*A Dissertation on*

# AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY

*Dissertation submitted to*

**THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY**

**CHENNAI - 600 032**

*in partial fulfillment of the regulations for the award of the degree of*

**M.S. DEGREE IN OPHTHALMOLOGY**

**COIMBATORE MEDICAL COLLEGE**

**COIMBATORE**

**MAY 2019**

# DECLARATION

I solemnly declare that this dissertation entitled "**AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY**" is a bonafide and genuine research work done by me under the supervision and guidance of **Dr.V.Thaialnayaki, M.S.,** Associate Professor of the Department of Ophthalmology, Coimbatore Medical College, Coimbatore.

This is submitted to The **Tamil Nadu Dr. M.G.R Medical University**, Chennai in partial fulfillment of regulations required for the M.S. Ophthalmology, Branch III Degree Examination to be held in **MAY 2019**.

Date: **Dr.Prayagi Kandoth**

Place:

# CERTIFICATE

This is to certify that the dissertation entitled "**AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY**" is a bonafide research work done by **Dr. Prayagi Kandoth**, Postgraduate in M.S. Ophthalmology, Coimbatore Medical College, Coimbatore under my direct guidance and supervision, to my satisfaction, in partial fulfillment of the regulations required for the award of M.S Degree in Ophthalmology (Branch III).

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Chief Guide
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Head of the Department
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# CERTIFICATE –II

This is to certify that this dissertation work titled **" AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY"** of the candidate **Dr. Prayagi Kandoth** with registration number 221613202 for the award of **M.S. DEGREE IN OPHTHALMOLOGY**. 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 pages and result shows **4% percentage** of plagiarism in the dissertation.

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| **Course** | : | **MS (Ophthalmology) Post Graduate** |
| **Period of Study** | : | **1 year** |
| **College** | : | **Coimbatore Medical College & Hospital.** |
| **Dissertation Topic** | : | **An analytical study correlating the significance of serum lipids in the development of clinically significant macular edema in patients with diabetic retinopathy.** |

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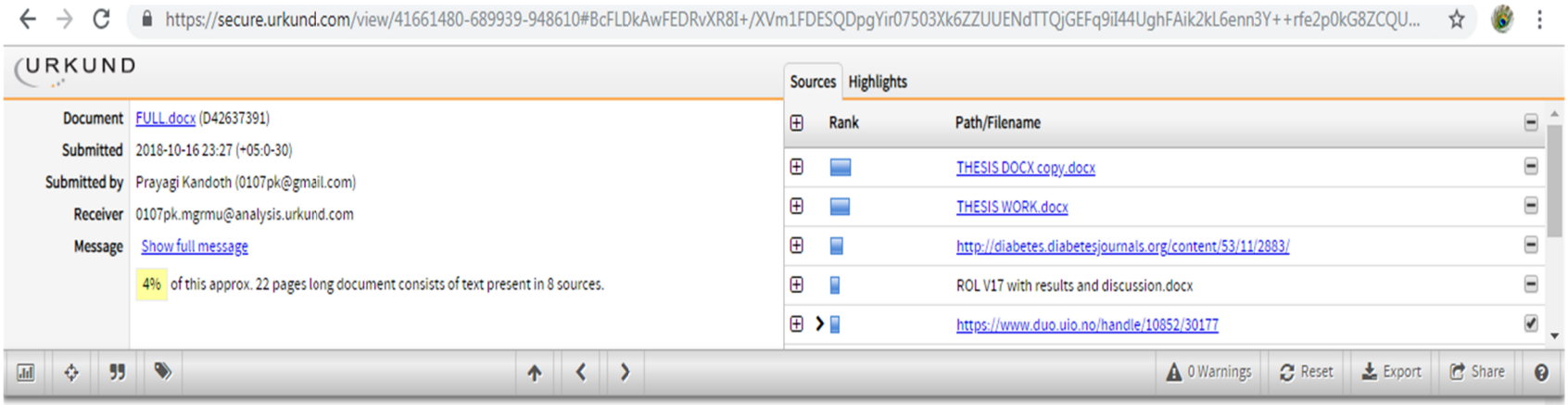


A Dissertation on AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY

Dissertation submitted for M.S.Degree in Ophthalmology MAY 2019

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU

Declaration

DECLARATION I solemnly declare that this dissertation entitled "AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY" is a bonafide and genuine research work done by me under the supervision and guidance of Dr. J. Saravanan M.S., Assistant Professor of the Department of Ophthalmology, Coimbatore Medical College, Coimbatore. This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of regulations required for the M.S. Ophthalmology, Branch III Degree Examination to be held in MAY 2019.

Date: Dr.Prayagi Kandoth Place:

Certificate

# ACKNOWLEDGEMENT

With deep sense of gratitude and due respect, I bestow my earnest and sincere thanks to **Dr.B.ASOKAN, M.S.,M.Ch.,** The DEAN, Coimbatore Medical College Hospital, Coimbatore for permitting me to do this research work.

I am extremely thankful to **Dr.S.PADMANABAN, M.S.,D.O,** Head of the Department, Ophthalmology, Coimbatore Medical College Hospital for his valuable guidance and moral support which helped me in completing this work successfully.

It is my heartfelt privilege to convey my deepest sense of gratitude and regards to my respected teacher and mentor **Dr.V.Thaialnayaki, M.S.,** Associate Professor for his erudite guidance and prudent suggestions. She painstakingly went through each and every word of my thesis and with her profound knowledge, close supervision and constant encouragement added quality to my thesis. Working under her supervision has been a wonderful experience both at academic and personal level.

I am thankful to my co guide **Dr. J. Saravanan, M.S,** Senior Assistant for providing continuous support and constructive suggestions throughout the study. His encouragement, constructive criticism and suggestions added quality to my thesis.

I owe my gratitude to my Assistant Professors **Dr.C.Jeevekala M.S.,D.O., Dr.P.Sumathi M.S., Dr.K.Malligai D.O.,DNB, Dr.P. Mohanapriya M.S., Dr.K.Sathya M.S., Dr Karthikeyan M.S.,** and **Dr. Haripriya M.S.** for helping and guiding me in completing this work.

Moreover I wish to thank all my beloved colleagues, seniors and all the technicians in the Department of Ophthalmology for their significant inputs and cooperation.

I am thankful to all the patients who took part in my research work, without whose participation the study would not have materialized.

Last but not the least I thank with all my heart my parents and my sister who always bestowed me with their love and encouragement. I am especially indebted to them for their patience, love and motivation they offered to me throughout.

# ABBREVIATIONS & ACRONYMS

| | | |
|---|---|---|
| DM | - | Diabetes Mellitus |
| DR | - | Diabetic Retinopathy |
| CSME | - | Clinically Significant Macular edema |
| DME | - | Diabetic Macular Edema |
| OCT | - | Optical Coherence Tomography |
| SBP | - | Systolic Blood Pressure |
| DBP | - | Diastolic Blood Pressure |
| TC | - | Total Cholesterol |
| TG | - | Triglyceride |
| HDL-C | - | High Density Lipoprotein Cholesterol |
| LDL-C | - | Low Density Lipoprotein Cholesterol |
| CNP | - | Capillary Non Perfusion |
| OHA | - | Oral Hypoglycemic Agent |
| WHO | - | World Health Organization |
| FAZ | - | Foveal Avascular Zone |

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# INTRODUCTION

Diabetes Mellitus (DM) is an important health issue today. According to World Health Organization (WHO), the number of people with diabetes has increased from 177 million in 2010 to 326.5 million in 2017,in the age group of 20-64 years. It is expected that the number will be around 438 million by the year 2040.In 2017 alone is considered, China is the country with highest number of people with diabetes, with 114 million people suffering from DM. Next to China, India has the next highest number. India contributes 49% of the world's diabetes burden, with around 72 million in 2017 .The data show that the number is expected to increase to 134 million by 2025.

Diabetic retinopathy is one of the leading causes of blindness in the world in the age group of 20 to 75 years. Diabetic retinopathy is seen to be affecting 75% of those with diabetes and tends to develop diabetic retinopathy after a mean duration of 15 years.[1] Blindness in diabetic retinopathy is due to tractional detachment of retina, long standing non clearing vitreous hemorrhage and due to diabetic macular edema. Macular edema is the leading cause of visual loss in diabetic retinopathy patients .Clinically significant macular edema (CSME) is seen in upto 4% of the people with diabetes.[1]

Increase in the severity of retinopathy leads to an increase in the incidence of macular edema. It is seen in non-proliferative as well as proliferative diabetic retinopathy.

The term diabetic maculopathy is applied to a range of pre retinal and intra retinal changes occurring at the macula. Retinal ischemia due to capillary and arteriolar non perfusion and breakdown of blood-retinal barrier are the major changes occurring intra retinally in diabetic retinopathy. Diabetic macular edema can develop in any stage of retinopathy and is seen to produce both the structural and functional changes at the macula.

The two major categories of macular edema in diabetic maculopathy are focal macular edema and diffuse macular edema.

1. Focal macular edema is characterized by focal leakage of micro aneurysms, dilated capillary segments and is often seen with hard exudates ring formation.

2. Diffuse macular edema is characterized by widespread retinal capillary abnormalities and is due to diffuse leakage following extensive breakdown of blood retinal barrier. It affects the entire posterior pole and often leads to cystoid macular edema.

**Effects of diabetes on macula:**

1) Pre retinal or intra retinal hemorrhage.

2) Macular edema- collection of fluid within the layers of retina in the macula with or without hard exudates and cystoid changes.

3) Traction at macula: It is caused by proliferation of fibrous tissue causing tangential traction or wrinkling of retina or detachment of retina at macula.

4) Lamellar or full thickness macular hole formation.

The ETDRS has defined Clinically Significant Macular Edema (CSME) as macular edema that meets certain criteria for location and size. In case of presence any one of the following criteria, CSME is said to be present:

i. Retinal thickening within 500µm of the center of the macula

ii. Exudates within 500µm of macula ,if associated with retinal thickening. The thickening may be outside the 500µm.

iii. Retinal thickening of one disc area(1500µm) or larger, any part of which is within one disc diameter of the center of macula.[2]

DME is divided into Centre involving (500µm) and Non center involving (outside 500µm),based on the criteria of involvement of fovea in Optical Coherence Tomography(OCT)[2]

Those patients with edema at the macula develop dimension of vision and those people whose macula is spared have an excellent visual acuity. Once DME develops, it is difficult to bring back the full vision and treatment is mainly aimed to stabilise the pretreatment vision and also vision improvement occur in few cases. Thus it is of prime importance to examine diabetic patients regularly before the vision drops, so that timely intervention can be done before the condition becomes beyond the scope of treatment.

There are particular retinal lesions identified on FFA has to be treated. These 'treatable lesions' associated with macular edema include-

1) Focal leaks > 500 µm from center of macula believed to be causing retinal thickening or hard exudates.

2) Focal leaks 300–500 µm from the center of macula believed to be causing retinal thickening or hard exudates, if there is surety that the remaining peri foveal capillary network will not be destroyed

3) Previously untreated areas of diffuse leakage.

4) Previously untreated avascular zones, other than ‘FAZ’ .

In the ETDRS, 2 types of photo coagulation methods were used for treatment of diabetic macular edema namely focal and grid.

Focal refers to direct treatment of all leaking micro aneurysms in the edematous retina, between 500-3000µm from the center of macula.

Grid treatment is used primarily for identifiable diffuse leakage areas and thickened retina.

Elevated lipid levels are associated with endothelial dysfunction, which appears to play an important role in the pathogenesis of DR, particularly in relation to break down of blood retinal barrier and development of CSME and hard exudates. Patients with diabetes are known to have severe lipid abnormalities like hyper cholesterolemia and elevated serum triglycerides. The WESDR, a population based study and the ETDRS found that increased levels of serum cholesterol were associated with increased severity of hard exudates in retina. Independent of accompanying macular edema, the severity of retinal hard exudates at base line was associated with decreased visual acuity in the ETDRS. The severity of retinal hard exudates also was a significant risk factor for moderate vision loss during the course of the study. The data are compelling to recommend lowering raised serum lipid levels in patients

with DR to reduce the risk of visual loss besides reducing the risk of cardiovascular disease. The current study done to assess the correlation of levels of serum lipids with the development of Clinically significant macular edema in our population.

# AN OVERVIEW OF DIABETES MELLITUS

Diabetes mellitus is classified by the American Diabetic Association into type 1 diabetes mellitus or IDDM( insulin-dependent diabetes mellitus) and type 2 diabetes mellitus or NIDDM (non– insulin-dependent diabetes mellitus. In type 1 DM, there is β-cell destruction, leading to absolute deficiency of insulin. It is either idiopathic or immune mediated. T2DM can be either insulin resistance with relative insulin deficiency or defect of insulin secretion with insulin resistance. Other forms include genetically mediated, secondary to endocrinopathies and DM following usage of drugs and induced by chemicals.[2] Type 2 DM consists of 90% and is predominant in 4$^{th}$ decade.Type2 DM in children and adolescents in on the rise.Type 1 DM is more associated with diabetic retinopathy.[3]In those presenting to clinicians it is seen to be more associated with type2 as the number of people with type2 is more.[2]

Chronic effects of DM includes nephropathy, neuropathy and retinopathy affecting the microvascular circulation of kidney, peripheral nerves and retina. Eventhough the changes in the metabolism affect the neurons and the support cells directly, it is the changes occurring in the vessels which leads on to the development of macular edema and new vessel formation.[1]

# CLASSIFICATION OF DIABETIC RETINOPATHY

The ETDRS (Early Treatment Diabetic Retinopathy Study) has classified Diabetic Retinopathy into NPDR(Non Proliferative Diabetic Retinopathy) and PDR(Proliferative Diabetic Retinopathy. This classification is based on the findings in the clinical examination and comparing it with the standard photographs. Very mild NPDR, mild NPDR, moderate NPDR, severe and very severe NPDR forms the further classification of NPDR. Mild-moderate, High risk PDR and (ADED) Advanced Diabetic Eye Disease forms the sub classification of PDR.[2]Chronically elevated blood glycemic levels is the most important factor leading to the development of diabetic retinopathy and its various complications.[1]

# RELEVANT ANATOMY

Various anatomic lesions associated with diabetic retinopathy includes:

**1) LOSS OF PERICYTES :**

It is one of the earliest and most specific signs of diabetic retinopathy. It was described for the first time by Cogan et.al.[4-6]They had examined mounts containing trypsin-digested retinal vessels from diabetic patients. Microvascular auto regulation is maintained mainly with the help of these contractile pericytes. Pericyte loss leads to changes of vascular intercellular contacts and inner blood–retina barrier impairment. These leads to venous dilation and beading that is seen in fundus examination clinically. Microaneurysms develop due to proliferation of endothelial cells following loss of intercellular contacts.[7]

The mechanism by which chronic hyperglycemia leads to pericyte degeneration remains largely unknown. The two leading hypotheses implicate the aldose reductase pathway and platelet-derived growth factor-beta (PDGF-β).

**2) CAPILLARY BASEMENT MEMBRANE THICKENING:**

Thickening of capillary basement membrane can be seen on electron microscopy and is lesion well associated with diabetic

retinopathy. Fibrillar collagen and “Swiss cheese” vacuolization of the basement membrane forms the other electron microscopic findings of DR. The biochemical mechanism leading to basement membrane thickening remains unknown but studies suggest a role for the aldose reductase and the sorbitol pathway.[8,9]

**3) MICROANEURYSMS:**

It is the earliest ophthalmoscopically visible sign of DR, appearing as tiny red intra retinal dots.[10]In light microscopy, it appears as grape-like or spindle-shaped dilations of retinal capillaries.[11] They can be either hypercellular or acellular. Loss of the ant proliferative effect of pericytes leads to the formation of hypercellular microaneurysms. Endothelial cell and pericyte apoptosis of the hypercellular microaneurysms leads to the development of acellular microaneurysms.[12]

**4) BREAKDOWN OF BLOOD–RETINA BARRIER :**

Blood–retina barrier breakdown is a significant pathophysiologic change in diabetic retinopathy leading to the development of edema, the leading cause of loss of vision in diabetic patients. One of the mechanisms of functional alteration of this barrier involves opening of the tight junctions known as zonula occludens that are seen between vascular endothelial cell processes.[13,14]

# BIOCHEMICAL MECHANISMS IN THE PATHOGENESIS OF DIABETIC RETINOPATHY

## 1) CHRONIC HYPERGLYCEMIA:

It is the most important causative factor leading to all the microvascular complications of diabetes, including diabetic retinopathy. Leukocyte activation followed by release of cytokines and adhesion molecules occurs. These molecules leads to the increased adhesion of leukocytes to the capillary walls leading to occlusion and hypoxia.[15-17]

In the aldose reductase theory ,increase of glucose inside the cells leads to increased activation of the aldose reductase pathway or the polyol pathway.[18,19]The sorbitol formed from glucose through this pathway accumulates inside the cell producing its various effects.

