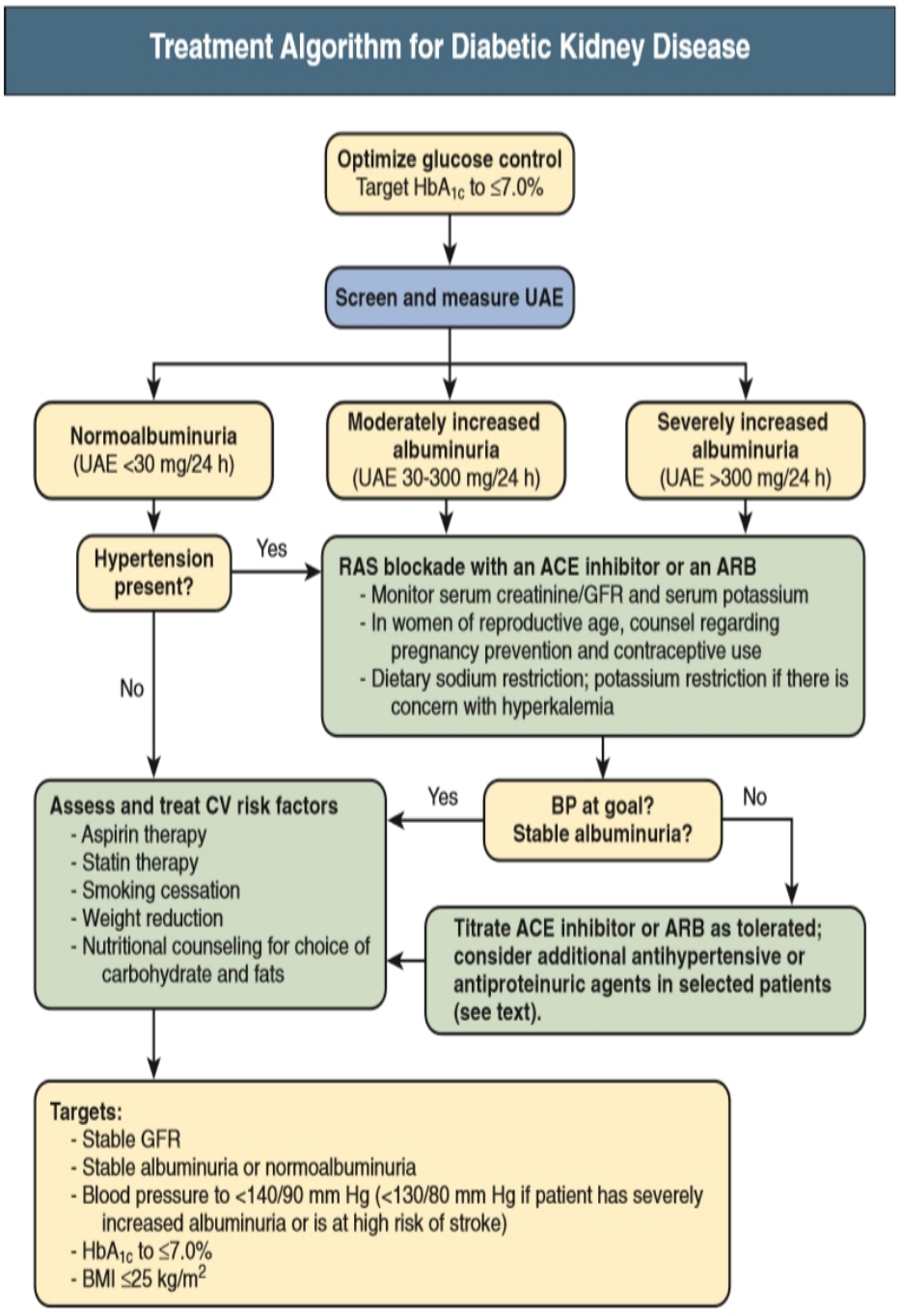
- ✓ fatigue
- ✓ Foamy urine
- ✓ Pedal edema
- ✓ Oliguria
- ✓ Anuria
- ✓ Puffiness of face
- ✓ Distension of abdomen

TREATMENT

- ✓ Control of blood sugar to near normal level
- ✓ Antihypertensive treatment
- ✓ Lipid lowering therapy
- ✓ Restriction of dietary protein
- ✓ Cessation of smoking

TREATMENT GOALS

- Glycaemic control
- Blood pressure control
- RAAS inhibition



RAAS INHIBITION

ACE inhibitors or ARB is recommended as first line therapy for diabetic nephropathy

Inhibition of RAAS slows the progression of diabetic nephropathy

GENERAL RECOMMENDATION

- ✓ The current recommendation by the national kidney foundation are to target BP of <130/80mmHg in diabetic patients
- ✓ Patient should adhere to a low sodium diet
- ✓ Use of diuretics may enhance the antiproteinuric effects of RAAS inhibition
- ✓ Beta adrenergic antagonist may be indicated in patients with arrythmia, CCF, CAD.
- ✓ Calcium channel blockers can be used in patients who lack these conditions.

NEW THERAPEUTICS FOR TREATMENT OF DIABETIC NEPHROPATHY[9]

AGENT	TARGET	EFFECT
Pentoxifylline	TNF-α blockade	Reduction of albuminuria in addition to ACE inhibitor/ARB
Pyridoxamine dihydrochloride (vitamin B6)	Advanced glycation end-product (AGE) inhibitor	Decreases AGE levels and ACR , and improves creatinine
Paracalcitriol (vitamin D)	Vitamin D	Reduction in ACR in diabetic nephropathy
Endothelin 1A antagonist (atrasentan)	Endothelin 1A receptor	Reduction in ACR, BP, and lipids in DN and nondiabetic CKD. Dose-related adverse effects fluid overload

AIMS AND OBJECTIVES

- To study the prevalence of retinopathy and nephropathy in newly diagnosed type 2 diabetes mellitus patients
- To study the relationship of development of nephropathy and retinopathy with various risk factors associated with type 2 diabetes mellitus like BMI, lipid profile , uric acid.

METHODOLOGY

Source of Data

Patients with newly diagnosed Diabetes mellitus presenting to department of medicine, Tirunelveli Medical College Hospital, Tirunelveli were taken for study. They came to physician either for routine health checkup or were admitted for some other diseases and diabetes mellitus was detected by chance for first time. The known cases of diabetes mellitus under treatment were excluded from study.

Sample Size: 100 cases

Sample procedure: Cross-sectional study

Study Duration: 1 year

INCLUSION CRITERIA

Newly diagnosed T2DM adult patients >20 years of age were included in the study.

According to ADA, criteria for diagnosis of T2DM are:

- Glycosylated hemoglobin (HbA1C) ≥6.5%.

OR

- Fasting plasma glucose ≥126 mg/dL (7.0 mmol/L).

OR

- 2-hour plasma glucose ≥200 mg/dL (11.1 mmol/L)

OR

- In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

Exclusion criteria

1. Congestive cardiac failure
2. Urinary tract infection
3. Known hypertensives
4. Fever
5. Renal diseases
6. Type 1 diabetes mellitus
7. Pregnancy
8. Refusal to be a part of the study

Method of collection of data Clinical history

- Detailed history regarding the symptoms of diabetes like polyuria, polydipsia, polyphagia and weight loss were taken.
- History of microvascular complications were taken in detail.

Diabetic Retinopathy

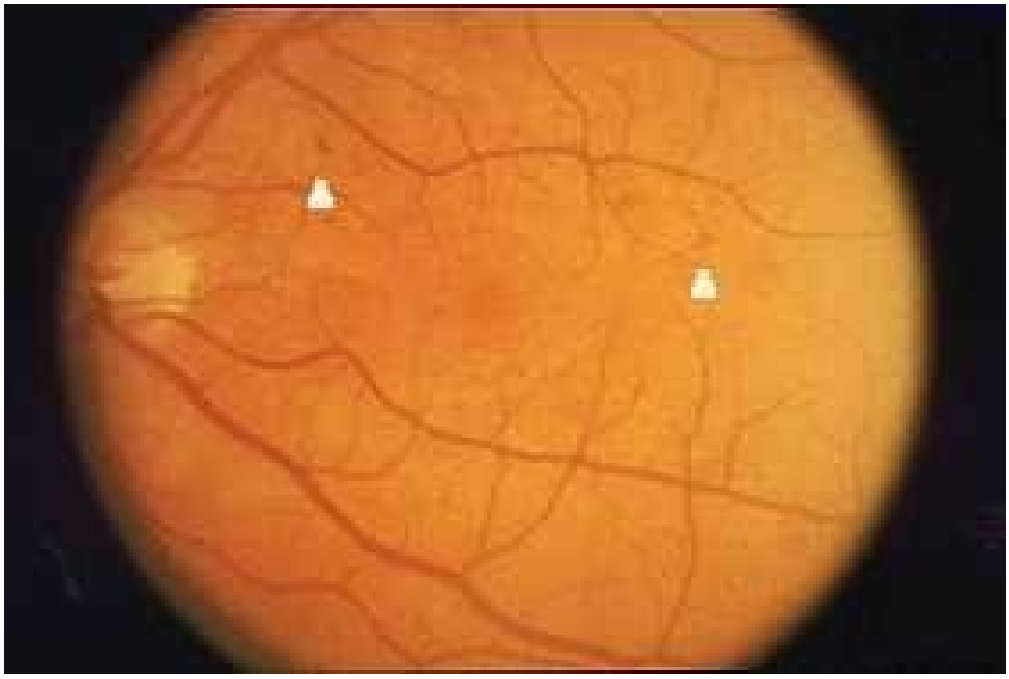
History of blurred vision, black spots, floaters and sudden visual loss

Diabetic Nephropathy

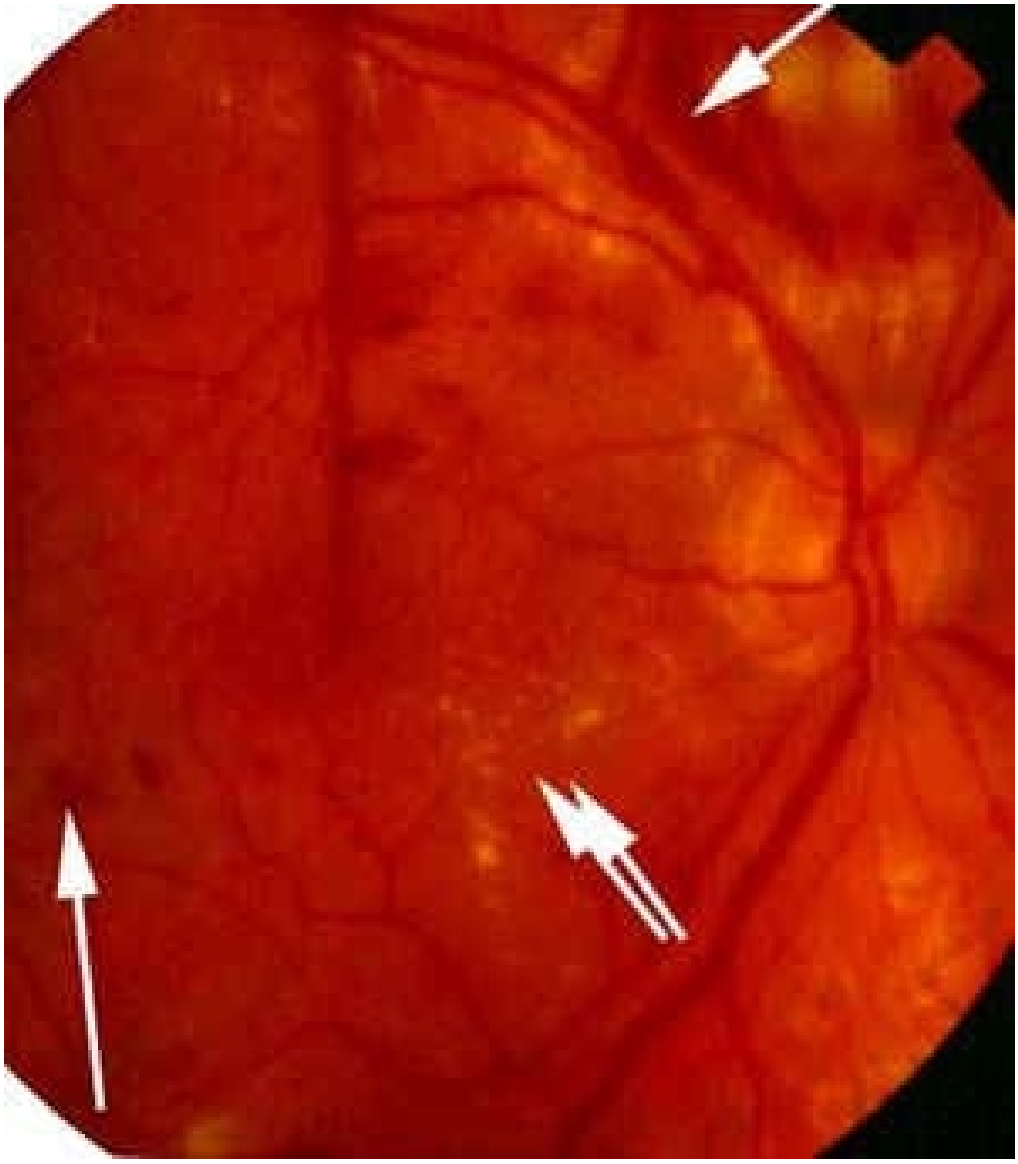
History of polyuria, oliguria, puffiness of face, distension of abdomen and pedal edema.