## 2) ADVANCED GLYCATION ENDPRODUCT (AGE) THEORY:

One another factor implicated in the development of complications in DM is the formation of AGEs or the Advanced Glycation End Products.[20] AGEs is the collective terminology used for proteins, lipids, and nucleic acids which undergo irreversible modification by reducing sugars or sugar-derived products through a reaction called Maillard

reaction. It is this reaction which is responsible for the browning of tissues with ageing.

**3) REACTIVE OXYGEN INTERMEDIATES (ROI) THEORY:**

It is one of the oldest theories proposing that increasing oxidative stress due to chronic hyperglycemia leads to complications of diabetes. Usually metabolism of glucose occurs via glycolytic and the tricarboxylic acid pathway, which produces adequate reducing equivalents for the production of Adenosine Triphosphate (ATP) via the oxidative phosphorylation. But high levels of glucose[21] leads to increased production of byproducts of oxidative phosphorylation, such as superoxide anion, also. Increased free radicals like superoxide anion leads to damage of mitochondrial DNA[22] and also reduces the levels of nitric oxide [23,24],thus promoting adhesion of leukocytes to endothelium [25] and damage of cellular proteins.[26]

# RISK FACTORS FOR DIABETIC RETINOPATHY

## 1) RACE:

Evaluation of race showed that the occurrence and the severity of type 1 diabetes is the same in the Africans Americans when compared to the whites [27,28] and that type 2 is more prevalent among African Americans as evidenced in the Atherosclerotic Risk in Communities (ARIC) study [29],the Cardiovascular Health Study [30] and the Multi Ethnic Study of Atherosclerosis [31].In a study conducted at Dr.Mohan's diabetic specialty centre at Chennai, by Dr Rema and Dr.Pradeepa, it was found that the prevalence of DR as much lesser than the age matched western population.[32]

## 2) GENETIC FACTORS :

A number of studies give more strong association of diabetic retinopathy and genetic factors than previously thought. [33,34] Possible causal factors implicated in the pathogenesis of diabetic retinopathy were aldose reductase activity, oxidative stress and platelet adhesiveness and aggregation, collagen formation, inflammatory process, glycation, protein kinase activity. Extensive study of these factors helped to conclude the association of these factors in the genetic predisposition of diabetes. There are already a number of studies that have reported associations

between retinopathy and mitochondrial DNA mutations and polymorphisms of the aldose reductase gene,[35,36] TNF-beta NcoI gene,[37] epsilon4 allele of apolipoprotein E gene,[38] paraoxonase gene,[39] endothelial nitric oxide synthase gene,[40] intercellular adhesion molecule-1 (ICAM-1),[41] alpha2beta1 integrin gene,[42] vascular endothelial growth factor (VEGF) gene, and many more.[43,44]

**3) SEX:**

On comparison of young males with females the former had higher prevalence of proliferative retinopathy.[2] However, there were no significant differences in progression of diabetic retinopathy between the sexes.[46,47,48] Among those with type 2 diabetes there were no significant difference in the prevalence or rates of progression to PDR between the sexes, in WESDR.[47,49,50]

**4) AGE AND PUBERTY:**

In WESDR type 1 diabetics had increased prevalence and severity as the age advanced. Irrespective of the duration of diabetes, those younger than 13years had no evidence of diabetic retinopathy.

In the WESDR, after adjusting for other risk factors, it was observed that those who had attained menarche had three times increased risk for retinopathy as compared to the premenarchal.

**5) DURATION OF DIABETES :**

It is the most consistent association of diabetic retinopathy as understood from all the studies done related to it. Duration of diabetes is seen to affect the frequency, severity and the development of maculopathy.[45] The WESDR showed a 14% prevalence in men and a 24% prevalence among women after a 3-4 years of detection of diabetes in those with type1 DM. However, in patients who has longer duration of diabetic history(19-20 years),it was observed that men had more prevalence 50% of PDR when compared to women(33%).Those with type 2 developed retinopathy earlier than those with type 1.

**6) AGE AT DIAGNOSIS:**

Age at diagnosis did not show any relation to incidence or progression of diabetic retinopathy in any of the groups in WESDR.[46,50]

**7) BLOOD PRESSURE :**

Blood pressure is a significant predictor of the incidence of DR in patients with type1 DM, according to the WESDR.[48]When other risk

factors were adjusted, it was found that the relationship between BP and retinopathy remained only in the younger onset group. However, in the WESDR no relationship was found with the incidence and progression of retinopathy in type 2 DM patients.[51] In contrast to it in the UKPDS it was observed that BP is a risk factor for the development of retinopathy. It as seen that the complications of DM reduced by 13% for every 10mmHg reduction in BP. No threshold was found for any retinopathy endpoint.[52] In the WESDR, increase in diastolic BP by 10 mmHg lead to a 330% and 210% increase in the 4-year risk of developing macular edema in those with type 1 DM and type 2 DM respectively.[53]

**8) SERUM LIPIDS:**

In the WESDR, higher serum total cholesterol was associated with higher prevalence of retinal hard exudates which was seen in both the younger and the older onset groups taking insulin. In contrast, type 2 diabetics using oral hypoglycemic agents, did not show such association.[54] In the ETDRS, baseline high values of serum lipids were observed to be associated with increased risk of developing hard exudates in the macula and decreased visual acuity.[55] Santos et al. did a study on Mexican population with type 2 DM and found that [56] the frequency of

severe retinal hard exudates was higher in those with the epsilon4 allele polymorphism of the apolipoprotein E gene.

**9) PROTEINURIA AND DM NEPHROPATHY:**

Studies concluded that those patients who had gross proteinuria or showed evidence of microalbuminuria on testing had more prevalence of retinopathy also.[45,47,49,57,58,59,60,61,62,63] It was estimated that the lipid, platelet and the rheological abnormalities seen in case of nephropathy is reason for retinopathy .Chronic kidney disease people are noted to have higher risk of macular edema and it as seen to improve when the renal function recovered well.

**10) SMOKING :**

Smoking due to its effect of increased carbon monoxide in blood leads to tissue hypoxia and also because of its ability to increase the platelet adhesion and aggregation ,it results in increased complications of diabetes [64, 65]

**11) ALCOHOL:**

As usage of alcohol leads to decreased glucose level, decreased inflammation and reduced platelet aggregation, it was expected to be beneficial in the prevention of development of DR. In the EURODIAB

Prospective Study of Complications in those with type 1 DM, regular alcohol usage was found to have protective effect by delaying the progression of retinopathy. [66]There were other studies with conclusion contratry to the above mentioned study.[67,68] A population-based study done in Australia, showed no association between alcohol use and diabetic retinopathy.[69] The ADVANCE Retinopathy Measurements (ADREM) also showed no evidence of relation between alcohol and retinopathy in type 2 DM patients. In the UKPDS, men who had alcohol consumption history and were recently diagnosed to be diabetic, showed an increased severity of retinopathy.[70]

**12) PHYSICAL ACTIVITY:**

As proved by many studies, adequate exercises have a beneficial effect on glycemic control, and hence associated with decreased prevalence and incidence of diabetic retinopathy.[71]

**13) OTHER OCULAR FACTORS:**

Becker[72] reported that a decreased prevalence and severity of diabetic retinopathy were seen in those with glaucoma. Other studies have reported similar results. There were other studies also reporting similar results but has never been confirmed by a methodologically precise epidemiological study.

Myopia is another one factor found to be associated with lesser prevalence and severity of diabetic retinopathy.[73] This association of myopia was proven in a study by Rand et al.[74] who found an interesting interaction between myopia of greater than 2 diopters and HLA-D-group antigens.

**INVESTIGATIONS :**

1. Fundus Fluorescein Angiography(FFA)
2. Fundus Auto Fluorescence(FAF)
3. Optical Coherence Tomography(OCT)
4. Optical Coherence Tomography Angiogram(OCTA)

## TREATMENT OF DIABETIC MACULAR EDEMA (DME)

DME is the most important cause of drop in visual acuity in patients with diabetic retinopathy. The ETDRS has proved that in those with CSME laser treatment helps in the reduction of visual loss. In the ETDRS, after a 3 year follow up, it was observed that CSME patients who received no treatment, 33% had significant visual loss. Irrespective of the visual acuity, patients should be given laser treatment as it helps to reduce the loss of vision by 50%.

**Mechanism of action of laser in DME:**

1) Focal laser helps to improve DME by stopping the leakage from the microaneurysms by coagulating it.

2) Laser treatment help to maintain the integrity of the outer blood retinal barrier by decreasing the substances which cause mitosis of endothelial cells. Lesser leakage and more absorption reduce the DME.

3) Laser treatment helps to reduce the oxygen consumption by destroying some of the photo receptors and retinal pigment epithelial cells which consume most of the oxygen. Scarring

following treatment also leads to thinning of the retina which allows for better diffusion of oxygen from the choroids.

4) Dilated and leaky retinal capillaries become narrower and less leaky after laser treatment.

**The techniques of laser treatments in DME are:**

## 1)LASER TREATMENT:

A)**Focal treatment:** Involves application of laser burns to the micro aneurysms and micro vascular lesions in the center of rings of hard exudates located 500 to 3000 µm from the center of the macula.

Spot size :50-100 µm

Duration : 0.1 seconds

Power: sufficient to obtain gentle whitening or darkening of the lesions.

Treatment up to 300 µm from the center of the macula may be considered if CSME persists despite previous treatment and visual acuity is < 6/12. In these cases a shorter exposure time of 0.01 seconds is recommended.

**B)Grid treatment**: It is used for diffuse retinal thickening located more than 500 µm from the center of the macula and 500 µm from temporal margin of the optic disc.

Spot size:100 µm

Duration: 0.1 sec

One burn width apart.

It takes around 4 months for the edema to resolve. Approximately 70% of eyes achieve stable visual acuity, 15% show improvement and 15% subsequently deteriorate. Re treatment may be considered after 4 months.

**3)Anti VEGFs:** These act by binding to various Vascular Endothelial Growth Factor ( VEGF) receptors and thus inhibiting angiogenesis, vascular permeability and lymphangiogenesis caused by increased expression of the VEGF molecules. Ranibizumab is a humanized murine monoclonal antibody fragment, which binds to VEGF-A, Pegabtinib binds to VEGF-A 165 isoform. Aflibercept/ VEGF trap binds both VEGF and PIGF(placental like growth factor)

**4)Intra vitreal triamcinalone acetonide:** may be attempted for those cases that fail to respond to conventional laser photocoagulation. It effects decreases after six months and macular edema frequently returns.

**5) Intravitreal implants:** Intravitreal insert called Iluvein give sustained release of fluocinolone acetonide and was seen to produce significant benefit in the treatment of DME.

Another intravitreal implant is the one containing dexamethasone (Ozurdex).

5)**Posterior subtenon triamcinalone acetonide:** when given along with laser it may aid in improving vision.

6)**Pars Plana Vitrectomy:** is considered when macular edema is associated with tangential traction. OCT aids in demonstrating eyes with marked vitreoretinal traction that may benefit most from surgery.

7)Hypo lipidemic drugs have been shown to reduce the severity of hard exudates and sub foveal lipid migration in eyes with CSME in type 2 diabetes mellitus patients with dyslipidemia and may become an important therapeutic adjunct.

# AN OVERVIEW OF HYPERLIPIDEMIA

Lipids are a heterogeneous group of water insoluble organic molecules that can be extracted from tissues by non polar solvents. Due to their insolubility in aqueous solutions, body lipids are generally found either compartmentalized or transported in association with proteins as lipoproteins. These particles include chylomicrons , very low density lipoprotein(VLDL) , low density lipoprotein(LDL) and high density lipoprotein(HDL).

Triglycerides and the esterified form of cholesterol (Cholesteryl ester) are non-polar and hydrophobic and comprise the lipoprotein core. Phospholipids and some free cholesterol are amphipathic molecules (soluble in both aqueous and lipid envelop) which cover the surface of the particle, where they act as interface between plasma and core components.

Cholesterol is an important lipid which has a very low solubility in water. The actual plasma concentration of cholesterol in healthy people is usually 150-200mg/dl. Due to the presence of plasma lipoproteins, cholesterol has high solubility in blood. Majority (around 70%) of the cholesterol in plasma lipoproteins exist in the form of cholesterol esters and only 30% occurs as free in the circulation.

It is the precursor of bile acids, which are synthesized in the liver and that facilitates the absorption of triglycerides and fat soluble vitamins. It is also acts as the precursor of various steroid hormones.

**HYPERLIPIDEMIAS:**

The most widely accepted classification of hyperlipidemia is that of **Frederickson's classification**. It is divided into

1) **Type I**:showing an increase of chylomicrons. Eruptive xanthomas and hepatomegaly is seen.
2) **Type IIA/Primary Familial Hypercholesterolemia**. It is caused due to LDL receptor defect. It may be due to deficiency of LDL receptor as such or due to defective binding of B-100 to the receptor or due to defective internalisation of the receptor –LDL complex. Common cause of coronary artery disease and tuberous xanthoma.
3) **Type IIB/Hyperlipoprotenemia**: There is increase of both cholesterol and triglycerides and an increased production of apo-B, thus leading to an elevation of LDL and VLDL. Corneal arcus is a manifestation of it.

4) **Type III:**It is characterised by an increase in VLDL and chlomicrons. It causes palmar xanthoma and also leads to a high incidence of vascular diseases.

5) **Type IV/Familial Endogenous Type:**It is due to over production of TG by the liver. VLDL is seen to be elevated. It is associated with diabetes mellitus, ischemic heart disease and obesity.

**6) Type V:** Increase in VLDL and Chylomicrons is noted. Ischaemic heart disease is highly associated.

**Classification of Total cholesterol, LDL Cholesterol and HDL Cholesterol values:**

| | **Total Cholesterol** | **Triglycerides** | **HDL-C** | **LDL-C** |
|---|---|---|---|---|
| **Desirable** | <200 mg/dL | <150mg/dL | >60 mg/dL | <130 mg/dL |
| | 5.2 mmol/L | <1.7 mmol/L | >1.55mmol/L | <3.36 mmol/L |
| **Borderline** | 200-239 mg/dL | 150-199mg/dL | 35-60mg/dL | 130-150 mg/dL |
| | 5.5-6.18mmol/L | 1.8-2.2 mmol/L | 0.9-1.55 mmol/L | 3.36-4.14 mmol/L |
| **Undesirable** | >240 mg/dL | >200mg/dL | <35mg/dL | >160 mg/dL |
| | >6.21 mmol/L | >2.3 mmol/L | <0.9mmol/L | >4.14 mmol/L |

**Treatment of dyslipidemias:**

**Non Pharmacological Treatment :**

It includes dietary modification. Physician should carefully asses the current diet taken by the patient and suggest modification to improve the dyslipidemic status.

Other factors include, cessation of smoking, decrease alcohol intake, weight reduction and regular exercises.

**Pharmacological Treatment :**

**1) HMG COA reductase inhibitors (statins):** HMG COA reductase inhibits the rate limiting step in cholesterol biosynthesis and inhibitors of this enzyme decreased cholesterol synthesis. It not only causes dose dependent reduction in plasma levels of LDL & TGs but also an increase in HDL levels.

Eg. Lovastatin, Provastatin, Simvastatin, Fluvastatin, Atorvastatin, Rosuvastatin.

**2)Cholesterol absorption inhibitors:**

Ezetimibe is a cholesterol absorption inhibitor that blocks the cholesterol absorption from the micelle in the intestine. It has been shown to decrease

cholesterol also by almost 60%. It can be used in combination with statins.

**3) Bile Acid sequestrants (Resins) : BAS**

It bind bile acids in the intestine and promote their excretion. To maintain bile acid pool size, the liver diverts cholesterol to bile acid synthesis.

Eg. Cholestyramine, colestipol, colesevelam

**4) Nicotinic Acid (Niacin):**

Niacin is a lipid modifying agents which inhibit lipolysis leading to a decreased level of plasma TG and LDL levels and an increased HDL – C. It is also the only currently available lipid lowering agent that significantly reduces plasma levels of lipoprotein(a).

**5) Fibric acid derivatives (Fibrates**):

These decrease the levels of triacylglycerols by activating lipoproteinlipase and inhibiting secretion of VLDL by acting as agonists of PPAR alpha, a nuclear receptor involved in the regulation of carbohydrate and lipid metabolism. Fibrates are the most effective drugs available for reducing TG, VLDL and increasing HDL C.

Eg:Gemfibrosil

**6) Omega 3 Fatty Acids (Fish oils)**

This polyunsaturated fatty acids are present in high concentrations in fish and in flax seeds. Use of low dose of Omega 3 has been proved to decrease fasting TG levels and also cause a reduction in cardiovascular events in CHD patients.

**7) Probucol**

It is believed to act at $ABCA_1$.It acts by increasing the LDL catabolism and prevents accumulation of LDL in arterial walls. Disadvantage is that it also decreases the levels of HDL.

**8)Vit E**

Its anti oxidant property decreases the oxidation of LDL, thus reducing atherosclerosis.

# REVIEW OF LITERATURE

Major studies related to lipid abnormalities and diabetic retinopathy:

**The Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR)** XIII [54]was done to elucidate the relationship of development of hard exudates with serum cholesterol. Serum total cholesterol showed a significant association with the severity of DR and hard exudates in those patients taking insulin. HDL was found to be unrelated to the severity of lesions.

**The Early Treatment Diabetic Retinopathy Study( ETDRS)** Report 22 [55] was published in 1996,which included 2709 patients with DR. Results showed that those with elevated serum cholesterol or serum LDL had more chance of developing hard exudates in the retina.

**The United Kingdom Prospective Diabetes Study(UKPDS)** [70] is a multi centre, randomised control study done in those with non insulin dependent diabetes mellitus patients. In this study it was noted that even HDL cholesterol levels were also associated with severity of diabetic retinopathy. They did not give any explanation for such an observation. In addition, LDL and triglycerides did not appear to be related to the DR severity.

**The Diabetes Control and Complications Trial (DCCT)** [76] evaluated the relationship between serum lipid levels and Clinically Significant Macular Edema(CSME),hard exudates and other end points in DR.Study was conducted in 1441 patients with type 1 DM. It was found from the study that increased LDL and total cholesterol to HDL ratio showed an increased possibility of development of CSME and hard exudates. They concluded that drugs to reduce the level of lipids decrease the risk of CSME.

**The Multi Ethnic Study of Atherosclerosis(MESA)** [31] was done in the multi ethnic population of US. In this study they found that DR,CSME or vision threatening retinopathy were not significantly associated with HDL,LDL and TG.

**The Chennai Urban Rural Epidemiology Study(CURES) Eye Study 2** [77] studied 1736 patients with type 2 DM with diabetic retinopathy and found that average value of serum HDL,TG and cholesterol were significantly higher than those without diabetic retinopathy. They also observed that DME was associated with non HDL cholesterol and LDL-C.

**Ucgun et.al** [78] from Turkey conducted a small study to assess the correlation between serum lipid levels and exudative maculopathy in diabetic patients with non-proliferative retinopathy. Fifty patients were equally divide into those with maculopathy and those without. Results showed a higher association of serum cholesterol and LDL in those with exudative maculopathy. It was also observed that TG,HDL and VLDL did not show any difference between the 2 groups.

Results from the **Singapore Malay Eye Study(SMES)** [79] showed that LDL cholesterol is an independent risk factor for any type of retinopathy.

**SN-DREAMS** Report Number 13[80] was a population based cross sectional study conducted by **Rajiv Raman et.al.**, at Sankara Nethralaya, it was found that out of the 1414 participants, one third had Diabetic macular edema and 6% of them had features suggestive of CSME. In their study they had compared risk factors associated with CSME and Non CSME separately and found that CSME patients had poor glycemic control, increased serum total cholesterol and microalbiminuria, whereas non CSME patients had high serum LDL, high non HDL cholesterol and increased cholesterol ratio.

**The Atherosclerotic Risk in Communities Study (ARICS)** [29] was done to assess the prevalence of diabetic retinopathy and its associations with atherosclerosis and vascular risk factors. Out of the 1600 patients enrolled 328(20.5%) had retinopathy, hard exudates were seen in 6.6%, proliferative diabetic retinopathy in 1.8% and 1.65 had macular edema. LDL cholesterol and plasma lipids were noted to be associated with retinal hard exudates.

**The Cardiovascular Health Study (CHS)** [30]was a population based cohort study done to see the relation between retinopathy and atherosclerosis and atherosclerotic risk factors. Their study showed that retinopathy was associated with higher systolic BP, increased levels of LDL and total cholesterols and presence of cardiovascular diseases. No association was found between retinopathy and HDL and triglycerides.

**The Beijing Eye Study (BES)** [81],a population based study in 2945 people, was aimed to determine the relation between various ocular disorders and dyslipidemia .They found that dyslipidemia was significantly associated with high IOP and atrophy of beta zone around the optic disc.

**The ADVANCE Study** [82] was done to find the association between HDL cholesterol and micro vascular diseases like retinopathy and nephropathy. In this study they found that HDL cholesterol had significant association in the development of renal micro vascular diseases but not in the retina. LDL cholesterol was found to be associated with the development of hard exudates and DME.

**The Hoorn Study** [83] is a population based study to elaborate the factors for retinopathy in diabetic as well as non-diabetic patients. Positive association with retinopathy was noted with elevated BP, serum cholesterol, triglyceride and increased BMI. Also, increased BP,LDL cholesterol and plasma total cholesterol showed associations with retinal hard exudates.

Study by **Jyothi Idiculla et.al** [84] of St. Johns Medical College, Bangalore, showed that those with increased levels of serum cholesterol and LDL had higher incidences of CSME.

A study was conducted at government general hospital, Gundur, by **Rajasekar et.al**.,[85] to find out the incidence of CSME in DR patients and to find out its associated risk factors. It was found that incidence of CSME increased with the duration of diabetes, level of glycemia control, lipid profiles, hypertension and nephropathy. They concluded that HbA1c

and high cholesterol levels were the most important factors or development of CSME.

In an article by **Sivaramareddy Kolli et,al.** [86],100 diabetic patients were divided equally into those with maculopathy and those without. They observed that, those with maculopathy had a longer history of diabetes and had higher range of serum lipids including serum cholesterol, triglycerides, VLDL and LDL levels ,than those without.

**Rehab Benarous et.al** [87] in their study found that, serum lipids were independently related to CSME, after adjusting for other common risk factors and also that no such correlation was found with the presence and severity of diabetic retinopathy, DME or thickness of the macula.

**Asesnsio Sanchez et.al.** [88]**,** in their study to find out the non ophthalmologic parameters as risk factors of CSME, found that HbA1c level was one of the main risk factors. It was observed that for each 1% increase in HbA1c,the risk of macular edema increased by double. Micro albuminuria also doubled the risk of edema of macula. LDL fraction of cholesterol was seen to increase the risk by a factor of 100%.Tobacco addiction also showed 100% association.

## REVIEW OF LITERATURE CORRELATING HYPOLIPEDEMIC DRUGS AND DR :

Many lipid lowering drugs are under evaluation for their possible protective role in DR.

A study by **Gordon et.al.** done in patients with diabetic retinopathy showed that statins (Pravastatin) is beneficial in improving DR and decreases hard exudate. In a recent study by **Sen et.al**., simvastatin was also found to be helpful in retarding the progression of DR. In another study atorvastatin was given for 4 months after application of laser treatment for CSME .Those patients who received atorvastatin showed prevention of edema extension into the central retina.

**The Collaborative Atorvastatin Diabetes Study (CARDS)**, showed that usage of 10mg of atorvastatin daily lead to a decrease in the laser therapy when compared with those not taking. But there was no effect on the progression of retinopathy.

**The Action to Control Cardiovascular Risks In Diabetes( ACCORD),**participants were divide into 2 groups, one receiving fenofibrate with simvastatin and other group receiving placebo with simvastatin. It was observed that the rate of progression of DR was lower

(6.2%) in those who took fenofibrates when compared to placebo (10.2%).

In a study done on patients with hyperlipoproteinemia, usage of etofibrate for 6 months showed a reduction of hard exudates.

**The Fenofibrates Intervention and Event Lowering in Diabetes (FIELD)** study was a multinational trial done on those with type 2 diabetes, where participants were divided into 2 groups, those receiving fenofibrate and those on placebo. Study showed a decreased requirement of first laser and also lowered the progression rate in those who received fibrate.

# AIM & OBJECTIVES

**AIM :**

To correlate the levels of serum lipid and presence of clinically significant macular edema in diabetic retinopathy patients.

**OBJECTIVES:**

1) To study the serum lipid profile in patients with diabetic retinopathy.

2) To compare the serum lipid profile of patients with and without clinically significant macular edema.

3) To compare and analyze the present study with reference to other studies on serum lipid profile in diabetic retinopathy.

4) To emphasize the importance of doing serum lipid profile as a routine investigation in patients with diabetic retinopathy and to initiate treatment for those appropriate.

# MATERIALS AND METHODS

## STUDY GROUP:

The study was conducted on patients with diabetic retinopathy visiting department of ophthalmology, Coimbatore Medical College Hospital, Coimbatore.

## PERIOD OF STUDY:

The study was conducted for a period of one year from January 2017 to December 2017.

## INCLUSION CRITERIA:

Patients aged more than 40 years having type 2 diabetes mellitus with retinopathy, in euglycemic status during the time of study, attending ophthalmology outpatient department and are willing to participate in the study and have given consent to undergo blood tests for serum lipid levels and renal parameters, were included in the study.

## EXCLUSION CRITERIA:

1) Patients with type 1 diabetes mellitus.

2) Patients with any pre existing retinal manifestations of systemic diseases, other than DR.

3) Patients who have had treatment for diabetic retinopathy

4) Patients on treatment for dyslipidemias.

5) Patients with macular dystrophies and degenerations.

6) Patients with ocular anomalies.

7) Patients with ocular trauma.

**SAMPLE SIZE:**

200 patients divided in to two groups of 100 each.

Group A: Patients with diabetic retinopathy with clinically significant macular edema.

Group B: Patients with diabetic retinopathy without clinically significant macular edema.

**SELECTION OF STUDY SUBJECTS:**

Hundred consecutive patients each of Group A and Group B presenting to ophthalmology outpatient department and those referred from other departments with diabetic retinopathy fulfilling the inclusion criteria of the study were selected.

**METHODS:**

Informed written consent was obtained from the selected patients and data collected using structured questionnaire which comprises socio-demographic characteristics like age, gender, history of duration of diabetes mellitus, history of usage of insulin, duration of insulin usage, treatment history, presence of other systemic conditions like hypertension and renal disease.

Anterior segment evaluation was done by slit lamp examination. After dilatation of the pupil with tropicamide and phenylephrine eye drops, fundus examination was done using direct ophthalmoscopy, indirect ophthalmoscopy and slit lamp bio microscopy with +90D lens. Appropriate fundus photographs and FFA were taken. Diabetic retinopathy was classified according to ETDRS classification.

All patients were advised overnight fasting and blood samples were taken for estimation of fasting blood glucose, serum lipid levels, blood urea and serum creatinine. Systolic and Diastolic Blood pressures were measured in sitting posture. Those patients with higher fasting blood glucose values in initial recording were included in the study only after maintaining a euglycemic status for 1 month.

# RESULTS AND OBSERVATIONS

The data analysis was performed using statistical software package SPSS version 22.0.Both the descriptive and inferential statistics were used. The continuous variables were summarized as mean with standard deviation. The categorical variables were summarized as frequencies and proportions. The comparison of continuous variables were done using unpaired ‘t’ test and comparison of categorical variables were done using Chi square test or Fisher’s exact test depending on distribution. P value of less than 0.05 was considered significant.

Group A : Patients with clinically significant macular edema

Group B : Patients without clinically significant macular edema

**Table -1: Age Distribution of Cases**

| Age groups (in years) | Group A | | Group B | |
|---|---|---|---|---|
| | No. | Percentage | No. | Percentage |
| 40- 50 | 14 | 14 | 16 | 16 |
| 51 -60 | 41 | 41 | 46 | 46 |
| 61 – 70 | 34 | 34 | 32 | 32 |
| 70-80 | 10 | 10 | 6 | 6 |
| >80 | 1 | 1 | 0 | 0 |
| Total | 100 | 100 | 100 | 100 |
| Range | 40 – 87 yrs | | 42 – 77 yrs | |
| Mean | 60.07 yrs | | 58.78 yrs | |
| S.D | 8.41 | | 7.77 | |
| P | 0.701 | | Not Significant | |

Patients with CSME had mean age of 60.07 years and without CSME 58.78 years

Age was not found to be significantly associated with the presence of CSME.(p=0.701)

**Chart- 1:Age wise distribution**

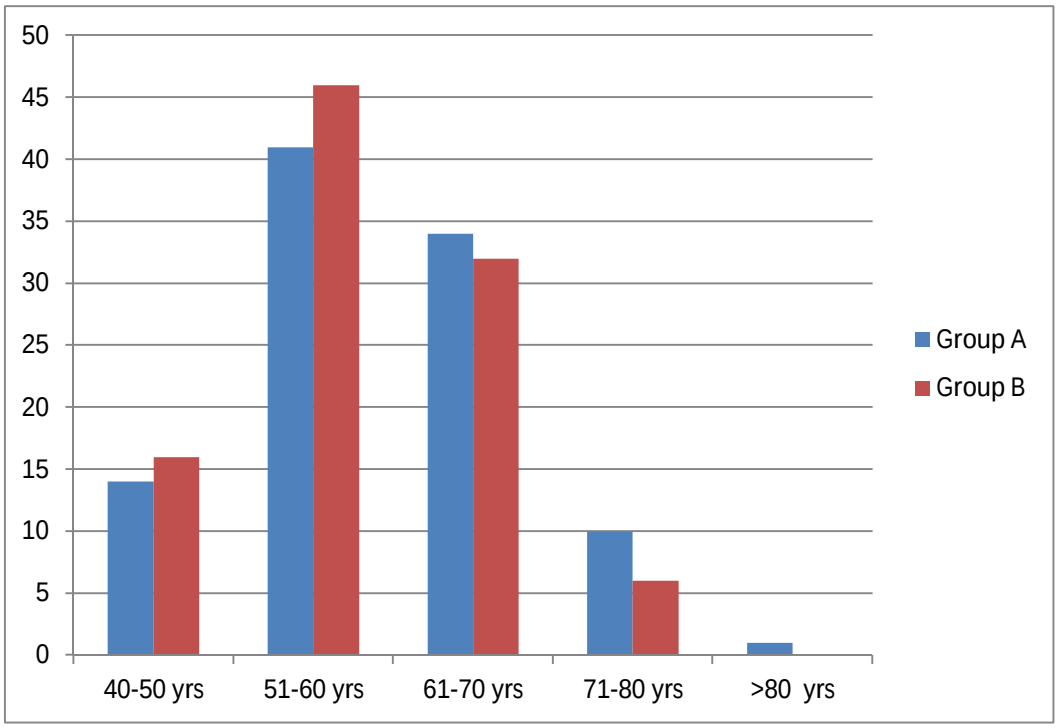


**Table -2: Sex Distribution of Cases**

| Sex | Group A | | Group B | |
|---|---|---|---|---|
| | No. | Percentage | No. | Percentage |
| Male | 57 | 57 | 50 | 50 |
| Female | 43 | 43 | 50 | 50 |
| Total | 100 | 100 | 100 | 100 |
| P | 0.321 Not significant | | | |

Males slightly predominated the group with CSME with 57% and both were equal in numbers in the non CSME group.