Direct ophthalmoscopic examination of fundus

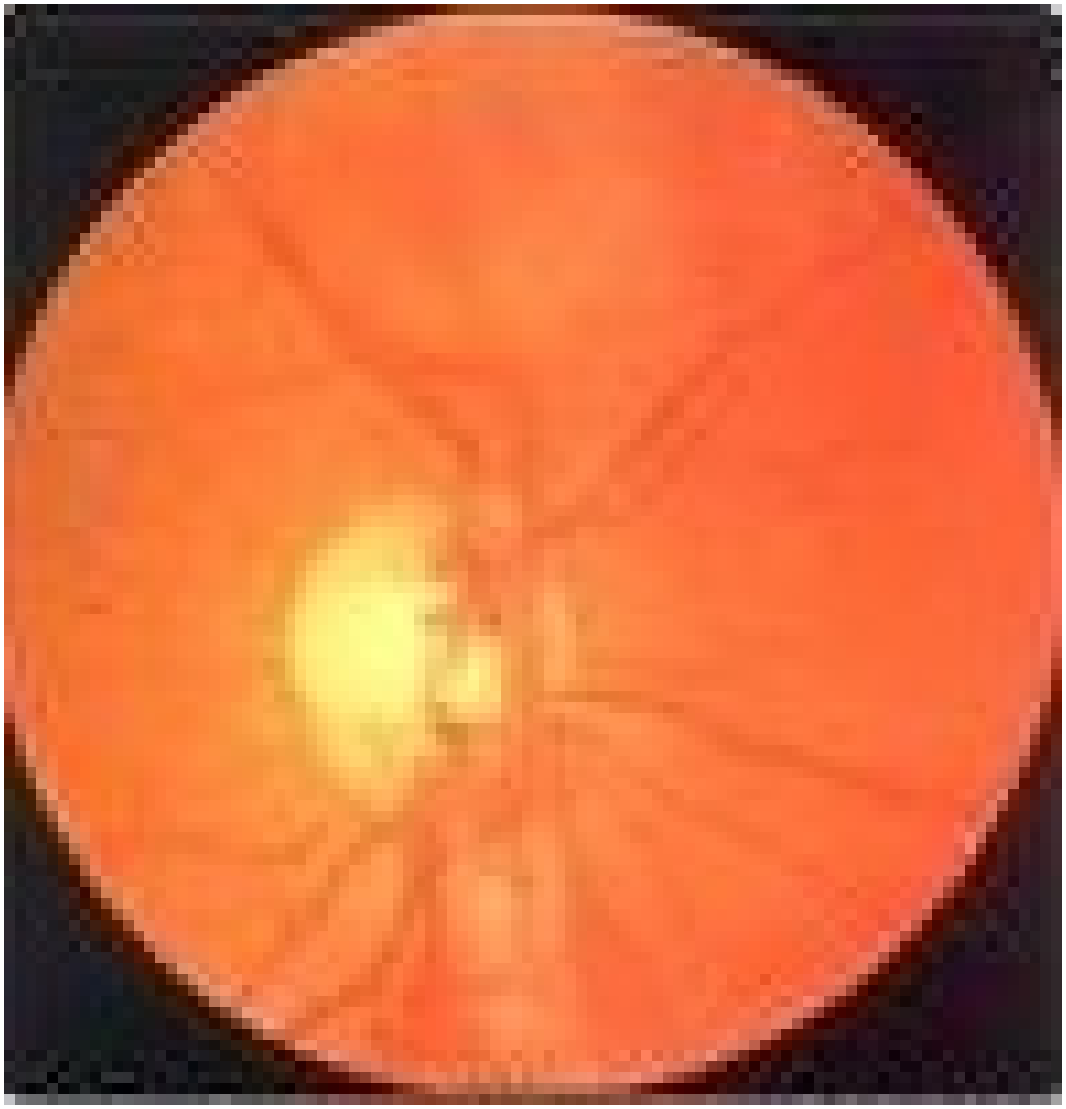
Fundus examination was done and results were classified as Normal, Non-proliferative and proliferative retinopathy. It was confirmed by ophthalmologist.



Background diabetic retinopathy



Preproliferative Diabetic Retinopathy



Proliferative Diabetic Retinopathy

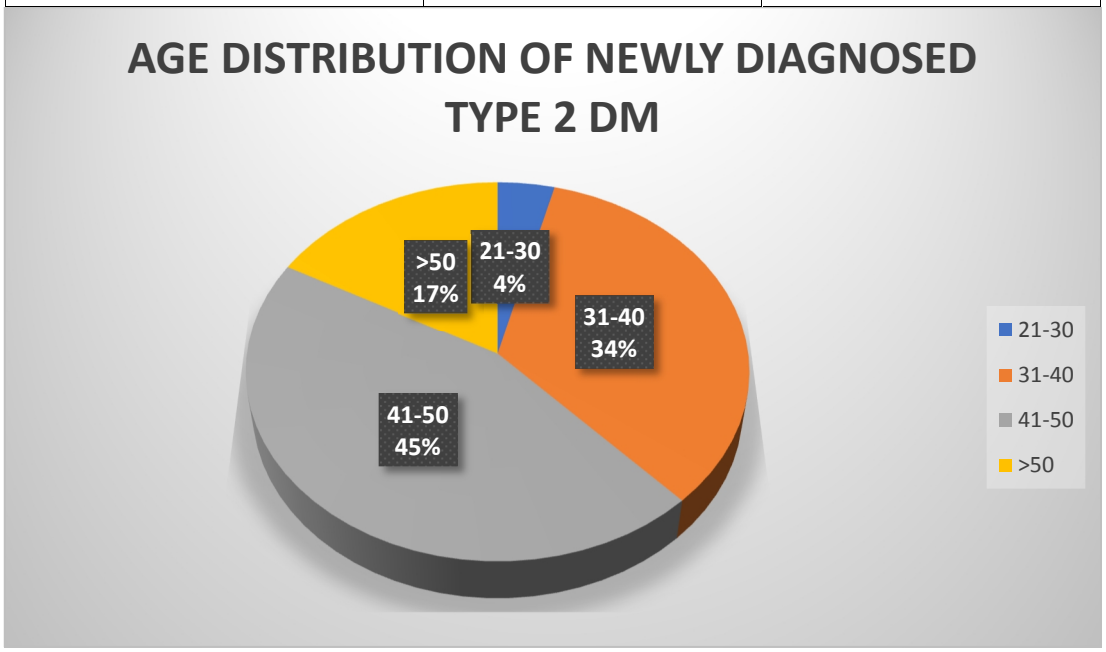
Investigations

1. Fasting plasma glucose
2. Postprandial plasma glucose
3. Blood routine
4. Urine for proteinuria
5. Blood urea & Serum Creatinine
6. Proteinuria detection by protein Creatinine Ratio estimation.

RESULTS

AGE DISTRIBUTION OF NEWLY DIAGNOSED TYPE 2 DM

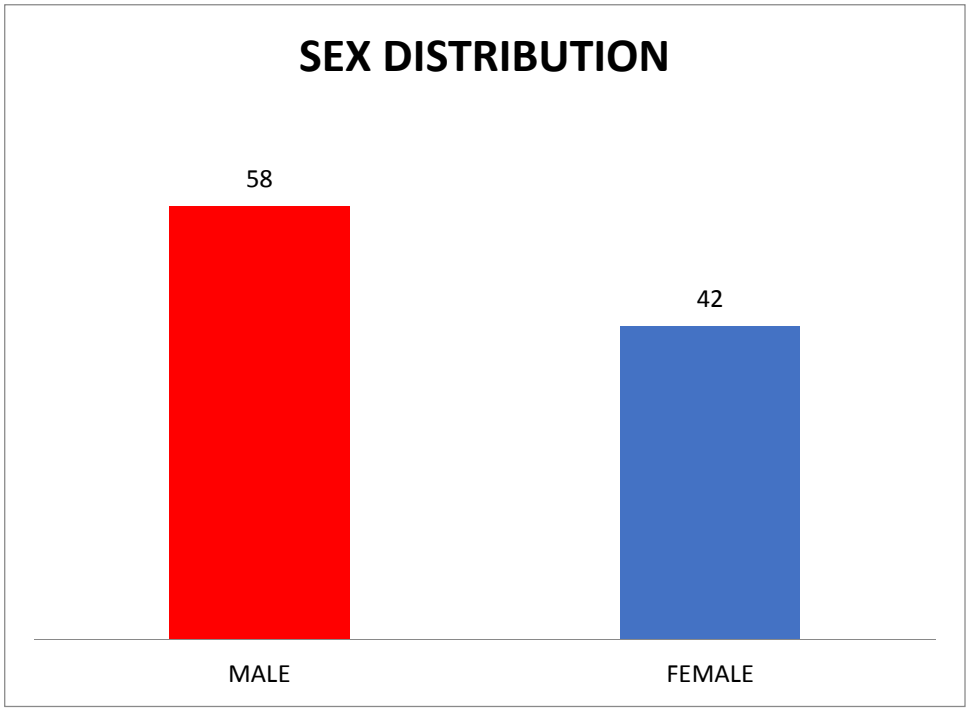
AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
21-30	4	4%
31-40	34	34%
41-50	45	45%
>50	17	17%



Above data suggests that out of total 100 patients with newly diagnosed diabetes, 45% of the patients were within age of 41-50 years, 34% of them were within 31-40 years. So maximum number of patients were clustered between 31-50 years of age (34%+45%=79%).