**Chart-2:Sex distribution**

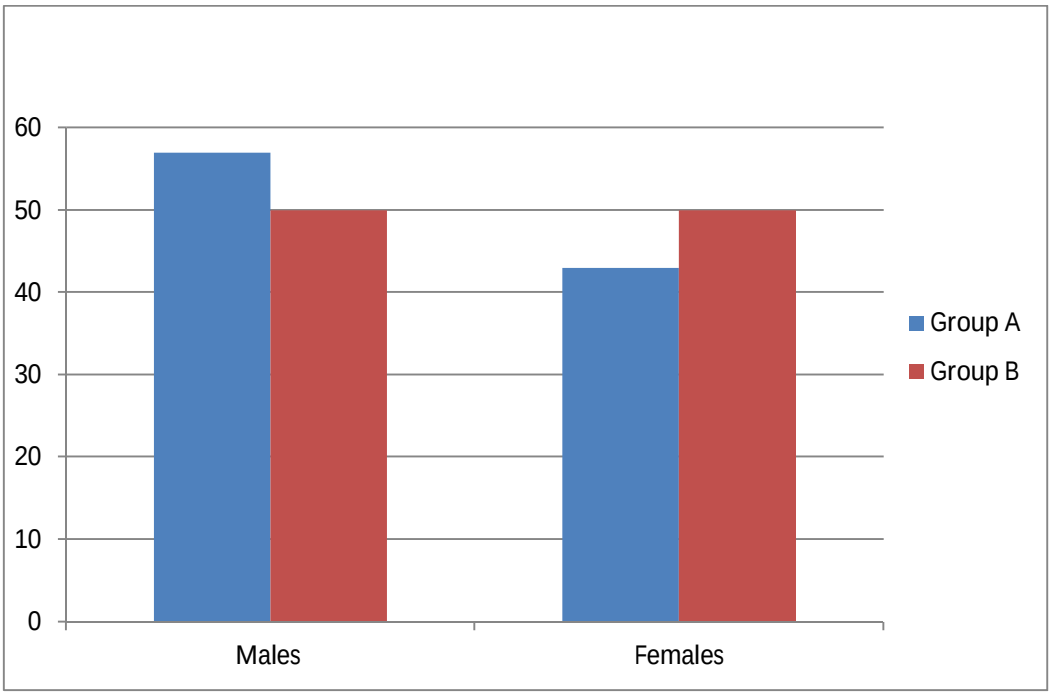


**Table -3: Duration of DM**

| Duration of DM (in years) | Group A | | Group B | |
|---|---|---|---|---|
| | No. | Percentage | No. | Percentage |
| Newly detected | 11 | 11 | 14 | 14 |
| Up to 5 years | 2 | 2 | 17 | 17 |
| 6 – 10 years | 31 | 31 | 68 | 68 |
| 11 – 15 years | 41 | 41 | 1 | 1 |
| > 15 years | 15 | 15 | 0 | 0 |
| Total | 100 | 100 | 100 | 100 |
| P value (< 15 yrs. to > 15yrs) | 0.0000 | | Significant | |

41% of the patients with CSME had type 2 Diabetes Mellitus for duration ranging from 11-15 years whereas most of the patients without CSME had diabetes mellitus for a duration ranging from 6-10 years comprising 68% of the total. Duration of DM showed statistical significance

## Chart-3:Duration of diabetes with CSME

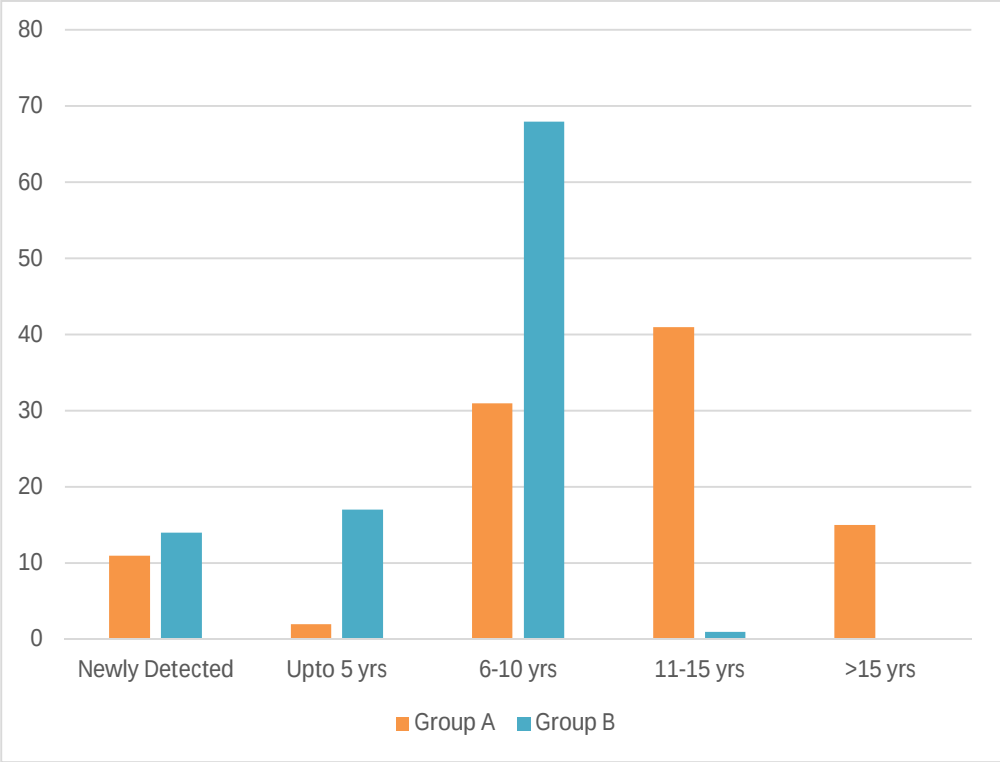


**Table -4: Insulin usage**

| Insulin use | Group A | | Group B | |
|---|---|---|---|---|
| | No. | Percentage | No. | Percentage |
| Yes | 44 | 44 | 47 | 47 |
| No | 56 | 56 | 53 | 53 |
| P | 0.670 Not significant | | | |

Among the patients with CSME, 44% were on insulin and 47% among the patients without CSME were on insulin. Insulin usage was not found to be significantly related to presence or absence of CSME.

**Chart-4:Insulin Usage**

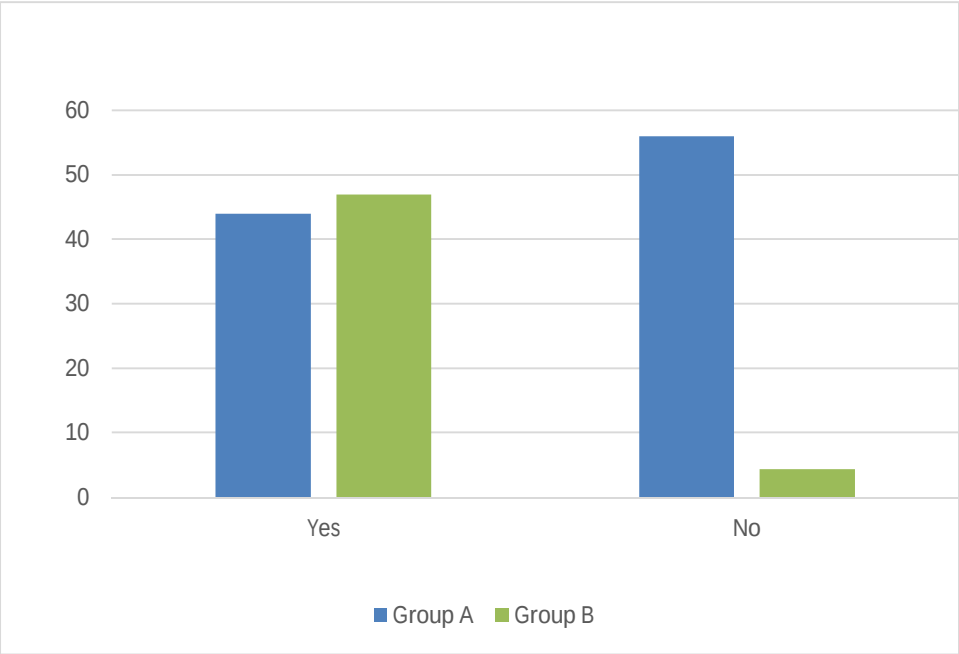
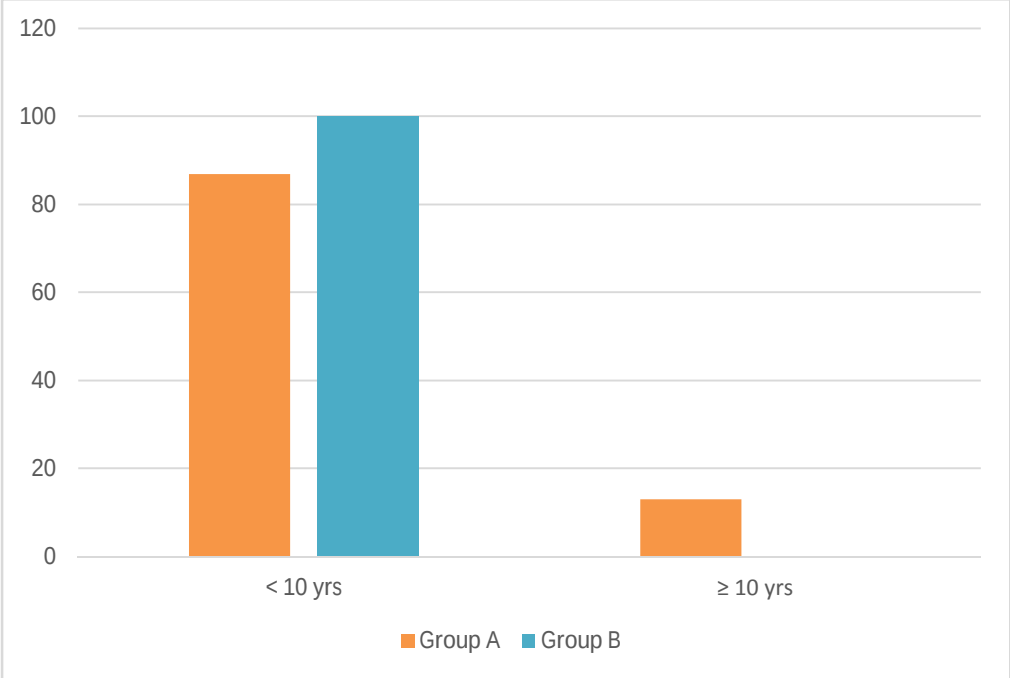


**Table -5: Duration of insulin usage**

| Duration | Group A | | Group B | |
|---|---|---|---|---|
| | No. | % | No. | % |
| < 10 yrs | 87 | 87 | 100 | 100 |
| ≥10yrs | 13 | 13 | 0 | 0 |
| p | 0.0000 Significant | | | |

Duration of insulin usage for more than 10 years showed statistical significance to the development of CSME(p=0.0000)

**Chart-5:Duration of insulin usage**



## Table -6: Severity of Diabetic Retinopathy

| DIABETIC RETINOPATHY SEVERITY | RIGHT EYE | | | | LEFT EYE | | | |
|---|---|---|---|---|---|---|---|---|
| | A | | B | | A | | B | |
| | No. | % | No. | % | No. | % | No. | % |
| No DR | 2 | 2 | 17 | 17 | 4 | 4 | 20 | 20 |
| Very Mild NPDR | 2 | 2 | 7 | 7 | 6 | 6 | 5 | 5 |
| Mild NPDR | 7 | 7 | 36 | 36 | 12 | 12 | 45 | 45 |
| Moderate NPDR | 38 | 38 | 29 | 29 | 45 | 45 | 23 | 23 |
| Severe NPDR | 19 | 19 | 9 | 9 | 16 | 16 | 4 | 4 |
| Very Severe NPDR | 14 | 14 | 0 | 0 | 7 | 7 | 0 | 0 |
| PDR | 18 | 18 | 2 | 2 | 10 | 10 | 3 | 3 |

Those with CSME and without CSME had most of the patients with Moderate NPDR.

**Chart-6:Severity of diabetic retinopathy in right eye**

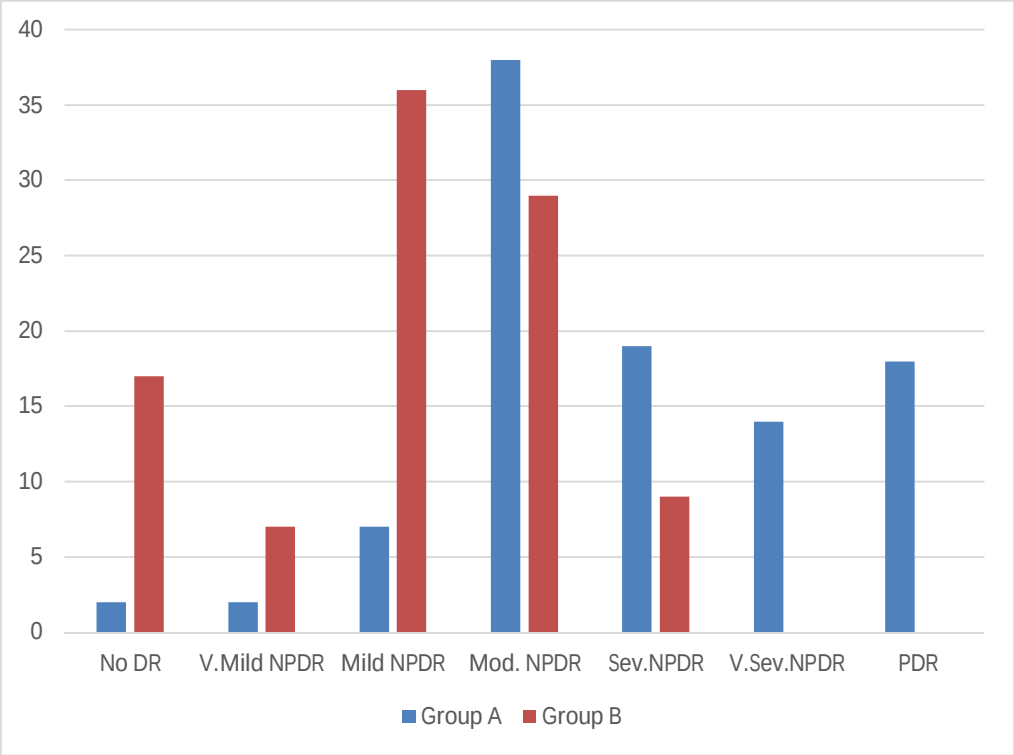



**Chart-7:Severity of diabetic retinopathy in left eye**

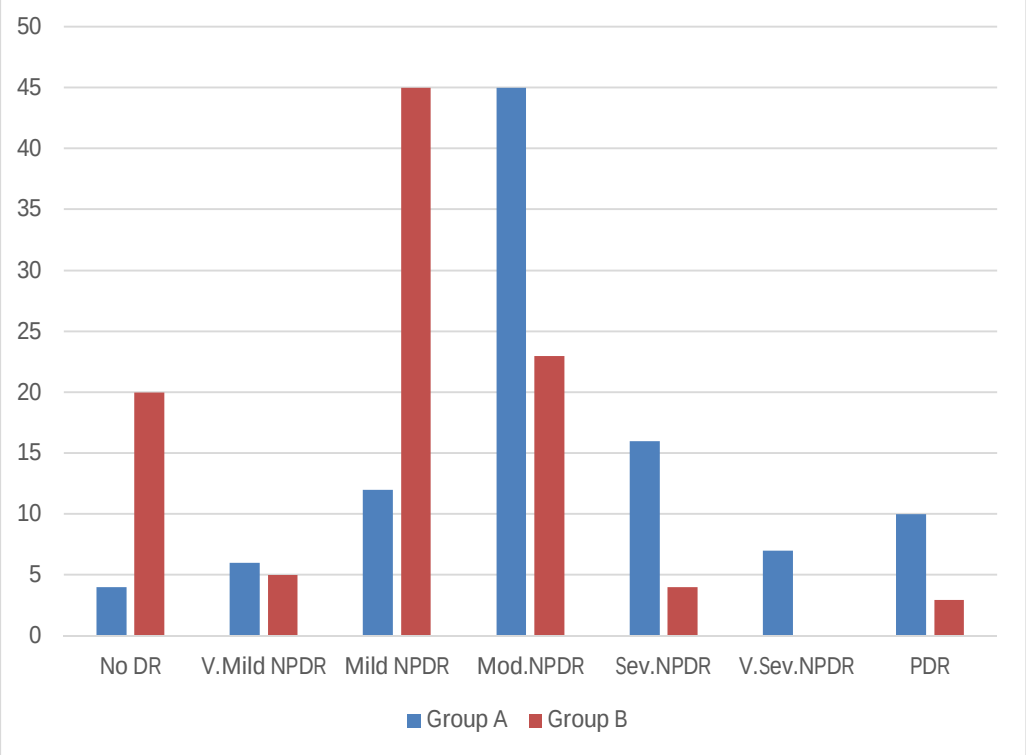


**Table -7:Eye wise distribution:**

| CSME | Right Eye | | | | Left Eye | | | |
|---|---|---|---|---|---|---|---|---|
| | A | | B | | A | | B | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 69 | 69 | 0 | 0 | 62 | 62 | 0 | 0 |
| No | 31 | 31 | 100 | 100 | 38 | 38 | 100 | 100 |

Among the patients who had CSME, it was more common in right eye in our study than in left eye.