SEX DISTRIBUTION OF NEWLY DIAGNOSED TYPE 2 DM

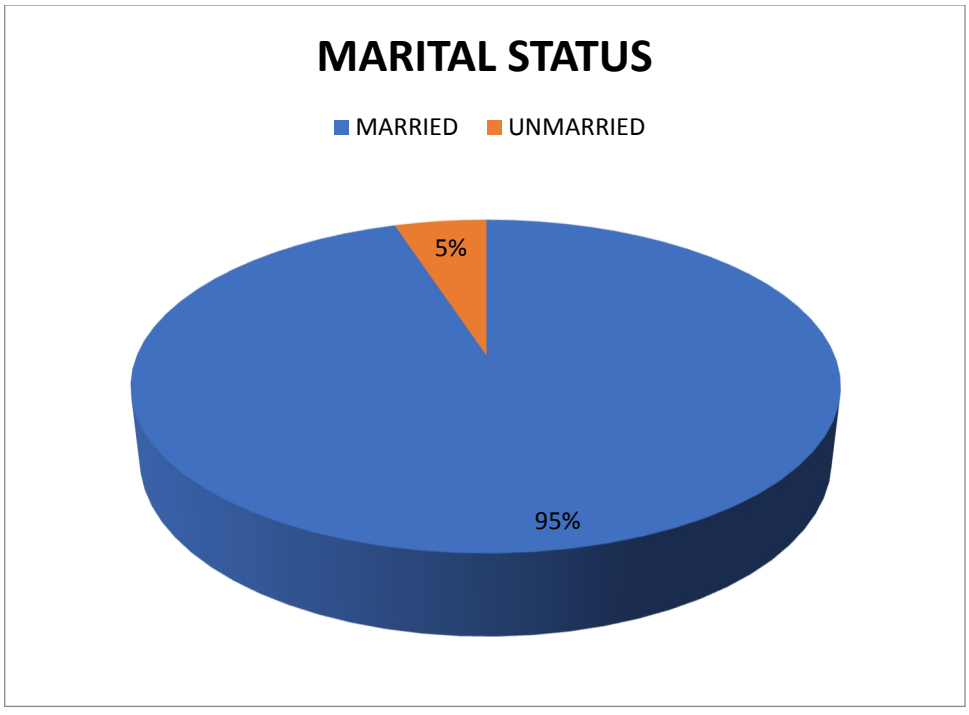
SEX	NO OF PATIENTS	PERCENTAGE
MALE	58	58%
FEMALE	42	42%



Above data suggests that out of 100 patients with newly diagnosed type2 diabetes mellitus,58% were males (58 males) and 42% were females (42 females)

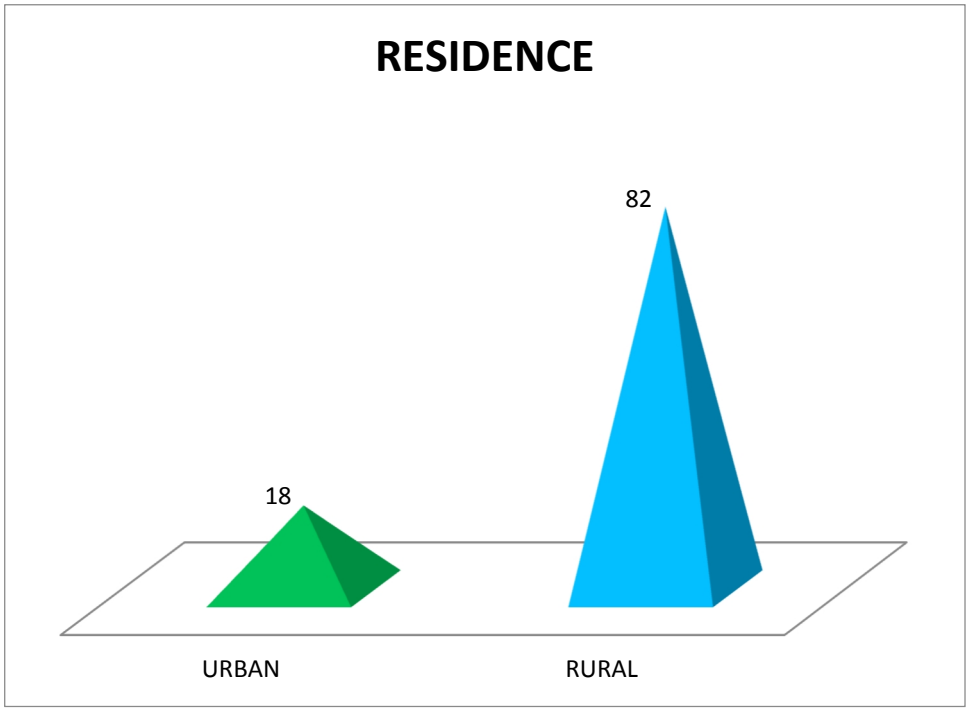
MARITAL STATUS

MARITAL STATUS	NO OF PATIENTS	PERCENTAGE
MARRIED	95	95%
UNMARRIED	5	5%



RESIDENCE

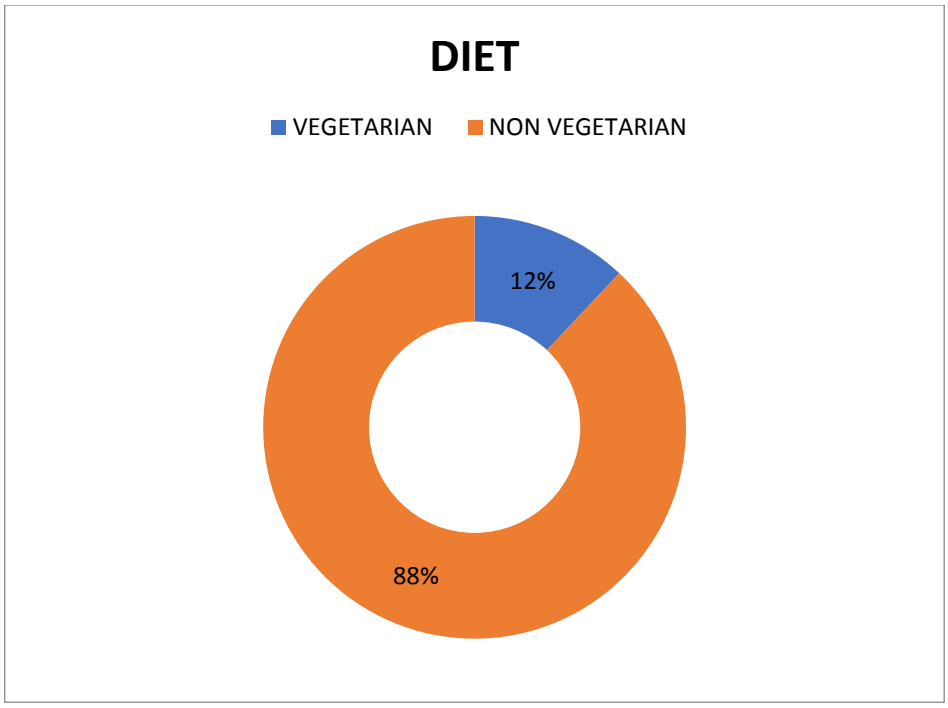
RESIDENCE	NO OF PATIENTS	PERCENTAGE
URBAN	18	18%
RURAL	82	82%



Above data suggests that out of total 100 patients with newly diagnosed type2 diabetes mellitus, 82% were residing in rural areas.

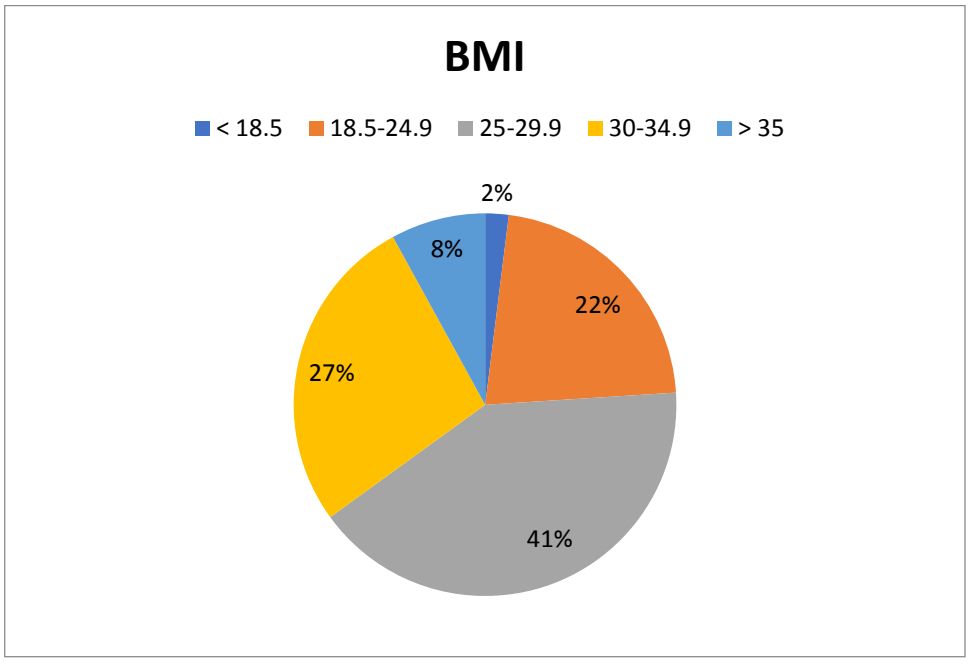
DIET

DIET	NO OF PATIENTS	PERCENTAGE
VEGETARIAN	12	12%
NON VEGETARIAN	88	88%



DISTRIBUTION OF BODY MASS INDEX IN NEWLY DIAGNOSED TYPE 2 DM PATIENTS

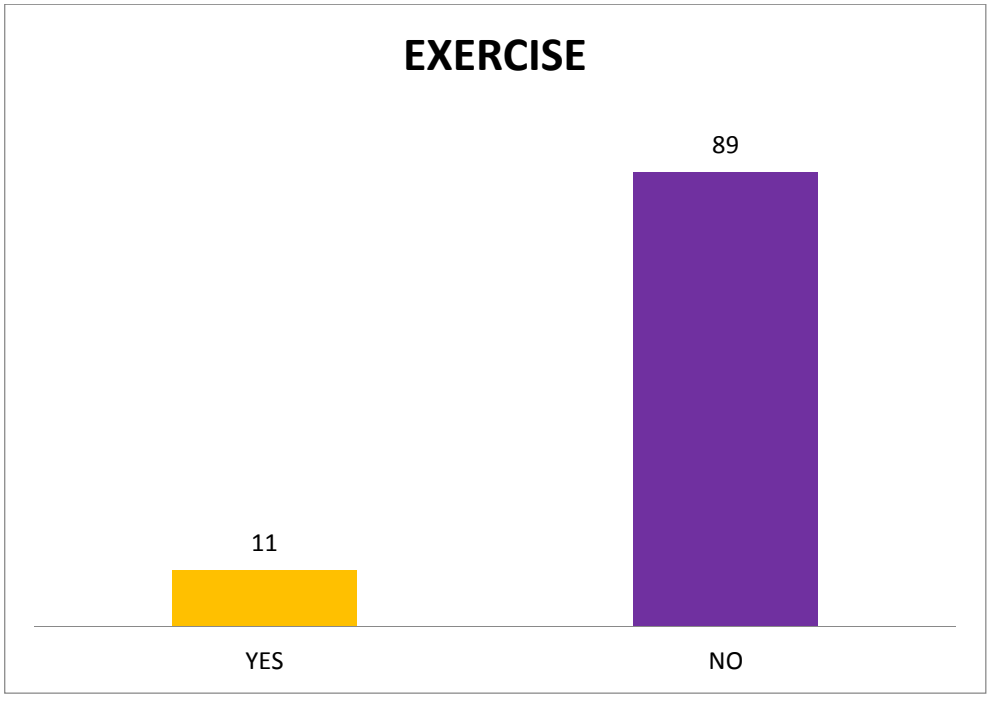
BODY MASS INDEX	NO OF PATIENTS	PERCENTAGE
< 18.5	2	2%
18.5-24.9	22	22%
25-29.9	41	41%
30-34.9	27	27%
> 35	8	8%



Out of 100 newly diagnosed type2 diabetes, 41% of the patients were overweight, 27% of the patients were obese.

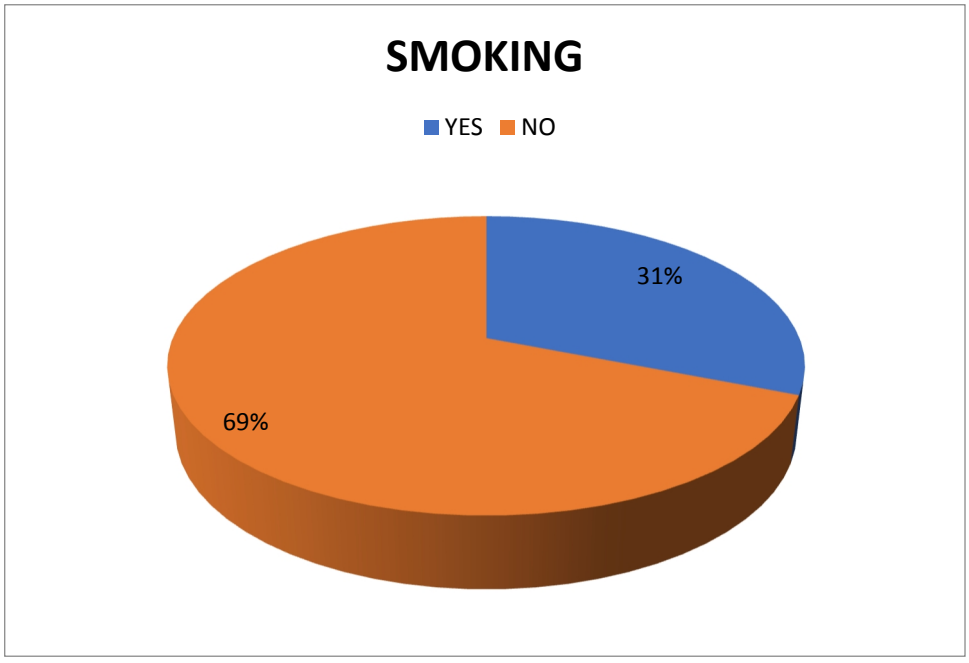
EXERCISE

EXERCISE	NO OF PATIENTS	PERCENTAGE
YES	11	11%
NO	89	89%



PREVALENCE OF SMOKING IN NEWLY DIAGNOSED TYPE 2 DM

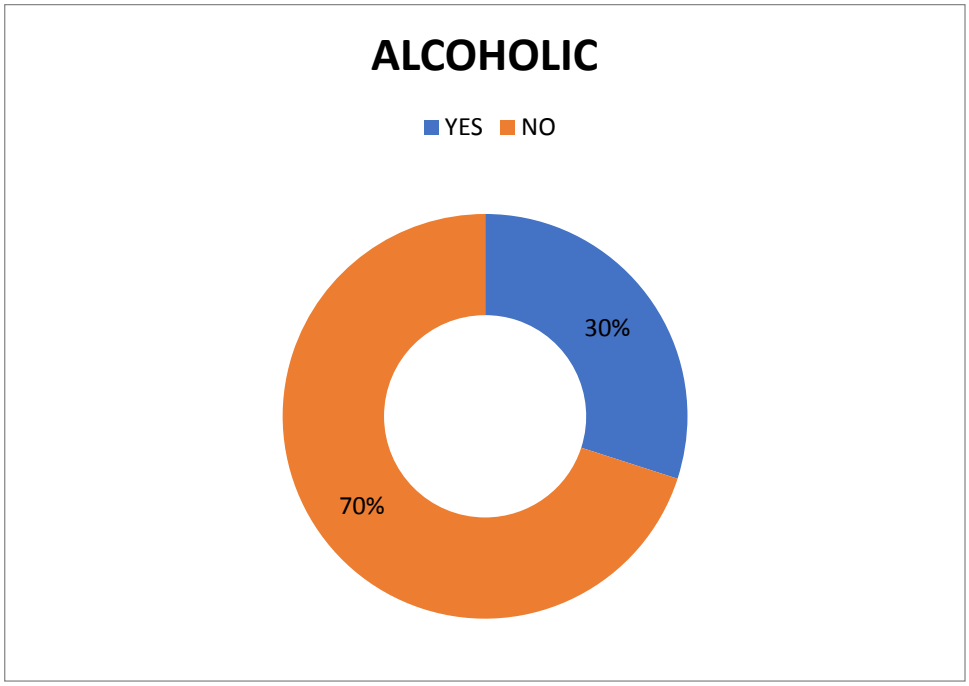
SMOKING	NO OF PATIENTS	PERCENTAGE
YES	31	31%
NO	69	69%



Out of 100 newly diagnosed type2 diabetes, 31% of the patients were smokers, 69% of the patients were non smokers .

PREVALENCE OF ALCOHOLIC IN NEWLY DIAGNOSED TYPE 2 DM

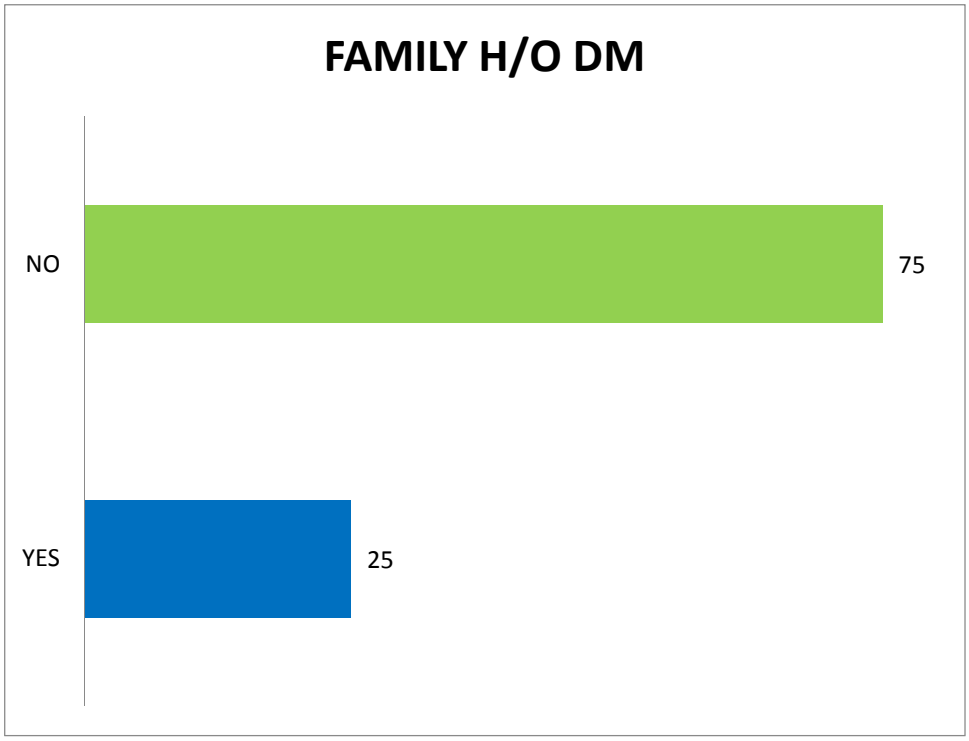
ALCOHOLIC	NO OF PATIENTS	PERCENTAGE
YES	30	30%
NO	70	70%



Out of 100 newly diagnosed type2 diabetes, 70% of the patients were non alcoholic.

FAMILY HISTORY

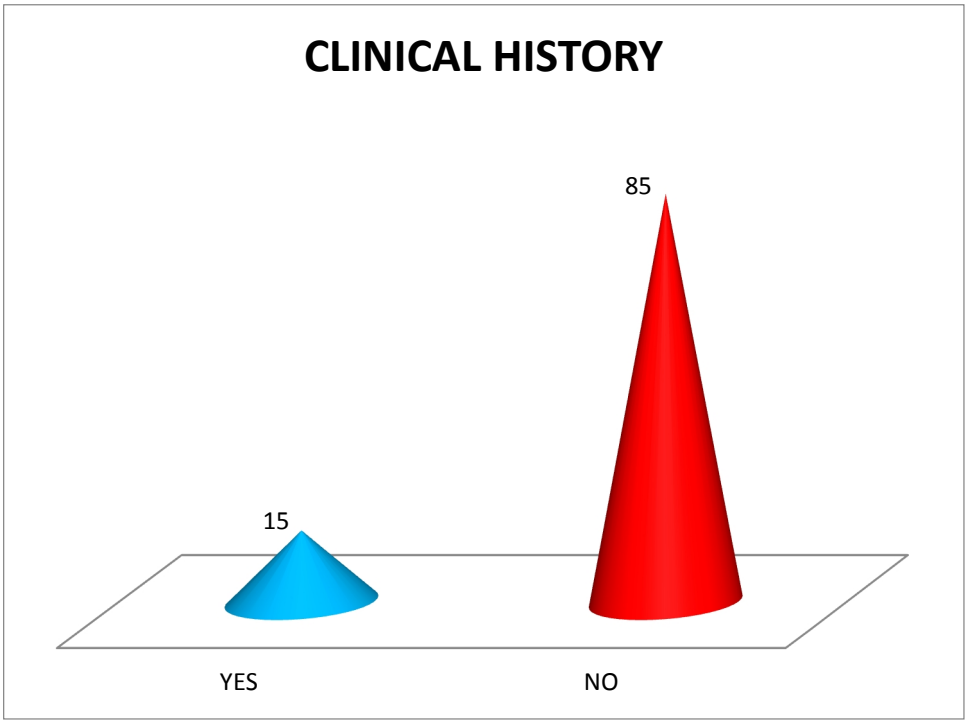
FAMILY H/O DM	NO OF PATIENTS	PERCENTAGE
YES	25	25%
NO	75	75%



75% of the patients had no family history of type2 diabetes mellitus.

CLINICAL HISTORY

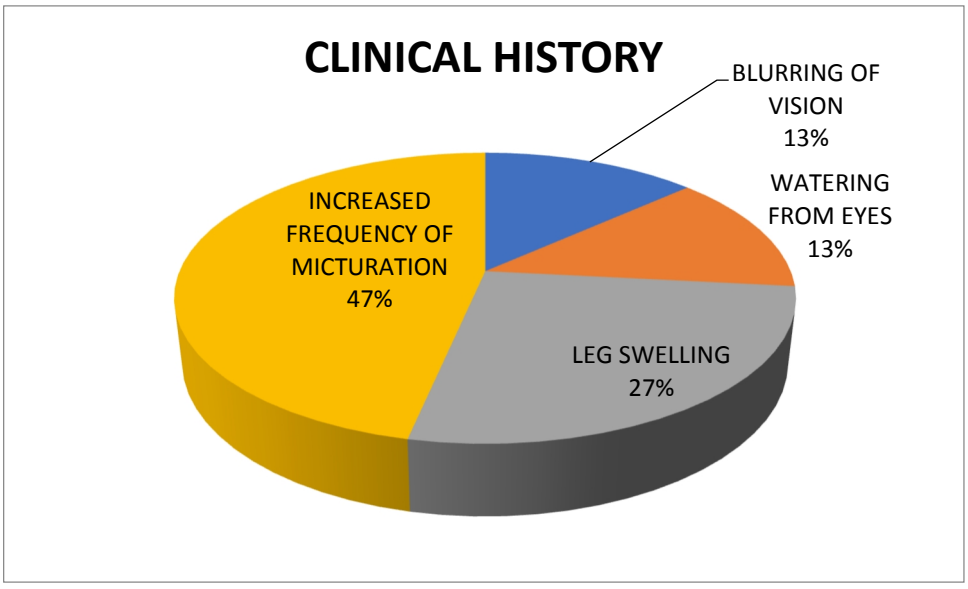
CLINICAL HISTORY	NO OF PATIENTS	PERCENTAGE
YES	15	15%
NO	85	85%



Out of 100 newly diagnosed type2 diabetes, 85% of the patients had no significant clinical history pertaining to diabetic nephropathy and retinopathy.