## B. Relationship between Lipid Profile and incidence of CSME in patients with Diabetic Retinopathy

### Table -8: Total cholesterol and CSME

| Total Cholesterol | Group A | | Group B | |
|---|---|---|---|---|
| | No. | Percentage | No. | Percentage |
| Normal (≤200mg/dl) | 5 | 5 | 57 | 57 |
| Abnormal (>200mg/dl) | 95 | 95 | 43 | 43 |
| Range | 140-518 | | 112 – 412 | |
| Mean | 318.69 | | 199.57 | |
| SD | 63.44 | | 49.52 | |
| P | 0.0000 | | Significant | |

Total cholesterol levels in patients of Group A ranged from 140 – 518 mg/dl with a mean of 318.69 mg / dl and Group B from 112 – 412 mg / dl with a mean of 199.57 mg / dl. Total cholesterol level was found to be statistically significant with a ‘p’ value of 0.0000.

## Chart-8:Correlation of total cholesterol and CSME

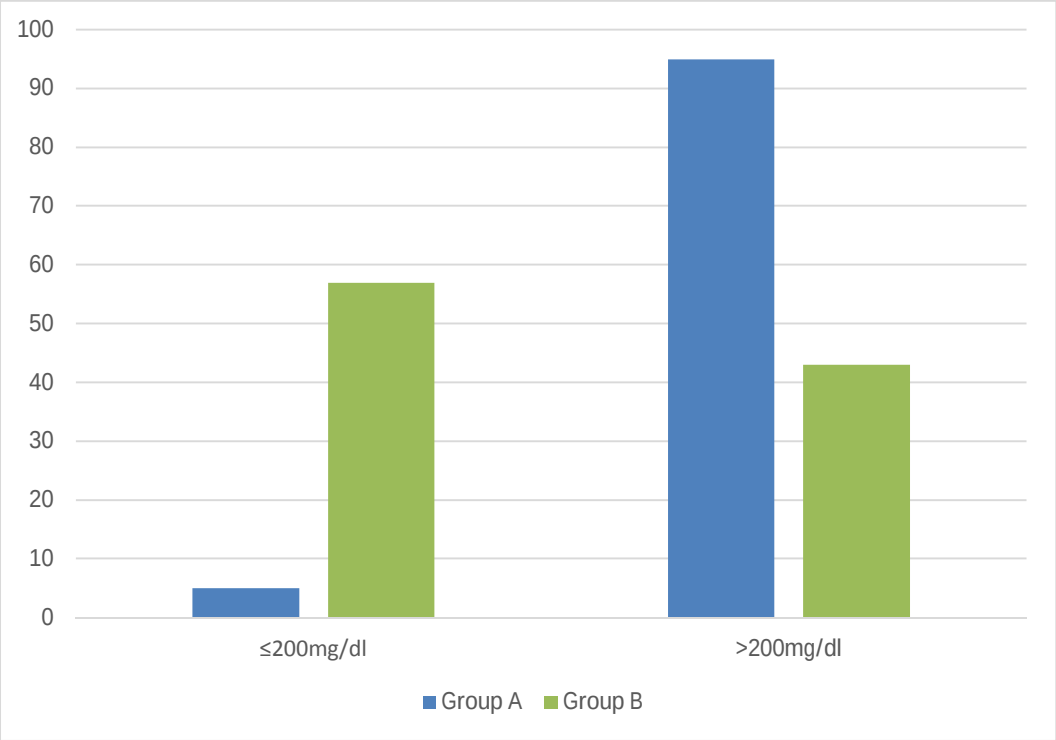


**Table -9: Triglyceride and CSME**

| TG | Group A | | Group B | |
|---|---|---|---|---|
| | **No.** | **Percentage** | **No.** | **Percentage** |
| Normal (≤150mg/dl) | 5 | 5 | 34 | 34 |
| Abnormal (>150mg/dl) | 95 | 95 | 66 | 66 |
| Range | 123 – 380 | | 78 – 338 | |
| Mean | 257.07 | | 164.89 | |
| SD | 57.54 | | 40.62 | |
| P | 0.0000 | | Significant | |

Patients without CSME had a mean triglyceride levels of 164.89 mg/dl whereas patients with CSME had a higher triglyceride levels with a mean of 257.07 mg / dl with the 'p' value of 0.0000, .Increased triglyceride levels were found to be significantly related to presence of CSME.

## Chart -9: Correlation of Triglyceride and CSME

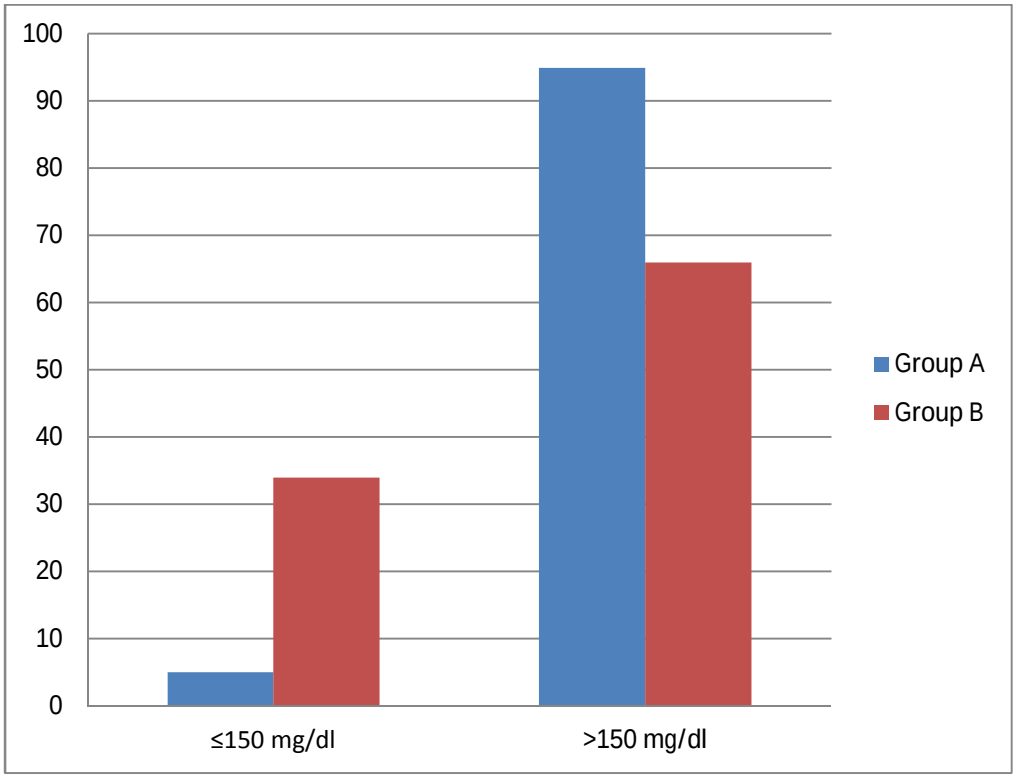


**Table -10: HDL and CSME**

| HDL | Group A | | Group B | |
|---|---|---|---|---|
| | **No.** | **Percentage** | **No.** | **Percentage** |
| Normal (> 40mg/dl) | 15 | 15 | 85 | 85 |
| Abnormal (≤40mg/dl) | 85 | 85 | 15 | 15 |
| Range | 12– 196 | | 22 – 68 | |
| Mean | 35.49 | | 49.13 | |
| SD | 25.03 | | 8.93 | |
| P | 0.0000 | | Significant | |

Patients with CSME had mean HDL-C levels of 35.49 mg/dl and those who did not have CSME had a mean of 49.13 mg / dl. Decreased serum HDL-C level was found to be significantly related to the presence of CSME.

## Chart-10:Correlation of HDL cholesterol and CSME

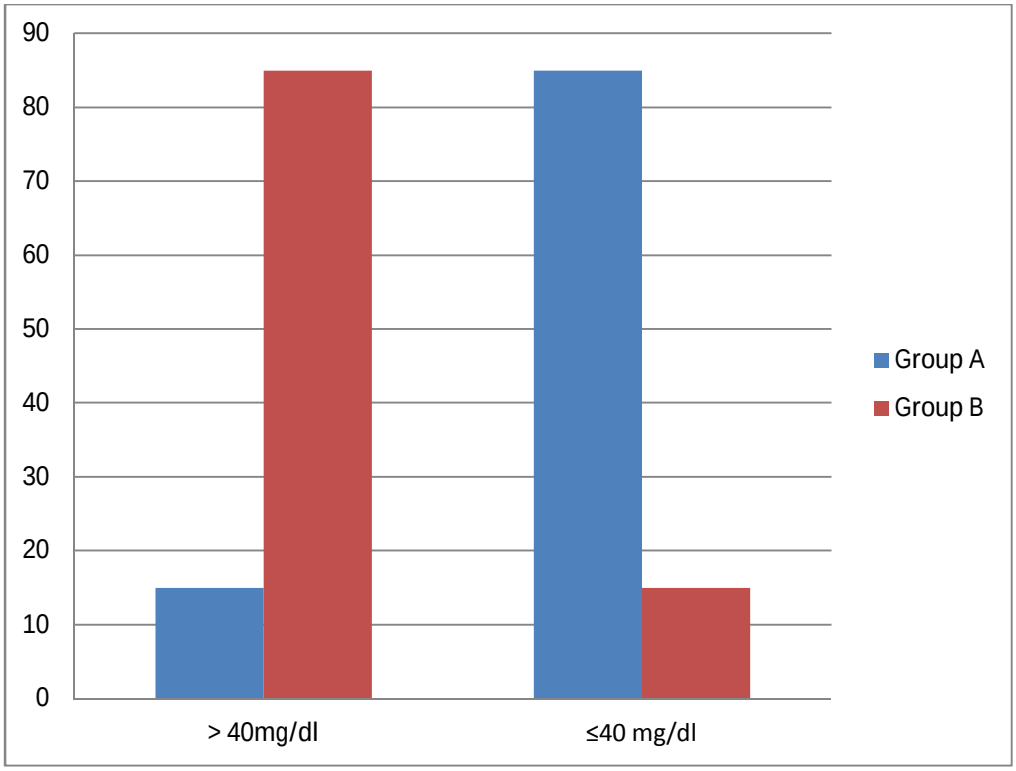


**Table -11: LDL and CSME**

| LDL | Group A | | Group B | |
|---|---|---|---|---|
| | **No.** | **Percentage** | **No.** | **Percentage** |
| Normal (≤ 130mg/dl) | 19 | 19 | 82 | 82 |
| Abnormal (>130mg/dl) | 81 | 81 | 18 | 18 |
| Range | 26- 402 | | 54 – 240 | |
| Mean | 195.48 | | 107.73 | |
| SD | 69.80 | | 37.45 | |
| P | 0.0000 | | Significant | |

Patients with CSME had higher serum LDL-C levels with a mean of 195.48 mg / dl compared to patients without CSME who had 107.73 mg% which was identified to be statistically significant.(0.0000)

## Chart-11: Correlation of LDL cholesterol and CSME

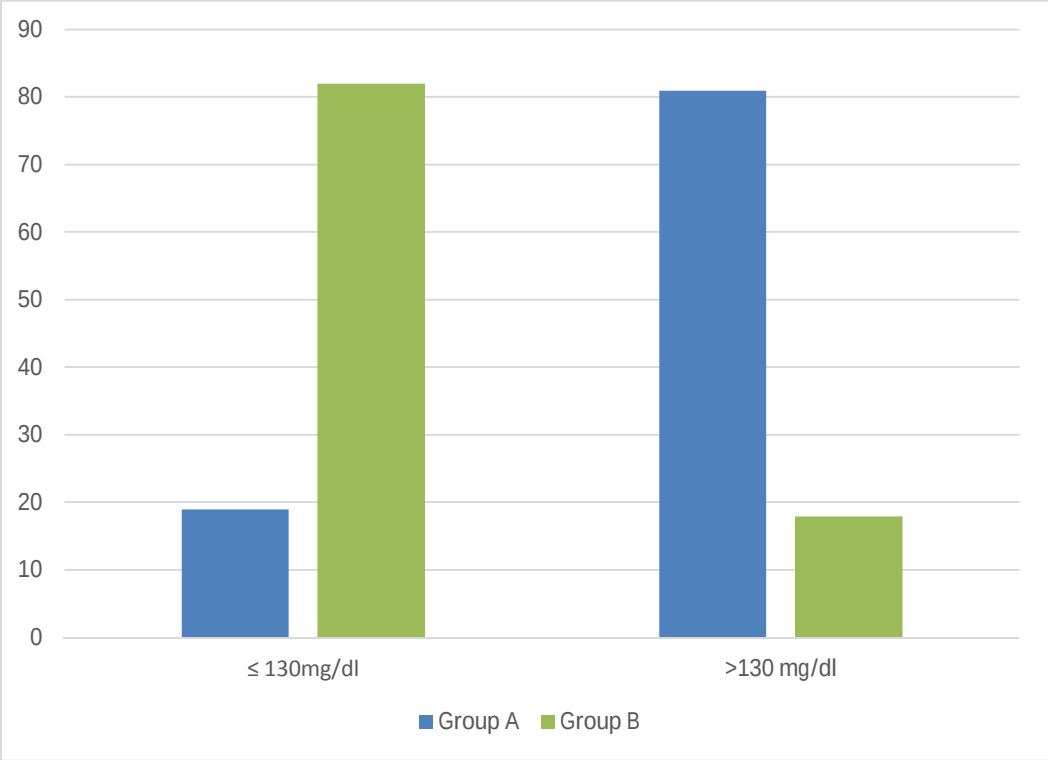


**Table -12: Blood Urea and CSME**

| Blood Urea | Group A | | Group B | |
|---|---|---|---|---|
| | **No.** | **Percentage** | **No.** | **Percentage** |
| Normal (≤ 23mg/dl) | 16 | 16 | 26 | 26 |
| Abnormal (>23mg/dl) | 84 | 84 | 74 | 74 |
| Range | 21 – 38 | | 17 – 70 | |
| Mean | 26.82 | | 28.35 | |
| SD | 3.23 | | 9.23 | |
| P | 0.1197 | | Not Significant | |

Patients in group A and B had blood urea level on the higher side and analysis showed no statistical significance.(p=0.1197)

## Chart-12: Correlation of Blood urea and CSME

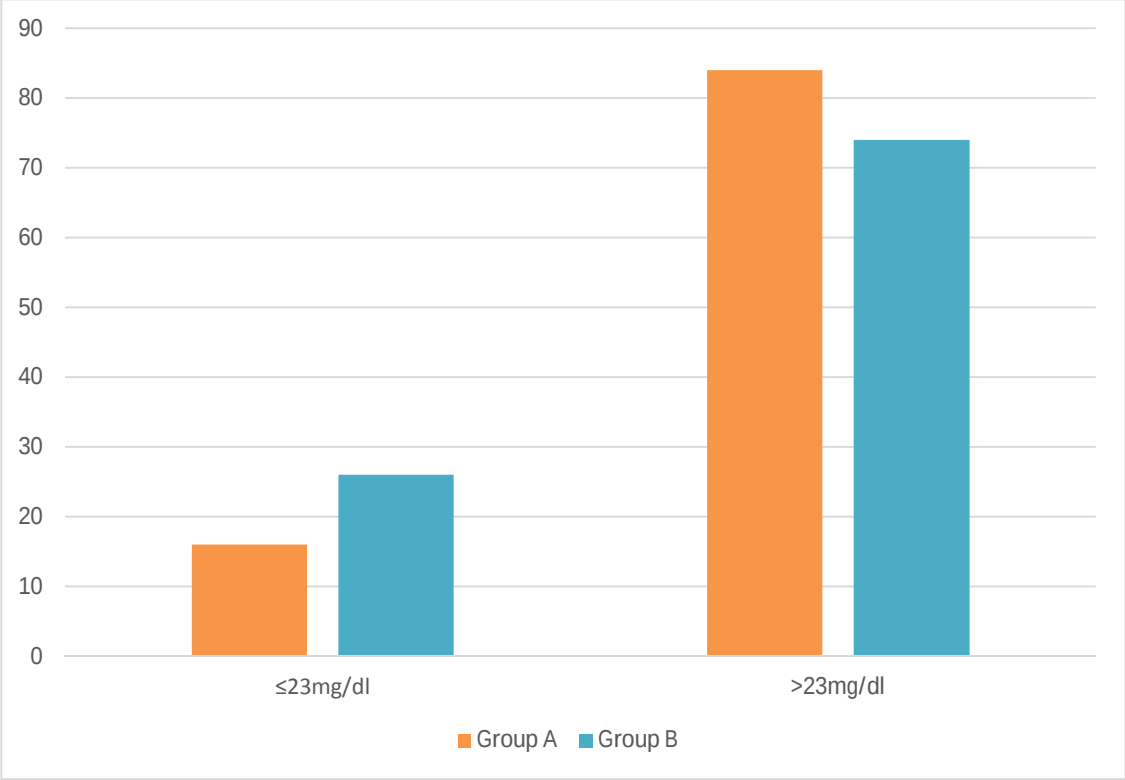


**Table -13: Serum Creatinine and CSME**

| Serum Creatinine | Group A | | Group B | |
|---|---|---|---|---|
| | **No.** | **Percentage** | **No.** | **Percentage** |
| Normal (≤1.3mg/dl) | 89 | 89 | 87 | 87 |
| Abnormal (>1.3mg/dl) | 11 | 11 | 13 | 13 |
| Range | 0.7– 2.2 | | 0.6 – 4.3 | |
| Mean | 1.003 | | 1.089 | |
| SD | 0.33 | | 0.66 | |
| P | 0.2470 | | Not Significant | |