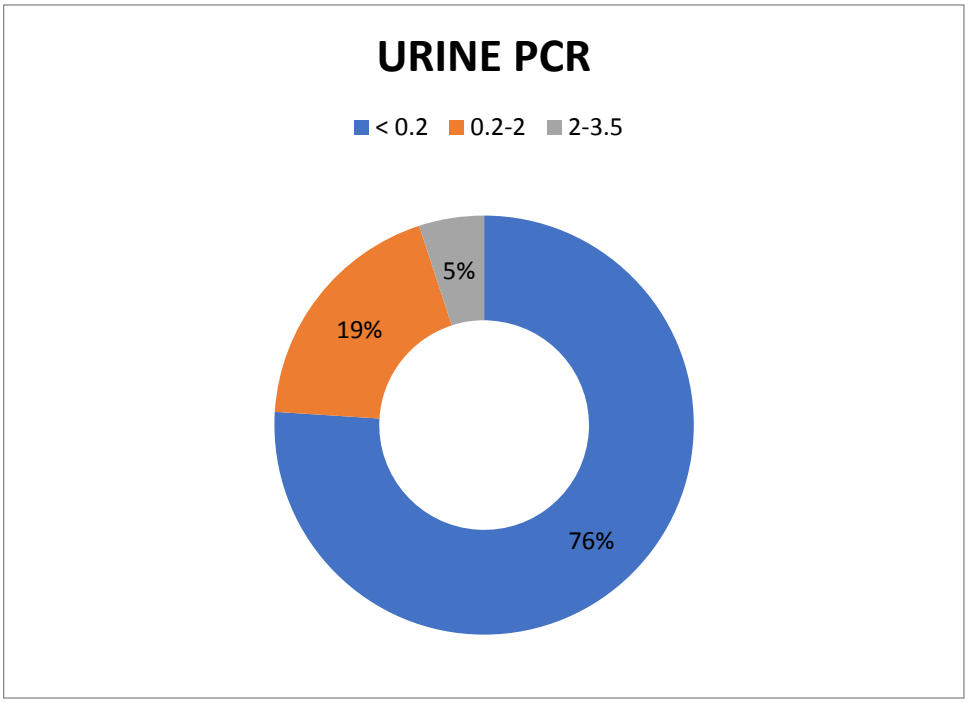
CLINICAL HISTORY

CLINICAL HISTORY	NO OF PATIENTS	PERCENT AGE
BLURRING OF VISION	2	13%
WATERING FROM EYES	2	13%
LEG SWELLING	4	27%
INCREASED FREQUENCY OF MICTURITION	7	47%



DISTRIBUTION OF URINE PROTEIN CREATININE RATIO IN NEWLY DIAGNOSED TYPE2 DM

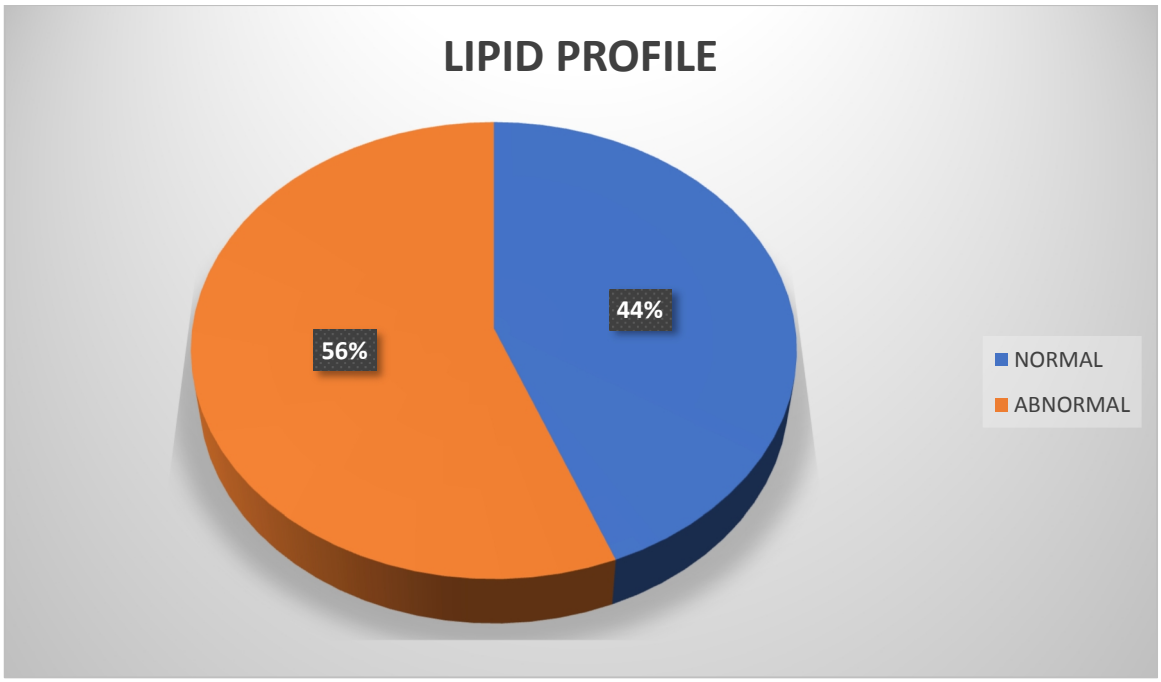
URINE PCR	NO OF PATIENTS	PERCENTAGE
< 0.2	76	76%
0.2-2	19	19%
2-3.5	5	5%



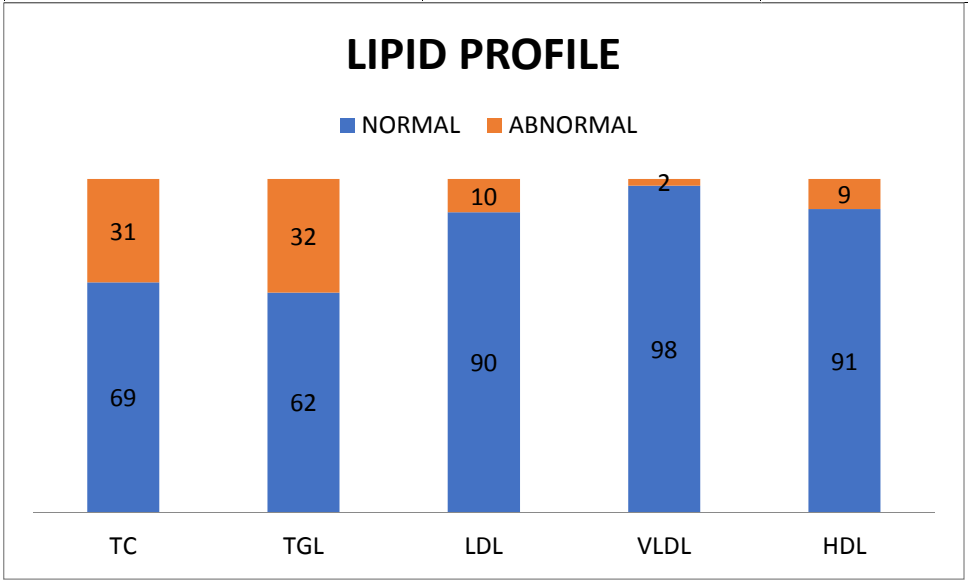
Above data suggests that out of 100 newly diagnosed type2 diabetes, 24% of the patients had significant proteinuria.

DISTRIBUTION OF LIPID PROFILE IN NEWLY DIAGNOSED TYPE2 DM

LIPID PROFILE	NO OF PATIENTS	PERCENTAGE
NORMAL	44	44%
ABNORMAL	56	56%



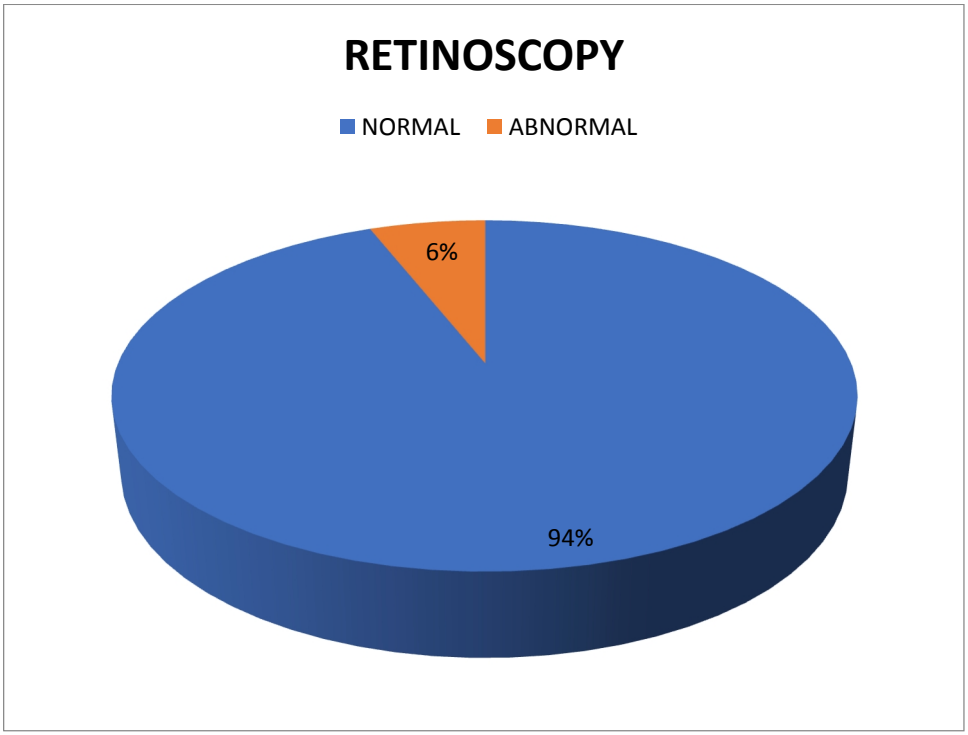
LIPID PROFILE	NORMAL	ABNORMAL
TC	69	31
TGL	62	38
LDL	90	10
VLDL	98	2
HDL	91	9



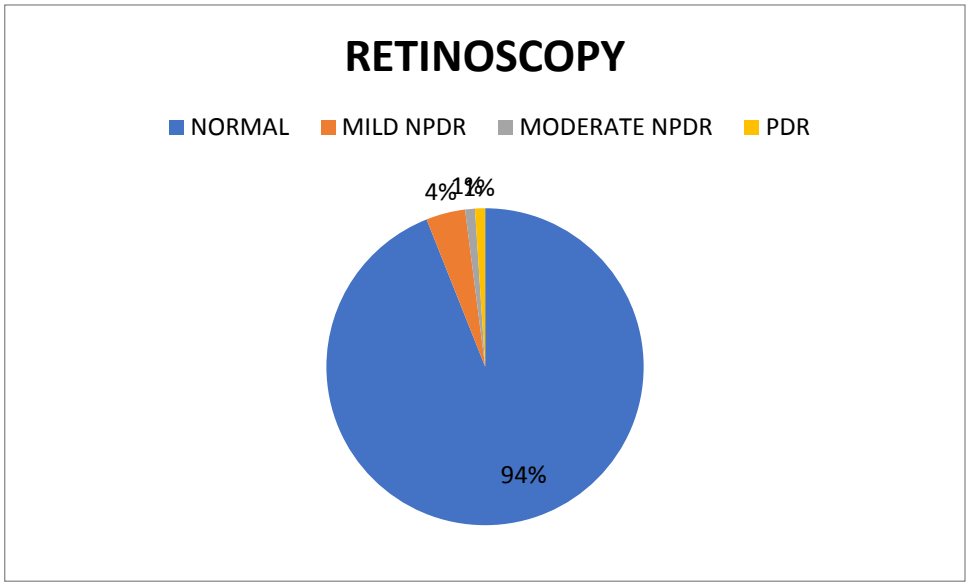
56% of the patients had abnormal lipid profile, among the patients with abnormal lipid profile 38% had hypertriglyceridemia, 31% had hypercholesterolemia.

PREVALENCE OF RETINOPATHY IN NEWLY DIAGNOSED TYPE2 DM

RETINOSCOPY FINDINGS	NO OF PATIENTS	PERCENTAGE
NORMAL	94	94%
ABNORMAL	6	6%



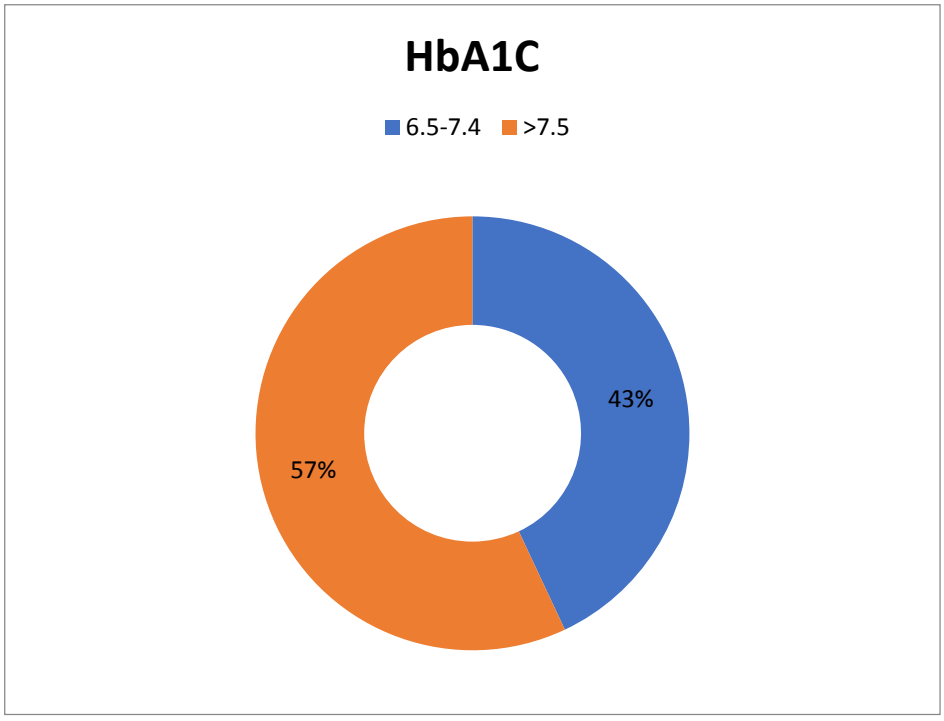
RETINOSCOPY FINDINGS	NO OF PATIENTS	PERCENTAGE
NORMAL	94	94%
MILD NPDR	4	4%
MODERATE NPDR	1	1%
PDR	1	1%



From the data, 6% of patients had abnormal retinoscopy findings, of which 4 patients had mild NPDR.

DISTRIBUTION OF HbA1C IN NEWLY DIAGNOSED TYPE2 DM

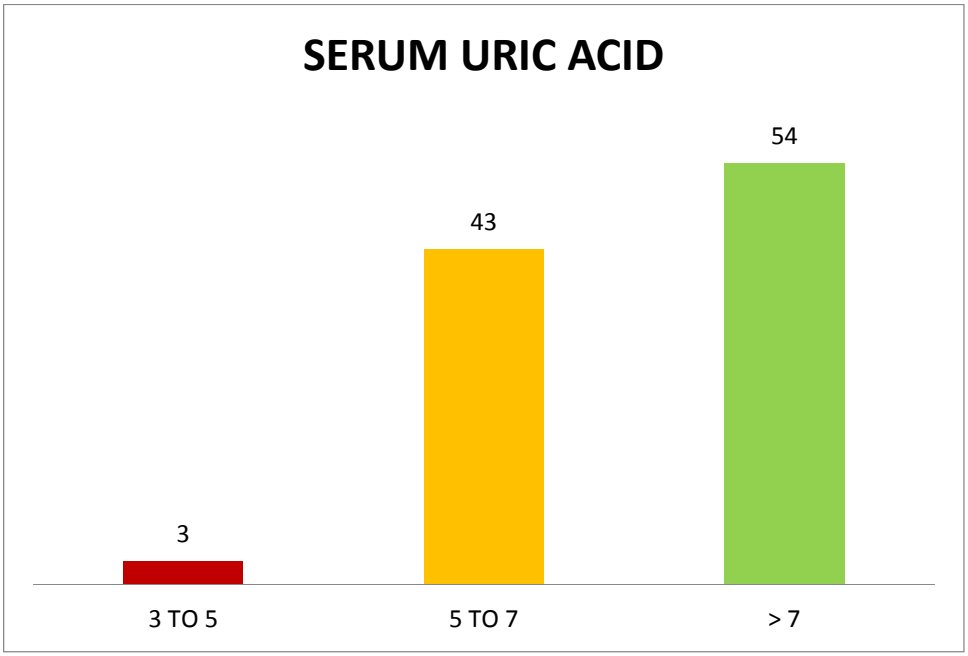
HBA1C	NO OF PATIENTS	PERCENTAGE
6.5-7.4	43	43%
>7.5	57	57%



From the above data 57% of the patients had HbA1C >7.5%

DISTRIBUTION OF SERUM URIC ACID IN NEWLY DIAGNOSED TYPE2 DM

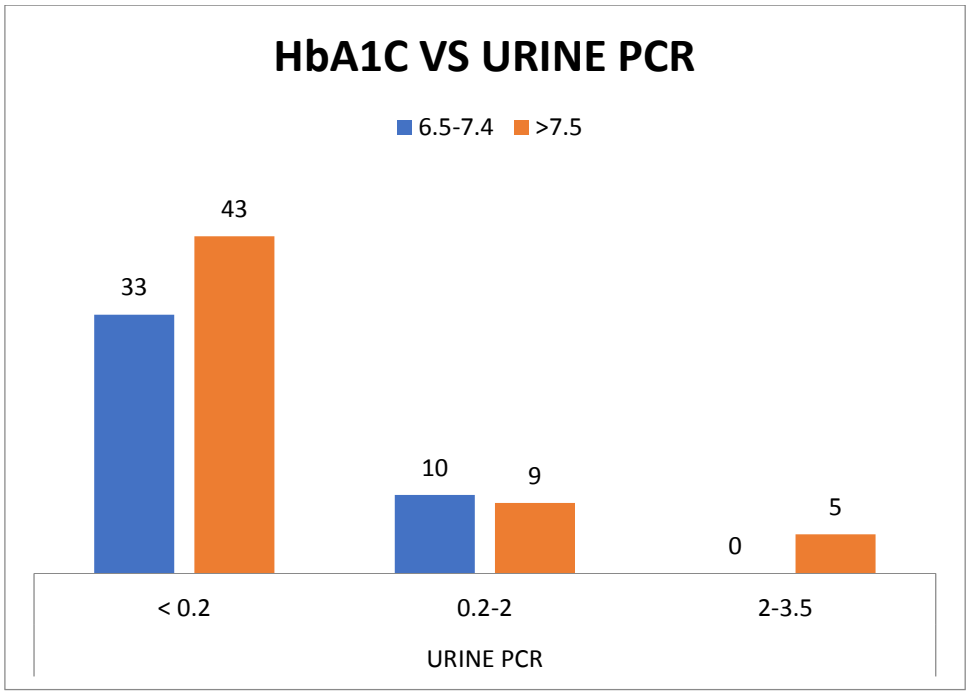
SERUM URIC ACID	NO OF PATIENTS	PERCENTAGE
3 TO 5	3	3%
5 TO 7	43	43%
> 7	54	54%



From the above data 54% of the patients had hyperuricemia.

RELATIONSHIP BETWEEN HbA1C AND URINE PCR

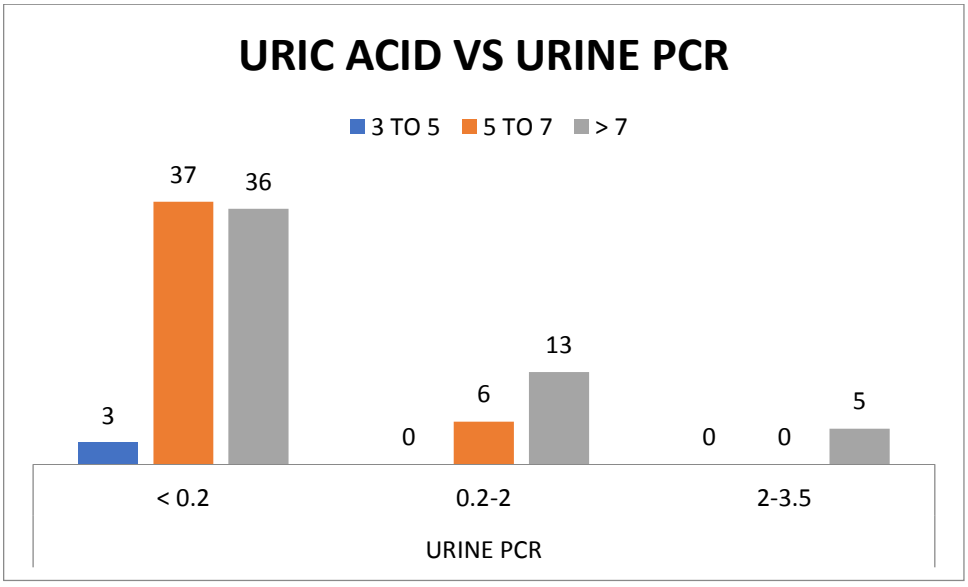
HBA1C	URINE PCR		
	< 0.2	0.2-2	2-3.5
6.5-7.4	33	10	0
>7.5	43	9	5



From the above data it is evident that out of 24 patients with proteinuria, 14 patients had HbA1C > 7.5%

RELATIONSHIP BETWEEN SERUM URIC ACID AND URINE PCR

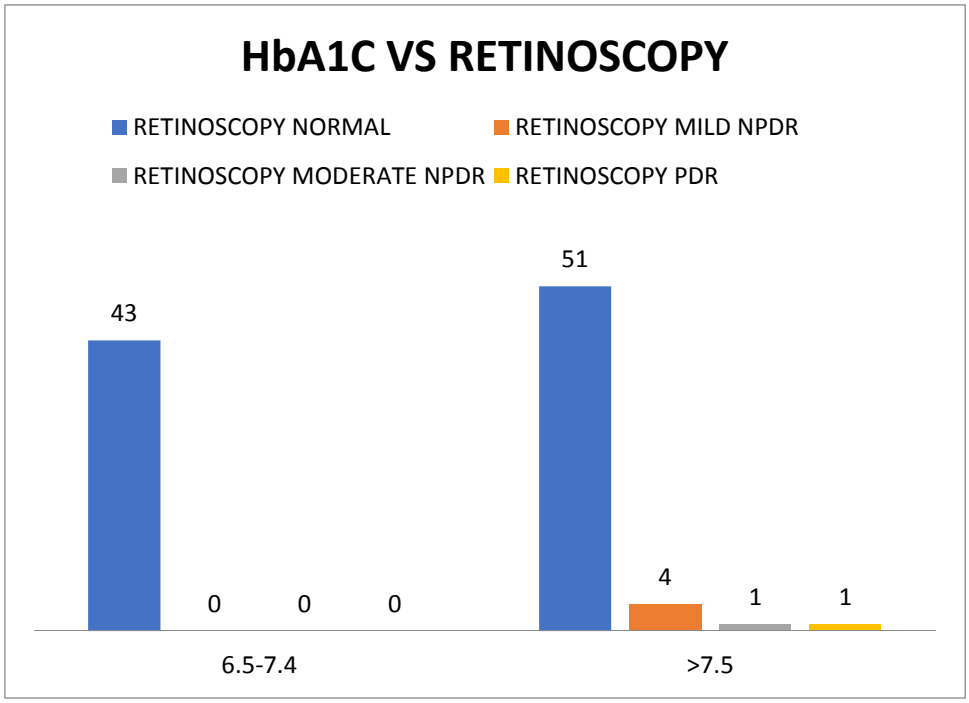
SERUM URIC ACID	URINE PCR		
	< 0.2	0.2-2	2-3.5
3 TO 5	3	0	0
5 TO 7	37	6	0
> 7	36	13	5



From the above data it is evident that out of 24 patients with proteinuria, 18 patients had hyperuricemia.