Patients with and without CSME were found to have decreased serum creatinine levels with mean value being 1.003 mg/dl and 1.089 mg/dl, respectively. It showed no statistical significance.(p=0.2470)

## Chart-13: Correlation of Serum creatinine and CSME

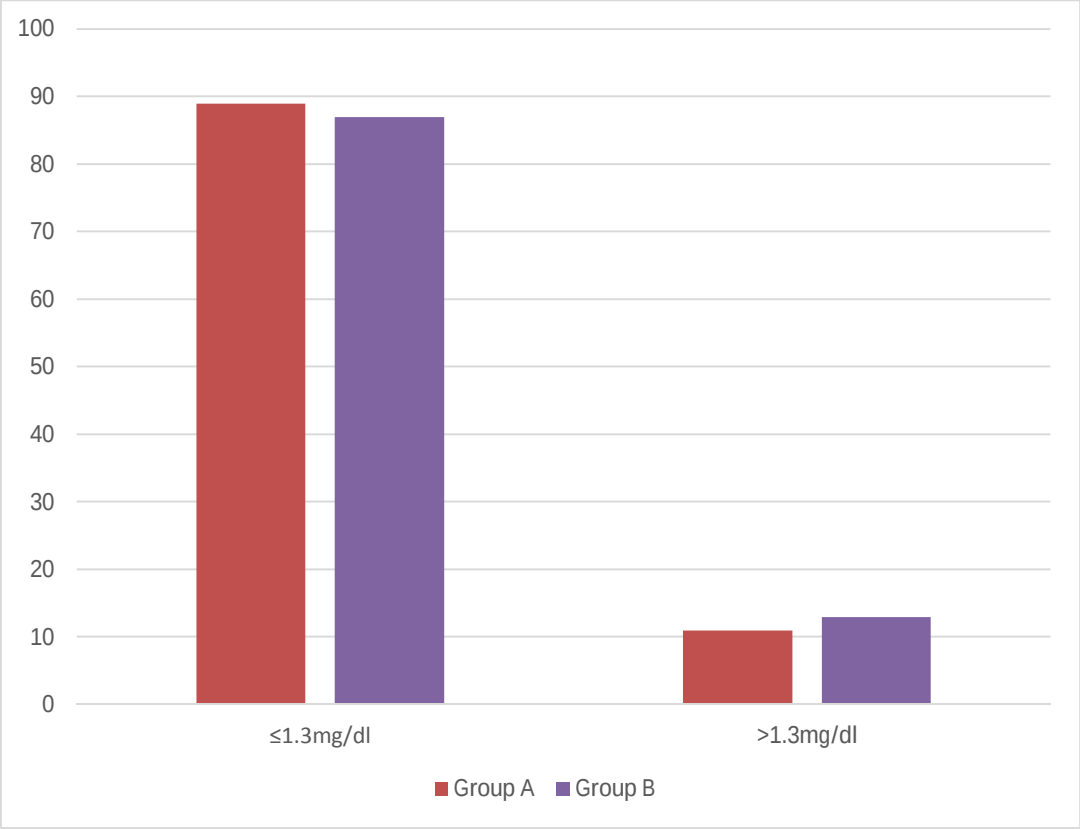
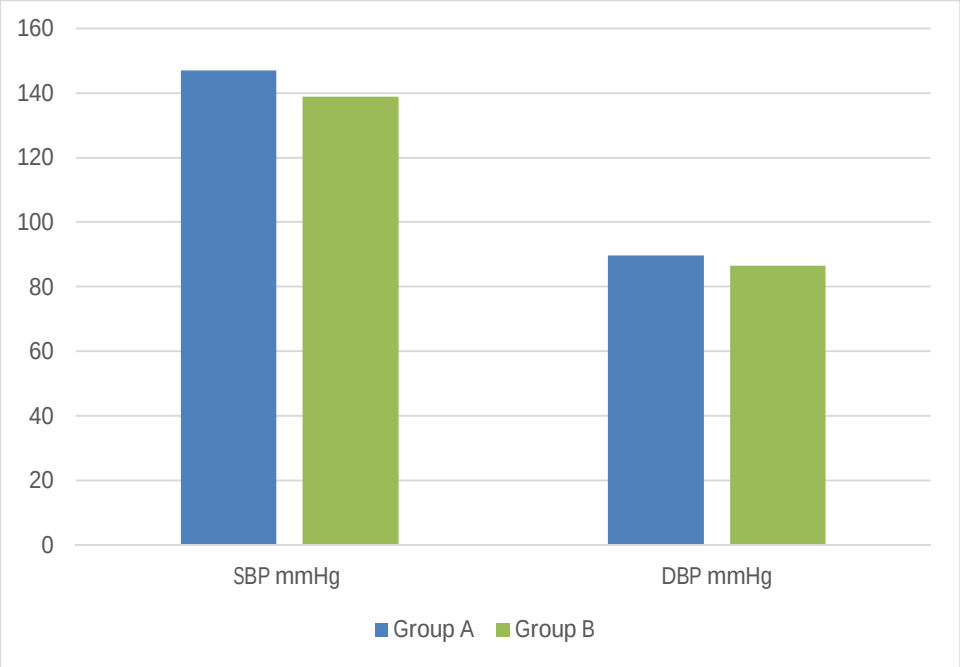


**Table -14: Blood Pressure and CSME**

| TGL | Group A | | Group B | | 'p' |
|---|---|---|---|---|---|
| | **Mean** | **SD** | **Mean** | **SD** | |
| Systolic BP(mm Hg) | 147.12 | 16.89 | 138.94 | 14.71 | 0.0003 Significant |
| Diastolic BP(mm Hg) | 89.78 | 8.85 | 86.6 | 7.18 | 0.0058 Significant |

Patients in both the groups had a mean SBP and DBP higher than normal values and showed statistical significance(P=0.0003 and P=0.0058 respectively)

## Chart-14: Correlation of Blood pressure and CSME



# DISCUSSION

Diabetes Mellitus is an important health problem affecting the whole world. Its impact is on the increase for the past many years, with 326.5 million people suffering from it as per the 2017 records. Diabetic Macular Edema forms the leading cause of blindness among the patients with diabetic retinopathy. There have been many studies correlating the severity of diabetic retinopathy and the various risk factors, including level of serum lipids. Studies also have been reported regarding the efficiency of hypolipidemic drugs in decreasing the severity of retinopathy, maculopathy and vision loss. This study was done in our setting to observe how much the lipid level varies in those with CSME and those without in our region and to stress the importance of lipid lowering as a part of diabetic retinopathy management.

Most of the results obtained in this study correlated with the available literature, both in the global scenario and the Indian set up. In the previously published studies, it was observed that the prevalence of diabetic retinopathy increases with duration of diabetes. In our study also DR severity increased with the number of years the patients has had DM. In our study, the most commonly seen stage of retinopathy was that of moderate NPDR, among those with CSME and without CSME. Among

the newly detected cases of diabetes most (75 %) had moderate NPDR and severe grades of retinopathy either in one or both eyes in both the groups. In the CSME group,92% had moderate NPDR and higher grades, whereas in the non CSME group, it was 50%.Out of the 21 newly detected cases,11 had CSME in either one or both eyes. This was observed to be in contrast with the Blue mountain study, where CSME was low (2%) in the newly diagnosed cases. This can be attributed to the fact that most patients especially from the rural areas get their diabetes detected after ophthalmoscopic examination when they present with complaints diminution of vision to ophthalmology OPD.

Those with CSME had most number of cases in the 11-15 years duration group and those without CSME had maximum number of patients in the 6-10 years duration group. The significance of duration of diabetes is well evident from its correlation to CSME development.($p=0.0000$).

Analysis of the age distribution of patients showed that most people were in the $5^{th}$ decade and that age and sex preponderance analysis did not show any statistical significance in the current study . Results of the important WESDR study also showed that the duration of diabetes is more significant risk factor than the age of onset of diabetes.

Thirty three eyes, out of the 400 eyes studied showed PDR,21 eyes showed very severe NPDR and 48 eyes had severe NPDR. Moderate NPDR accounted for the maximum (135 eyes).Mild and very mild NPDR was seen in 100 eyes and 20 eyes, respectively. Forty three eyes did not show any retinopathy changes.

Among those with CSME, 84 eyes(42%) had severe NPDR and higher grades of retinopathy ,while those without CSME had severe forms of retinopathy in 18 eyes(9%).Most of the patients in both the groups had moderate NPDR.41.5% of eyes had moderate NPDR in the CSME group and26% of eyes had moderate NPDR in the non CSME group.

In our study it was noted that not only the prevalence but also the severity of retinopathy also increases with the duration of diabetes. In the WESDR ,it was found that type 1 DM patients with more than 5 years of diabetes had 13% prevalence of retinopathy and severe forms of retinopathy i.e., PDR was rarely found among these people. It was also seen that when the duration increased to 10-15 years, more than 90% had retinopathy and many(25%) showed proliferative changes. Type 2 DM patients were noted to have earlier onset of retinopathy changes and also higher grades of severity in earlier years itself.

In our study,91 patients were using insulin compared with 109 people not using insulin (either newly detected or on OHAs).Those on insulin and having CSME was found to be 44 in number(48%%),while those on insulin and did not develop CSME made 56 in number accounting for 51.3%. Only13% of people using insulin for 10 years or more had CSME and none were using for 10 or more years in the CSME group. The analysis of duration of insulin usage was found to be statistically significant in the occurrence of CSME (p=0.0000)

It has been suggested that exogenous insulin may be a possible cause of atherosclerosis and retinopathy in people with type 2 diabetes. In the WESDR, there was no association between the amount or type of exogenous insulin used and the presence, severity, incidence or progression of retinopathy in the older onset group using insulin.

Clinically Significant Macular Edema (CSME) was found to be more in the right eye.

In the assessment of the study proper, it was observed that those with CSME had significantly increased levels of total cholesterol with an average value of 318.69mg/dL. Those without CMSE were found to have high values of total cholesterol but were less than that of those with CSME. The difference between the two groups was significant

statistically(p=0.0000).In a study done by Klein R et. al., it was observed that in type 2 diabetes group that did not use insulin, there was no relationship between total cholesterol and severity of hard exudates.

In the ETDRS also those with elevated serum cholesterol had more chance of developing hard exudates. Study by Ucgun et.al., also had similar conclusion of positive association of serum cholesterol and exudative maculopathy.

SN-DREAMS and the Hoorn study had also concluded that total cholesterol level as significantly associated with development of CSME.

In the Indian scenario also, separate studies done at Sankara Nethralaya and by Jyothi Idiculla et.al., had observed the significant correlation of total cholesterol and CSME.

Triglyceride levels in our study were observed to be higher in patients with CSME when compared with those without CSME. In the comparison of the 2 groups, statistical significance was present.(p=0.0000) .This as in contrast to the MESA study and study by Ucgun et.al., where the authors could not find any significant association between CSME and triglyceride levels. In a study done on the North Indian population by Sachdev N et.al., the author could find a significant association.

Lower levels of HDL-C was found to be associated with the development of CMSE in our study and was statistically significant.(p=0.0000).In the MESA study and study by Ucgun et.al., showed no significant association between HDL-C levels and presence of CSME.

Higher levels of mean LDL-C (195.48mg/dl) were found in the group with CSME when compared with the non CSME group(107.73mg/dl) and was found to be statistically significant(p=0.0000).Similar findings were observed in the ARICS study, the CHS study ,the Hoorn study and the MESA study. In the Indian scenario also , the CURES study done in South of India and a study by Sachdevn et.al., from North India also showed similar results. In another study done in the South Indian population ,SN-DREAMS, no correlation was observed between the levels of LDL-C and CSME.

In a study done at Macedonia by Golubovic-Arsovska, eventhough the levels of LDL-C was higher in those with CSME, there was no significant correlation observed.

Analysis of the blood urea levels and serum creatinine levels in our study population showed no significant correlation with CSME, with 'p' values 0.1197 and 0.2470 respectively. This was similar to study

published by Jew OM et.al., where they had found no significant association of blood urea and serum creatinine even though the values obtained in their study were high. A study by Rajasekhar P et.al., found a significant association of serum creatinine with the development of CSME.

Those with CSME had a mean Systolic blood pressure of 147.12mm of Hg and a mean diastolic blood pressure of 89.78 mm of Hg. Those without CSME had a mean SBP of 138.94 mm of Hg and a mean DBP of 8.6 mm of Hg. There is a significant association between both SBP and DBP with the development of CSME(p=0.0003 and 0.0058)

# SUMMARY

The study “An analytical study correlating the significance of serum lipids in the development of clinically significant macular edema in patients with diabetic retinopathy” was a case control study with 200 participants with diabetic retinopathy who had presented to the outpatient department of Coimbatore Medical College Hospital, Coimbatore.

Patients were selected based on the inclusion criteria and exclusion criteria. A detailed history regarding the duration of diabetes, history and duration of insulin usage, duration of hypertension were taken. Patients were examined using direct ophthalmoscope, slit lamp bio microscopy with + 90 D lens and with indirect ophthalmoscope.Colour fundus photographs were recorded and FFA was done. Blood investigations including fasting lipid profile, blood urea and serum creatinine were done for the selected subjects. Blood pressure was recorded. The above said variables were compared between those with and those without CSME.

Findings from the study concluded that there was no significant difference in the age and sex distribution between the 2 groups. Duration of diabetes showed a significant association with the severity of diabetic retinopathy as well as the development of CSME. Insulin usage showed

no significance whereas and duration of insulin usage showed significant association in the development of CSME.

Most of the patients with and without CSME were seen to be having moderate NPDR. In our study, it was observed that significantly higher levels of total cholesterol, triglycerides, LDL-C and significantly lower levels of HDL-C were found among the patients with CSME when compared to those without CSME.

Blood urea and serum creatinine were not found to be significantly associated with CSME.

Both the systolic blood pressure and the diastolic blood pressure were found to be significantly higher among those with CSME when compared with those not having CSME.

# CONCLUSION

The present study gives a good evidence of a significant correlation between higher levels of total cholesterol, triglycerides and LDL-C and lower levels of HDL-C with the presence of clinically significant macular edema. Hypertension is also associated with CSME.

Most of the diabetic patients are getting only oral hypoglycemics or insulin for the treatment of diabetes. The significance of lipid levels and the severity of hypertension in the development of exudative maculopathy is seen in the present study. Hence along with diabetes control drugs, lipid lowering drugs and anti-hypertensive drugs should be started for diabetic patients having dyslipidemia and hypertension. CSME is a vision threatening complication of diabetic retinopathy. Therefore, in order to prevent the loss of vision and to preserve or improve current visual acuity in those who have already developed CSME, adequate control of hyperglycemia, hypertension and dyslipidemia must be ensured, in every diabetic individual.