RELATIONSHIP BETWEEN HbA1C AND DIABETIC RETINOPATHY

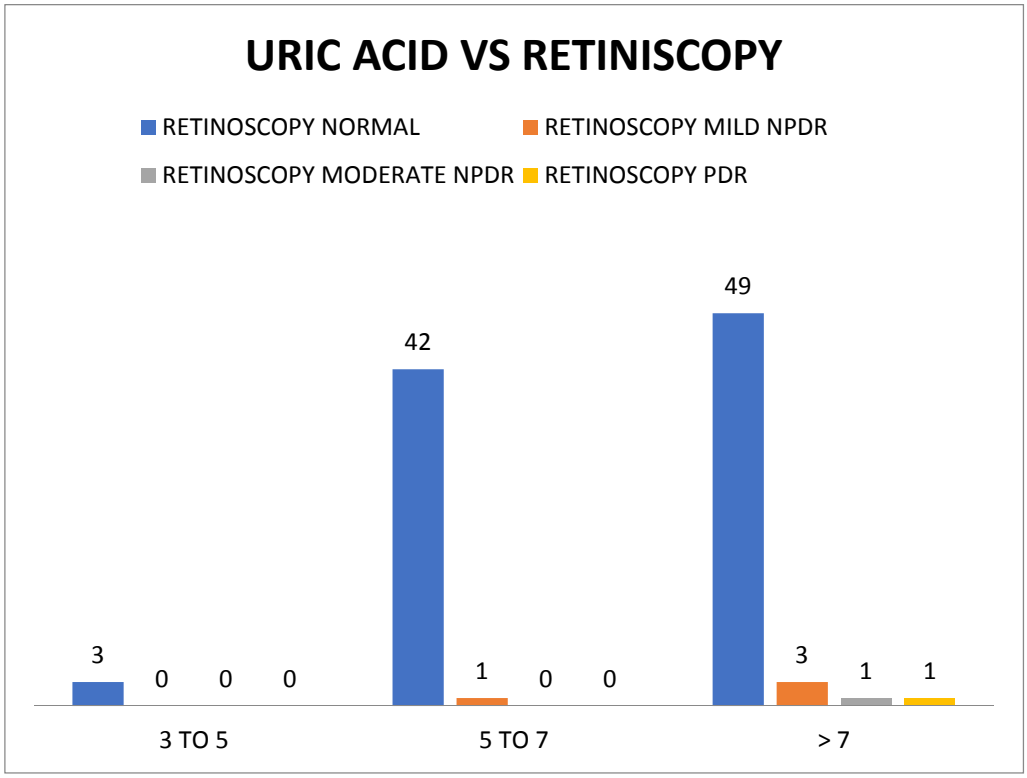
HBA1C	RETINOSCOPY			
	NORMAL	MILD NPDR	MODERATE NPDR	PDR
6.5-7.4	43	0	0	0
>7.5	51	4	1	1



From the above data out of 57 patients with HbA1C >7.5% , 6 patients had diabetic retinopathy.

RELATIONSHIP BETWEEN SERUM URIC ACID AND DIABETIC RETINOPATHY

SERUM URIC ACID	RETINOSCOPY			
	NORMAL	MILD NPDR	MODERATE NPDR	PDR
3 TO 5	3	0	0	0
5 TO 7	42	1	0	0
> 7	49	3	1	1



The above data suggests that out of 6% patients with retinopathy 5 patients had hyperuricemia.

DISCUSSION

Type 2 diabetes mellitus is an insidious illness with asymptomatic phase of many years during which body is exposed to ill effects of asymptomatic hyperglycemia. This study has confirmed that a large proportion of patients with type2 diabetes mellitus has developed diabetic nephropathy and retinopathy even before the time of diagnosis.

In our study mean age of our patient was 40 years, which confirms that in developing countries majority of patients with diabetes are in young, productive age group(31-50 years) as compared to developed countries who develop diabetes mellitus at a higher age (>65 years) as found in Wild S , Roglic G et al. and Ramachandran A et al. studies[10,11] .

In the our 100 patients evaluated for complication profile nephropathy was more common (24%) when compared to retinopathy (6%).

Previously a study by Mohan, et al[12].has shown that nephropathy is present in about 15-18% of patients with newly diagnosed type2 diabetes mellitus. Our study shows an increased percentage of patients (24%)

presenting with diabetic nephropathy at the time of diagnosis of diabetes mellitus

In our study we have found that lipid profile is abnormal in 56% of patients with newly diagnosed type2 diabetes mellitus of the 56% of patients 38 patients had hypertriglyceridemia and 31 patients had hypercholesterolemia.

In our study , we found that diabetic retinopathy as the least common microvascular complications(6%). This is in accordance with Sosale et al[13]. study which showed 6% patients had diabetic retinopathy .

CONCLUSION

Microvascular complications like diabetic retinopathy and diabetic nephropathy are a major cause of morbidity and mortality in type2 diabetes mellitus. In our study 24 out of 100 newly diagnosed type2 diabetes mellitus were presented with microvascular complications.

In conclusion, diabetic retinopathy is associated with vision threatening complication. It can be made out even at the time of diagnosis of type2 diabetes mellitus. So, vision loss due to diabetic retinopathy is preventable through strict glycemic control and routine fundoscopic examination by an ophthalmologist.

Early treatment helps to stabilize the visual acuity and prevent further loss.

Proteinuria may also be a marker of severe disease with widespread vascular damage. The proteinuria appears to be an important marker of greater diagnostic and prognostic importance .

So screening for early detection and identification of risk factors for nephropathy and retinopathy may prevent the progression of microvascular complications.

BIBILOGRAPHY

1. Harris MI, Klein R, Welborn TA, onset of NIDDM occurs atleast 4-7 years before clinical diagnosis. Diabetes care 1992; 15: 815-819.

2. Reema M, Deepa R, Mohan V. Prevalence of retinopathy at diagnosis among Type 2 DM patients attending diabetic centre in south India. Br J Ophalmol 2000; 84: 1058-1060.

3. Koher EM, Aldengton SJ, Manley SF, Mathews DR. et al. diabetic retinopathy at diagnosis of NIDDM and associated risk factors. Arch Ophthalmol 1998; 116:297-303.

4. Thomas H.Hostetter: "Diabetic nephropathy"Chapter-34 in The Kidney,Brenner and Rector (Eds) IV Edn, vol. II, Philadelphia. W.B.Saunders Company, 1991: 1695-1791pp.

5. Leo P. Creal, Donald Bornett, Rachmid Levine: "The history of diabetes", Chapter -1 in Joslins Diabetes mellitus, E.Ronald Kahn, Gordon C.Weir(Eds),New Delhi , B.I Waverly Pvt Ltd.1994;1 :13pp

6. Schadewaldt H: "History of Diabetes Mellitus" Chapter-, in

Diabetes, its Medical and cultural history, Van Englehardt D.(Ed), Berlin , Springer Verlag.

7. 7.Thomas N, Jeyaraman K, Asha HS, Velavan J. A practical guide to diabetes mellitus. JP Medical Ltd; 2012 Oct 30.

8. Mogensen CE: Definition of diabetic renal disease in insulin-dependent diabetes mellitus based on renal function tests. In Mogensen CE (ed). The Kidney and Hypertension in Diabetes Mellitus, 5th ed. Boston, Kluwer, 2000, pp 1328.

9. Taal MW, Chertow GM, Marsden PA, Skorecki K, Alan SL, Brenner BM. Brenner and Rector's The Kidney E-Book. Elsevier Health Sciences; 2011 Nov 1.

10. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27:1047-53

11. Ramachandran A. Socio-economic burden of diabetes in India. J Assoc Physicians India 2007;55 Suppl:9-12

12. mohan, EA, monagham B : Nephropathy in newly diagnosed type 2 diabetes. Diabetes cone 1994;17:1545–6.

13. Sosale A, Prasanna Kumar KM, Sadikot SM, Nigam A, Bajaj S, Zargar AH, et al. Chronic complications in newly diagnosed patients with Type 2 diabetes mellitus in India. Indian J Endocrinol Metab

2014;18:355-60.

14. ADA- Diabetes Care. 2010 January; 33(Supplement_1): S62S69. doi: 10.2337/dc10-S062

15. Margolis S. Diabetic microvascular complications: An overview. Adv Stud Med 2005;5:S260-3.

PROFORMA

1. Name : Mr/Mrs
2. Age: 1)20-30 2)31-40 3)41-50 4)>50
3. Sex: 1)Male 2)Female
4. Marital status: 1)Married 2) Unmarried
5. Residence: 1)Urban 2) Rural
6. Dietery habit of patient: 1) Vegetarian 2) Non – Vegetarian
7. Body Mass index of patient 1)<18.5 2)18.5-24.9 3)25-29.9 4)30-34.9 5)>35
8. Will you do exercise daily 1)Yes 2)No
9. Do you have habit of smoking? 1) Yes 2)No
10. Do you consume alcohol? 1)Yes 2) No
11. Family H/o diabetes mellitus 1)Yes 2) No
12. Clinical History 1)Yes 2) No

		Yes	No
1.a)	Blurring of vision	☐	☐
1.b)	Watering from the eyes	☐	☐
1.c)	Leg swelling	☐	☐
1.d)	Increased frequency of micturition	☐	☐
1.e)	Vomiting	☐	☐
1.f)	H/o floaters	☐	☐
1.g)	H/o Flashes of light	☐	☐

13. Urine PCR

    1) <0.2 2) 0.2 – 2 3) 2-3.5 4)>3.5

14. Lipid profile

    1) Normal 2) Abnormal

If Abnormal

2.a) Cholesterol

2.b) TG

2.c) LDL

2.d) VLDL

2.e) HDL

15. Retinoscopy findings

1. Normal
2. Mild NPDR
3. Moderate NPDR
4. Severe NPDR
5. PDR
6. CSME

16. HbA_1C

1) <5.6 2) 5.7 – 6.4 3) 6.5-7.4 4) ≥7.5

17. Serum Uric acid

1) <3 2) 3-5 3) 5-7 4) >7

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

KEY TO MASTER CHART

AGE

20-30	1
31-40	2
41-50	3
>50	4

SEX

Male	1
Female	2

MARITAL STATUS

Married	1
Unmarried	2

RESIDENCE

Urban	1
Rural	2

DIETARY HABIT

Vegetarian	1
Non vegetarian	2

BODY MASS INDEX

<18.5	1
18.5-24.9	2
25-29.9	3
30-34.9	4
>35	5

EXERCISE

Yes	1
No	2

FAMILY HISTORY OF DIABETES MELLITUS

Yes	1
No	2

CLINICAL HISTORY

Clinical history	Yes	No
Blurring of vision	1a	2
Watering from the eyes	1b	2
Leg swelling	1c	2
Increased frequency of micturition	1d	2
Vomiting	1e	2
Floaters	1f	2
Flashes of light	1g	2

URINE PCR

<0.2	1
0.2 – 2	2
2 - 3.5	3
>3.5	4

LIPID PROFILE

Lipid profile	Normal	Abnormal
Cholesterol	1	2a
Triglycerides	1	2b
LDL	1	1c
VLDL	1	2d
HDL	1	2e

RETINOSCOPY FINDINGS

Normal	1
Mild NPDR	2
Moderate NPDR	3
Severe NPDR	4
PDR	5
CSME	6

HbA1C

<5.6	1
5.7-6.4	2
6.5-7.4	3
>7.5	4

SERUM URIC ACID

<3	1
3-5	2
5-7	3
>7	4

SL.NO	NAME	AGE	SEX	MARITAL STATUS	RESIDENCE	DIET	BMI	EXERCISE	SMOKING	ALCOHOL	DM	HISTORY	URINE PCR	LIPID PROFILE	RETINOSOPY	HBA1C	URIC ACID
1	Balamurugan	3	1	1	1	2	4	2	1	1	1	1c	3	2a,2b	5	4	4
2	anisha	2	2	1	1	2	2	2	2	2	2	2	2	2a.2c	1	3	3
3	jayavel	3	1	1	2	2	2	2	1	1	2	2	2	2a.,2b	2	4	4
4	papathi	3	2	1	2	2	5	2	2	2	2	2	2	2a,2b	1	3	4
5	kumaravel	2	1	1	2	2	2	2	2	2	2	2	1	1	1	3	3
6	raj	3	1	1	1	2	3	2	1	1	2	1a	1	1	1	4	4
7	albert	3	1	1	2	2	3	2	2	1	1	2	1	2b	1	3	3
8	rajan	3	1	1	2	2	3	2	2	2	2	2	1	1	1	3	3
9	ameer	3	1	1	1	2	2	2	1	1	2	2	1	2a,2b	1	4	4
10	sivaraman	3	1	1	1	2	3	2	1	1	1	2	2	2b,2e	1	4	4
11	baskar	2	1	1	1	2	4	2	2	2	2	2	1	2b,2e	1	4	4
12	ragu	3	1	1	2	2	3	2	1	1	2	2	1	2b	1	4	3
13	sudalai	3	2	1	2	2	5	2	2	2	2	2	1	2a,2b	1	4	4
14	subulakshmi	2	2	1	2	2	5	1a	2	2	2	2	1	2a,2b	1	4	3
15	perumal	3	1	1	2	2	3	2	1	2	1	2	1	1	1	4	4
16	shanthi	3	2	1	2	2	4	2	2	2	2	2	2	2b	1	4	4
17	rathinam	3	1	1	2	2	4	2	1	1	2	2	1	2a	1	4	4
18	prem	1	1	2	2	2	1	2	2	2	2	2	1	1	1	3	3
19	meena	2	2	1	2	2	2	2	2	2	1	2	1	1	1	3	3
20	preethi	2	2	1	1	1	3	1a	2	2	2	2	1	1	1	4	4
21	alaguraj	3	1	1	2	2	3	2	2	2	2	2	1	1	1	4	3
22	anitha	2	2	1	2	1	2	2	2	2	1	2	1	2b	1	3	4
23	esakipandi	1	1	2	2	1	2	2	2	1	1	2	2	2b,2e	1	4	4
24	mani	2	1	1	2	2	2	2	2	2	2	2	1	1	1	3	3
25	padmavathi	3	2	1	2	2	3	2	2	2	2	2	1	1	1	4	3
26	malar	2	2	1	1	1	2	1a	2	2	2	2	1	1	1	4	3
27	selvi	2	2	1	1	2	3	2	2	2	1	2	1	1	1	3	4
28	kannan	2	1	1	2	2	2	2	2	2	2	2	1	1	1	3	4
29	paul jebaraj	4	1	1	2	2	4	2	1	1	2	2	1	2a	1	3	3
30	kathiravan	3	1	1	1	2	3	2	1	1	2	1d	1	1	1	4	4
31	raji	2	2	1	2	1	4	2	2	2	1	2	1	2a	1	4	3
32	karupayee	3	2	1	2	2	4	2	2	2	2	1d	1	2a,2e	1	4	4
33	thirupathi	3	1	1	2	2	4	2	1	1	2	2	1	2b,2c	1	3	3
34	thangaselvam	1	1	2	2	1	2	2	2	2	2	2	2	2a,2b	1	3	4
35	shanmugam	3	1	1	2	2	3	1a	1	2	2	2	1	1	1	3	3
36	antony	3	1	1	1	2	3	2	1	1	2	2	1	2b	1	4	4
37	mumtaz	2	2	1	1	2	4	2	2	2	2	2	1	2a,2e	1	3	3
38	beer moideen	3	1	1	2	2	4	2	1	1	2	2	2	2a,2c	1	3	4
39	pandiyan	2	1	1	2	2	4	2	2	2	2	2	1	2a,2b	1	4	4
40	prakash	2	1	1	2	1	4	2	2	2	2	2	1	2b,2e	1	4	4
41	esakiammal	3	2	1	2	2	5	2	2	2	2	2	1	2a,2c	1	4	4
42	paulraj	3	1	1	1	2	3	2	2	2	2	2	1	1	1	4	4
43	lakshmi	2	2	1	2	1	2	2	2	2	1	1b	1	1	1	3	2
44	subbaiah	3	1	1	2	2	3	2	1	1	2	2	1	1	1	3	4
45	salamon	2	1	1	2	2	5	2	1	2	2	2	2	2a,2e	1	3	3

SL.NO	NAME	AGE	SEX	MARITAL STATUS	RESIDENCE	DIET	BMI	EXERCISE	SMOKING	ALCOHOL	DM	HISTORY	URINE PCR	LIPID PROFILE	RETINOSOPY	HBA1C	URIC ACID
46	sudalai	4	1	1	2	2	4	2	1	1	1	1d	1	2a	1	4	4
47	chellathai	3	2	1	2	2	3	2	2	2	2	2	1	1	1	4	4
48	robin	1	1	2	1	2	1	2	2	2	2	2	1	1	1	3	2
49	selvam	2	1	1	2	2	2	2	2	2	1	2	1	1	1	4	4
50	ramayee	3	2	1	2	2	3	2	2	2	2	2	1	1	1	3	3
51	ilayaperumal	4	1	1	2	2	4	2	1	1	2	2	1	2a,2b	1	4	3
52	vadivu	3	2	1	2	2	5	2	2	2	2	2	1	2a,3b	1	4	4
53	velammal	4	2	1	1	1	3	1a	2	2	1	2	2	1	1	3	3
54	arumugam	4	1	1	1	2	3	2	2	1	1	2	2	2b	1	4	3
55	karthick	3	1	1	2	2	5	2	1	2	1	1d	1	1	1	3	3
56	natrajan	2	1	1	2	2	5	2	2	2	1	2	1	1	1	3	3
57	kasthuri	3	2	1	2	1	4	2	2	2	1	1c	3	2b	1	4	4
58	beer fathima	4	2	1	2	2	3	2	2	2	2	2	1	1	1	4	3
59	paramasivan	2	1	1	2	2	2	1a	2	1	2	2	1	1	1	4	3
60	sivaram	4	1	1	2	1	4	2	1	1	2	2	1	2b,2e	1	4	3
61	mupudathi	3	2	1	2	2	2	2	2	2	2	2	1	1	1	3	3
62	subramaniyan	3	1	1	2	2	3	1a	1	2	2	2	2	1	1	3	3
63	saraswathi	2	2	1	2	2	4	2	2	2	2	2	1	2a	1	4	4
64	parthiban	2	1	1	2	2	2	1a	2	2	2	2	1	1	1	4	4
65	chellammal	2	2	1	2	2	4	2	2	2	2	2	1	2b	1	4	3
66	dhanalakshmi	3	2	1	2	2	3	2	2	2	2	1a	1	1	1	4	4
67	govindammal	3	2	1	2	2	3	2	2	2	2	1d	1	1	1	4	3
68	badhar nisha	2	2	1	2	2	2	2	2	2	2	2	2	2a,2b	1	3	4
69	balasubramaniyan	4	1	1	2	2	3	2	1	1	1	1c	3	2b	1	4	4
70	petchiammal	3	2	1	2	2	3	2	2	2	2	2	1	1	1	3	3
71	saradha	4	2	1	2	2	2	2	2	2	1	1b	3	2a,2b	3	4	4
72	manigandan	2	1	1	2	2	2	2	2	2	1	2	1	2b,2e	1	4	4
73	bhakiyathai	4	2	1	2	2	3	2	2	2	2	1d	1	1	1	3	3
74	ganesh pandi	3	1	1	2	2	3	2	1	2	2	2	1	2a,2c	1	3	3
75	sivaranjani	2	2	1	2	2	3	2	2	2	2	2	1	1	1	4	3
76	avudaiammal	4	2	1	2	2	3	2	2	2	2	2	1	1	1	3	4
77	sundari	4	2	1	2	2	3	2	2	2	1	2	2	2a,2c	1	4	4
78	murugan	3	1	1	2	1	3	2	2	1	2	2	1	1	1	3	4
79	palaniselvam	4	1	1	2	2	4	2	1	1	2	2	1	2b,2d	1	3	3
80	veeralakshmi	2	2	1	2	2	4	2	2	2	2	2	1	2b	1	4	4
81	kamal	4	1	1	2	2	3	2	2	2	2	2	2	2b	2	4	4
82	velsamy	4	1	1	2	2	4	2	1	1	1	2	1	2b	1	4	3
83	shiney	2	2	1	1	2	2	2	2	2	1	2	1	2a,2c	1	3	4
84	shankarapandiyan	4	1	1	2	2	3	2	1	1	2	2	2	2b	1	4	4
85	balkeshwari	3	2	1	2	2	4	2	2	2	2	2	1	1	1	4	4
86	manikam	2	1	1	2	2	4	2	1	1	1	1c	3	2b	1	4	4
87	karthikeyan	3	1	1	2	2	3	1a	1	1	2	2	1	1	1	4	3
88	raja	2	1	2	2	2	2	2	2	2	2	2	1	1	1	3	3
89	rangasamy	3	1	1	2	2	3	1a	2	1	2	2	2	2b	2	4	4
90	periyasamy	2	1	1	2	2	4	2	2	2	1	1d	1	1	1	4	3

SL.NO	NAME	AGE	SEX	MARITAL STATUS	RESIDENCE	DIET	BMI	EXERCISE	SMOKING	ALCOHOL	DM	HISTORY	URINE PCR	LIPID PROFILE	RETINOSOPY	HBA1C	URIC ACID
91	muthulakshmi	3	2	1	1	2	3	2	2	2	2	2	2	2b,2c	2	3	3
92	kannammal	3	2	1	2	2	4	2	2	2	2	2	1	2a	1	3	4
93	sivasubbu	3	1	1	2	2	3	1a	1	1	2	2	2	1	1	3	4
94	esakimuthu	2	1	1	2	2	2	2	2	2	2	2	1	1	1	4	2
95	arunachalam	3	1	1	2	2	4	2	2	2	2	2	1	2a,2c	1	3	4
96	mariyappan	4	1	1	2	2	3	2	1	1	2	2	1	2a,2c	1	4	3
97	kanthasamy	3	1	1	2	2	4	2	1	2	2	2	1	2d	1	3	4
98	fathima	2	2	1	2	2	3	2	2	2	2	2	1	2a,2b	1	3	4
99	rajammal	3	2	1	2	2	3	2	2	2	2	2	1	1	1	3	3
100	vasantha	2	2	1	2	2	3	2	2	2	2	2	1	2a,2b	1	4	4