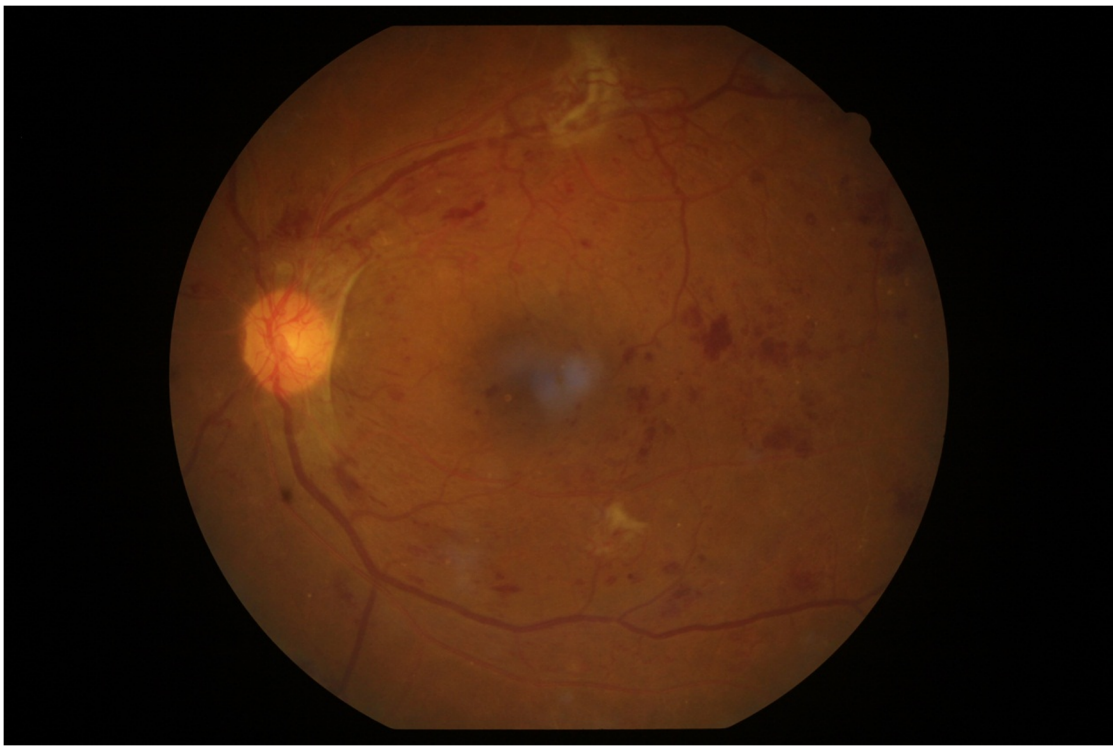


**Fig:1 Proliferative diabetic retinopathy without CSME**

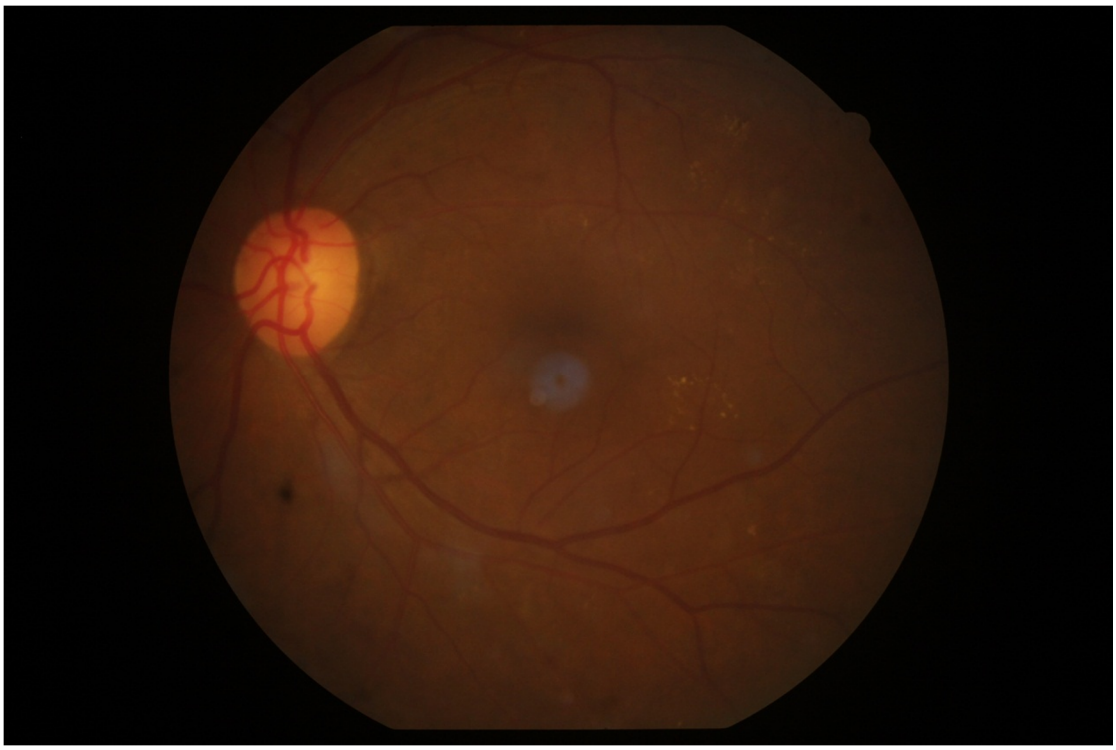


**Fig-2: Moderate NPDR with CSME with focal leakage**

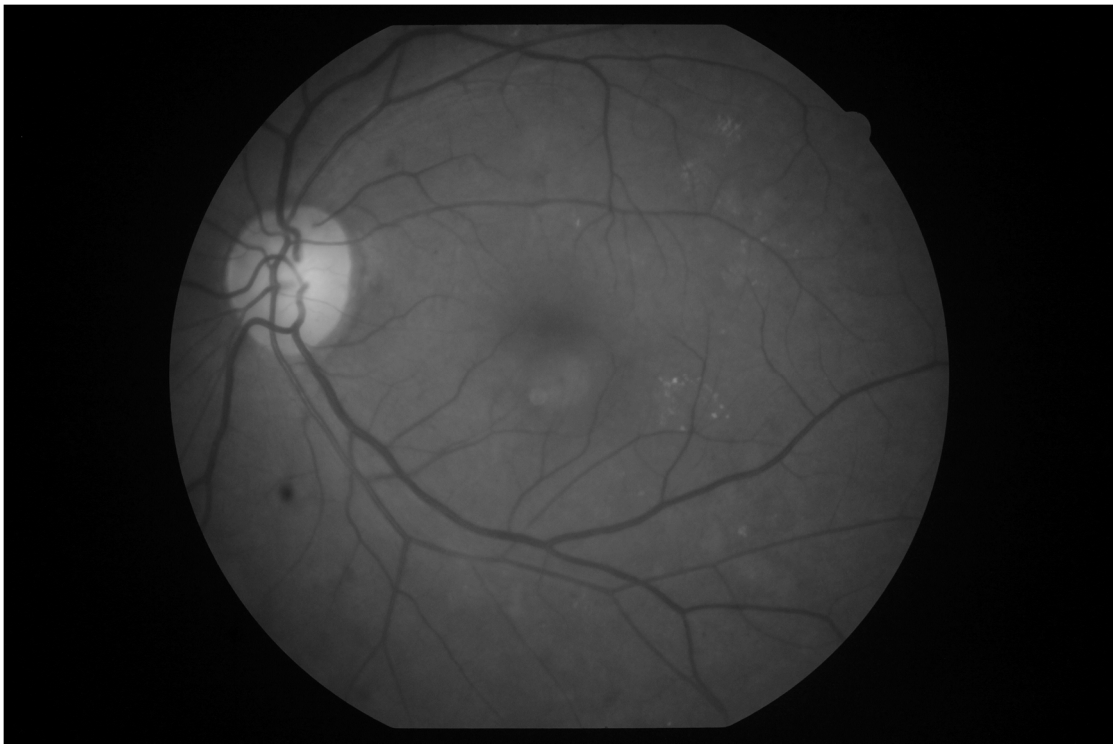


**Fig-3:Red free image of fig.2**

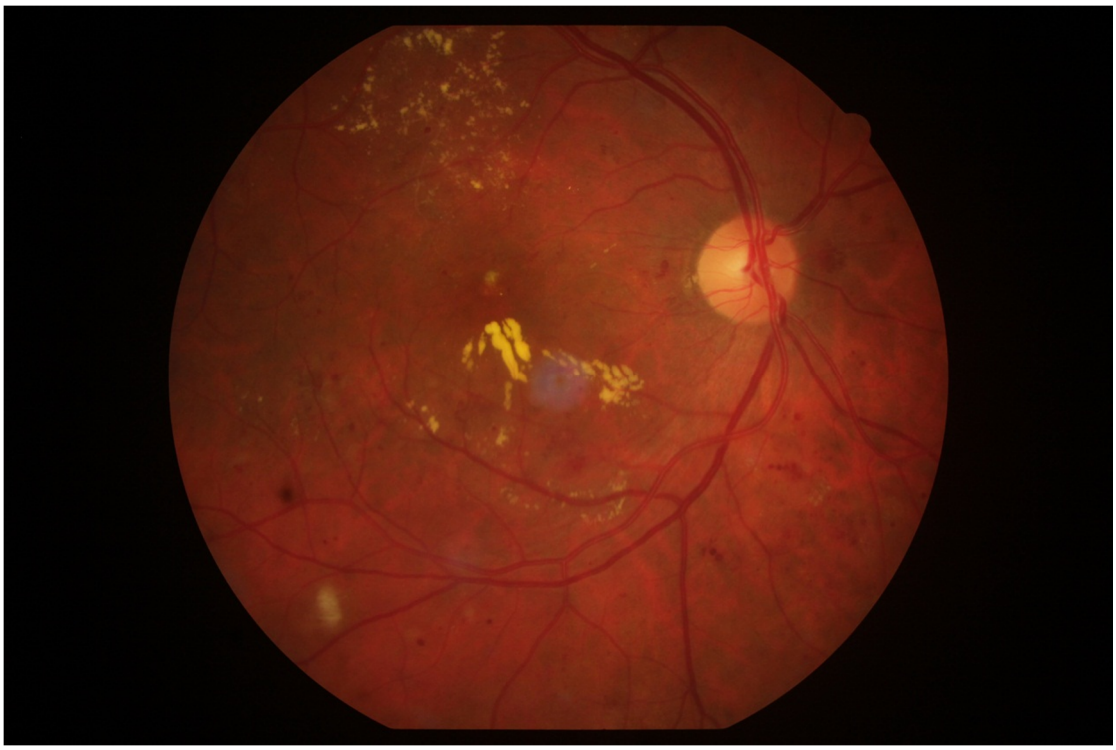


**Fig-4:Moderate NPDR with CSME with diffuse leakage**

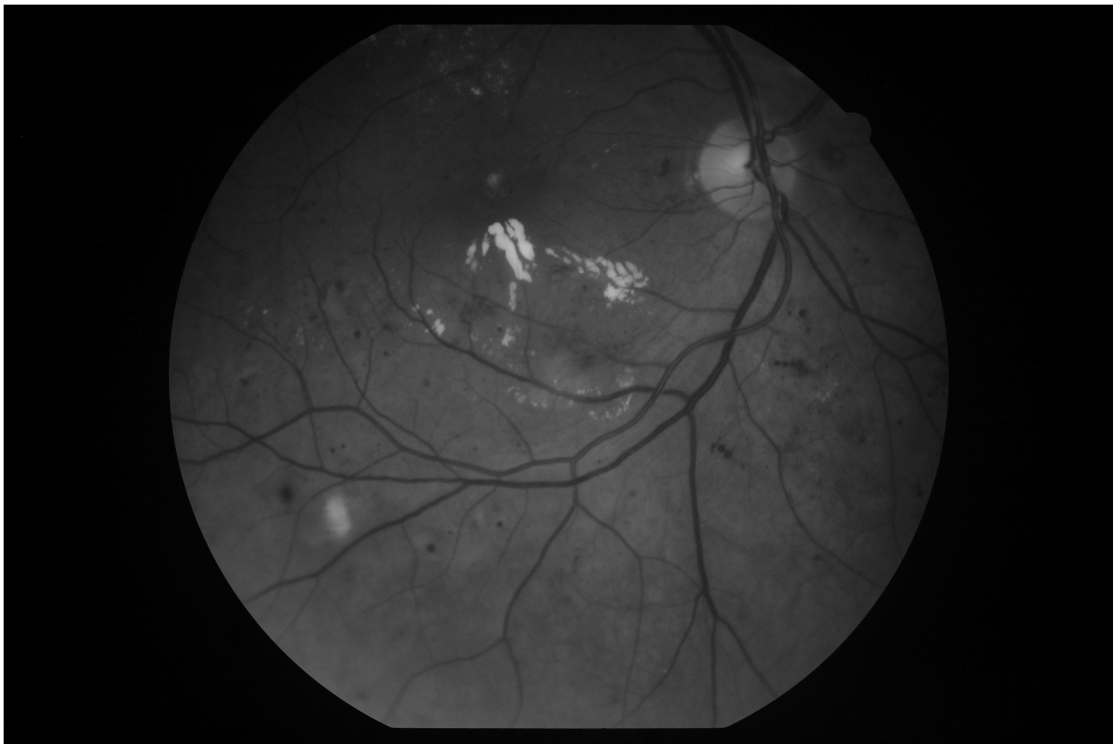


**Fig-5:Red free image of Fig.4**

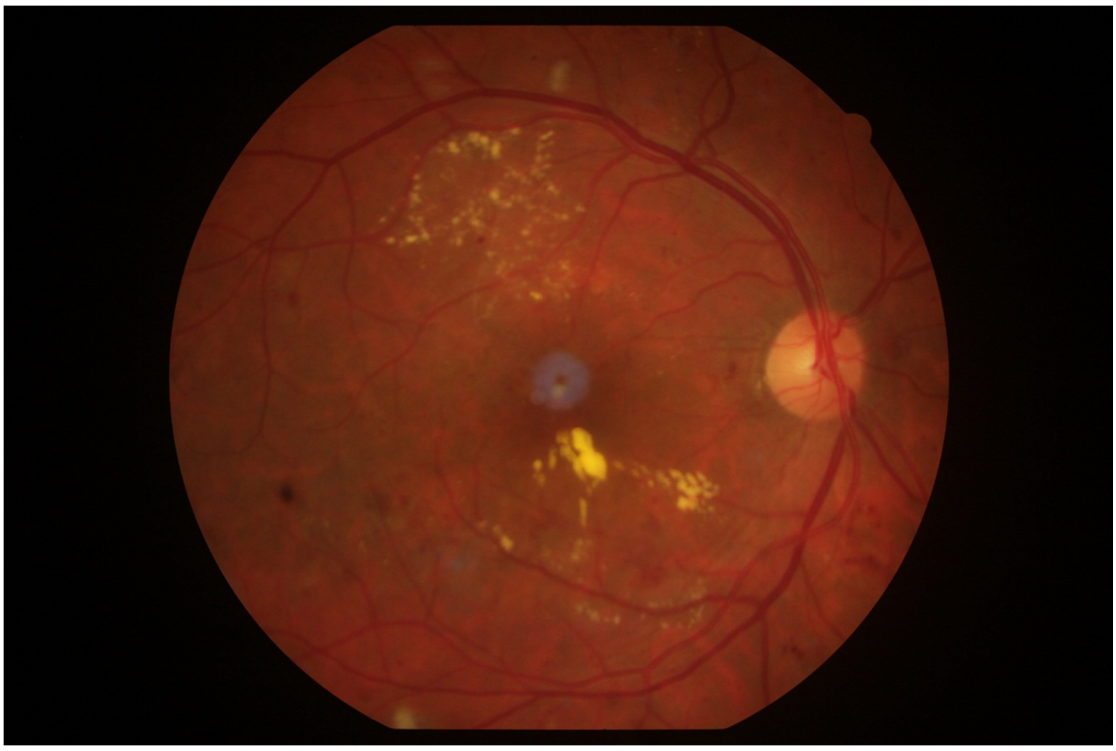


**Fig-6: Moderate NPDR with CSME right eye**

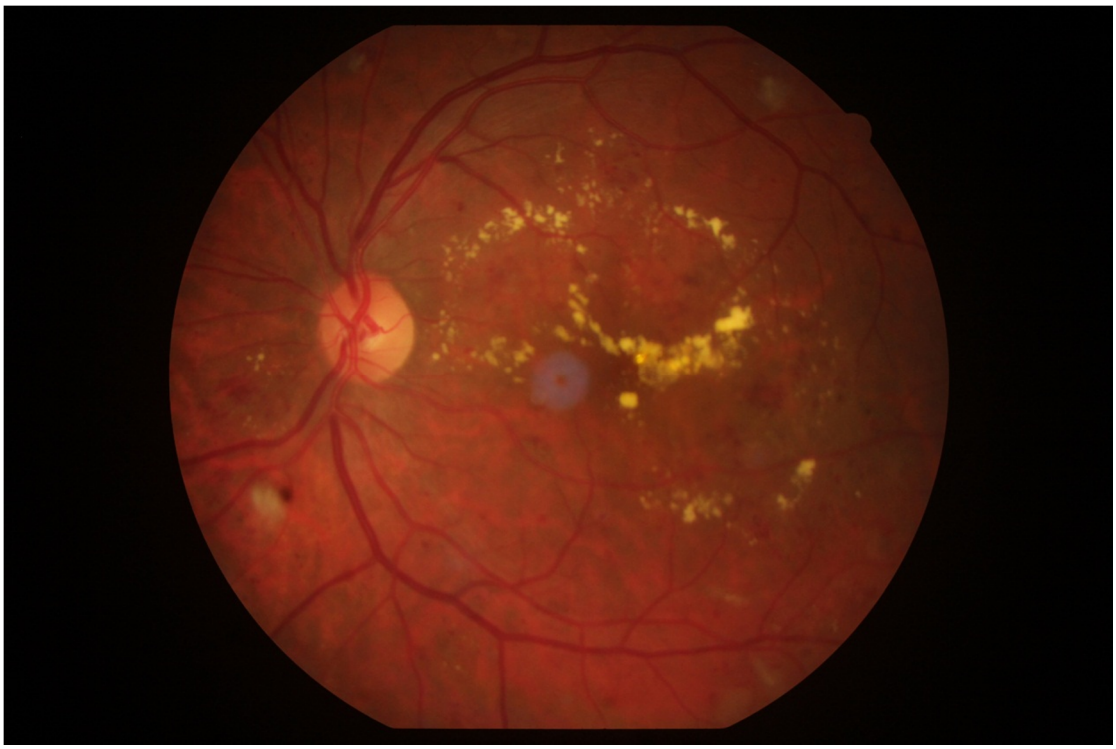


**Fig-7:Moderate NPDR with CSME left eye**

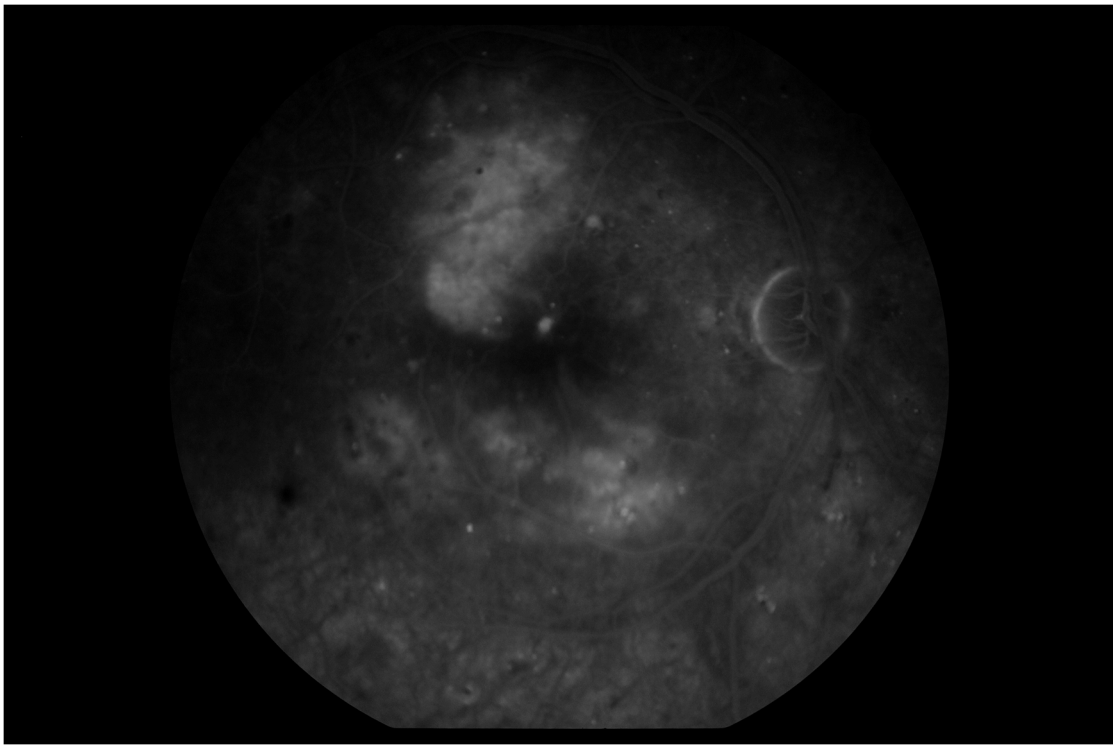


**Fig-8 : Fundus fluorescein angiography of fig.6**

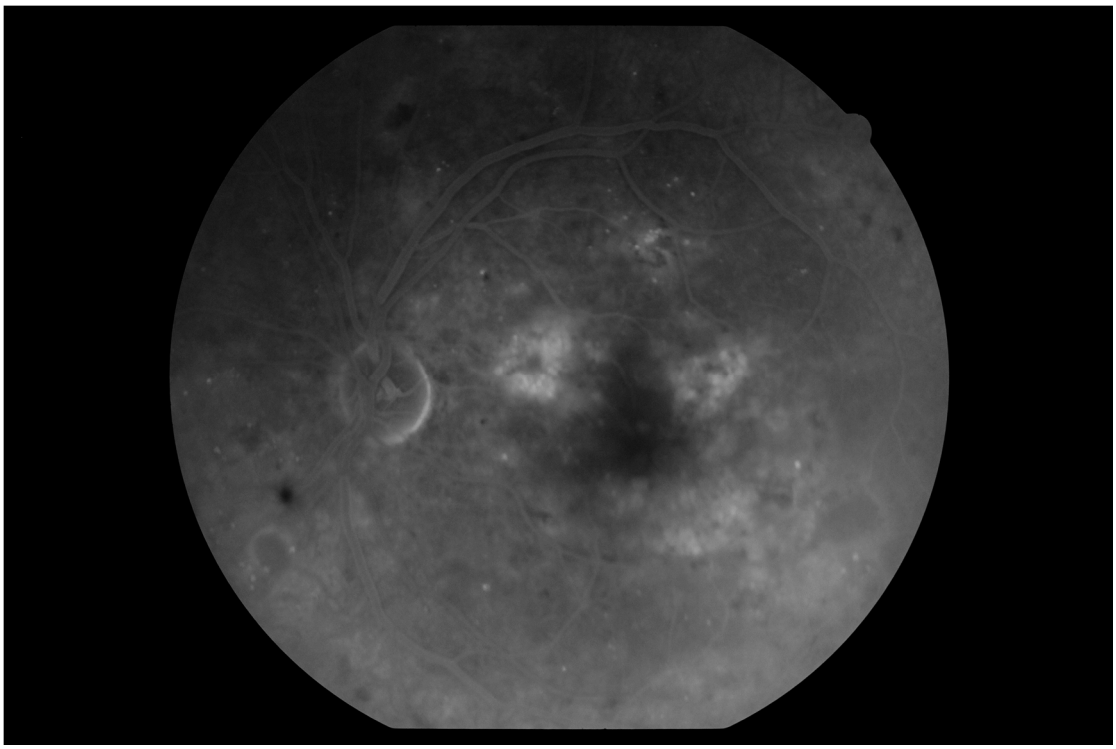


**Fig-9 : Fundus fluorescein angiography of fig.7**

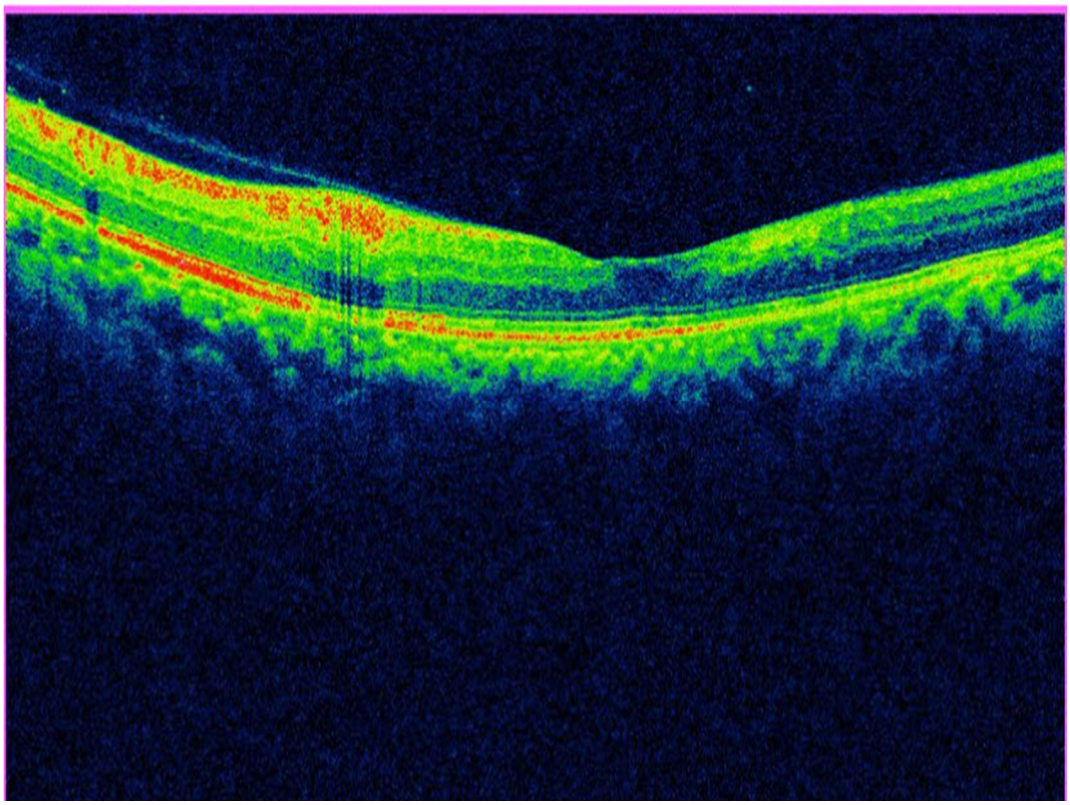


**Fig 10 : OCT showing macular edema**

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# PROFORMA

Name: Date

Age /sex: OP No:

History:

Duration of diabetes:

Insulin usage:Y/N

Duration of insulin usage:

History of hypertension:Y/N

Duration of hypertension:

Any other relevant Past history:

Clinical Examinaton:

RE

LE

1.Vision

2.Intraocular tension

3.Colour Vision

4.Fields

5.Slit Lamp examination with +90D lens:

i)Media-

ii)Disc-

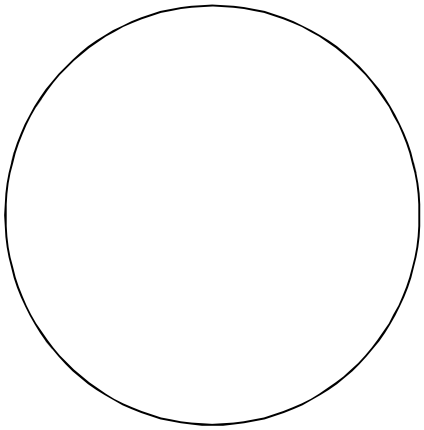
iii)Vessels-

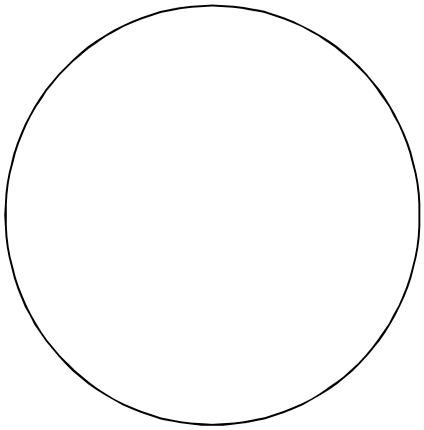
iv)Macula-

6.Fundus examination with indirect

Ophthalmoscope:

RE LE





7.Fundus photograph:

8.Fundus fluorescein angiography

## CONSENT FORM

Hereby I volunteer and consent to participate in this study "**AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY**". I was fully explained about the nature of this study by the doctor; knowing which I Mr/Ms/Mrs.……. Fully consent to volunteer in this study.

Date: Signature of the volunteer

Place:

Signature of the witness

## CONSENT FORM

I, **Dr. PRAYAGI KANDOTH** am carrying out a study on the topic, "**AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY**".

My research project guide is **Dr.V.Thaialnayaki, M.S.,**

My research project is being carried out in the Department of Ophthalmology, Coimbatore Medical College Hospital, Coimbatore.

**RESEARCH BEING DONE:**

AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY

**PURPOSE OF RESEARCH:**

To find out the association between serum lipid levels and development of clinically significant macular edema in diabetic retinopathy patients.

**PROCEDURE INVOLVED:**

The presence or absence of clinically significant macular o edema was found out by slit lamp bio microscopy with +90D lens and by indirect ophthalmoscopy. The levels of serum lipids were estimated by taking fasting blood samples. Blood urea, serum creatinine, systolic and diastolic blood pressures were also recorded.

You, Shri./Smt./Kum. _____, aged_____ years S/o/ W/o/D/o_____, residing at_____ _____ are requested to be a participant in the research study titled "**AN ANALYTICAL STUDY CORRELATING THE SIGNIFICANCE OF SERUM LIPIDS IN THE DEVELOPMENT OF CLINICALLY SIGNIFICANT MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY**" in Government Coimbatore Medical College, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any questions or seek any clarifications on the study that you may have before agreeing to participate.

**DECLINE FROM PARTICIPATION**

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

**PRIVACY AND CONFIDENTIALITY**

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

**AUTHORIZATION TO PUBLISH RESULTS**

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

**STATEMENT OF CONSENT**

I, _____, do hereby volunteer and consent to participate in this study being conducted by **Dr. PRAYAGI KANDOTH**. I have read and understood the consent from/or it has been read and explained to me in my own language. The study has been fully explained to me, and whenever I ask questions at any time.

Date: Signature/Left Thumb Impression of the Volunteer

Date: Signature and Name of witness

# xggj;go tk;

bgah;:

taJ : ghypdk;:

Kfthp:

muR nfhit kUj;Jtf;fy;Yhpapy; fz;kUj;Jtj;Jiwapy; gl;lnkwgogg[ gapYk; khz tp **kU.gu;ahfp fz;nlhj;** mth;fs; nkwbfhs;Sk; "**clkgpy; css;s bfhGggg[;rj;Jf;Fk/ rh;f;fiu nehapdhy; Vwgl;l fz;ghjpggpw;Fk/ rkgej;jk; css;sjh**" vdW kUj;jth;fs; brayak; fz;ghpnrhjidf;F ehd; KGkdJld; rkkjpf;fpnwd; Ma;tpd; bra;Kiw bjhlhghd midj;J tpgu';fisak; nfl;L vdJ ren;njf';fisj; bjstpgLj;jpf; bfhz;nld;

ehd;,e;j Ma;tpy;vd;id ghpnrhjid braya KGkdJlDk/ Rarpe;jida[lDk;rkkjpf;fpnwd;

vdJneha;gwwpa ,e;j Ma;tpy;v';fsJ midj;J tpgu';fSk; ghJfhf;fggLtJld;neha;gFjpapd;g[ifggLk;kwWk;,jd;Kot[fs; Ma;tpjHpy; btspapLggLtjpy; Mlnrgid ,y;iy vdgijj; bjhptpj;Jf;bfhs;fpnwd; ve;j neuj;jpYk;,e;j Ma]tpypUe]J ehd] tpyfpf]bfhs]s vdf]F chpik cz]L vdgijaik] mwpntd]

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# KEY TO MASTER CHART

**1-** Male

2- Female

DR- Diabetic Retinopathy

Stages of diabetic retinopathy-0-No diabetic retinopathy

1-Very Mild NPDR

2-Mild NPDR

3-Moderate NPDR

4-Severe NPDR

5-Very Severe NPDR

6-PDR

CSME- Clinically Significant Macular Edema

RE- Right Eye

LE- Left Eye

Insulin usage-1-Yes

2-No

SBP- Systolic Blood Pressure

DBP- Diastolic Blood Pressure

DM- Diabetes Mellitus

HTN- Hypertension

HDL-High Density Lipoprotein

LDL-Low Density Lipoprotein

Y-Yes

N-No

# MASTER CHART

| S. No | Name | Age (yrs) | Sex | Total cholesterol | Triglyceride | HDL | LDL | DR Stage RE | DR stage LE | CSME RE | CSME LE | SBP | DBP | Blood urea | S Creatinine | Insulin usage | Duration of DM | Duration of Insulin usage | Duration of HTN |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Kalimuthu | 54 | 1 | 256 | 234 | 20 | 120 | 2 | 3 | N | Y | 130 | 80 | 30 | 1.2 | 1 | 10 | 8 | 8 |
| 2 | Muthu | 64 | 1 | 265 | 253 | 26 | 281 | 4 | 2 | Y | N | 150 | 90 | 26 | 0.7 | 1 | 15 | 10 | 10 |
| 3 | seethayammal | 44 | 2 | 280 | 266 | 14 | 166 | 3 | 1 | Y | N | 120 | 70 | 32 | 0.9 | 1 | 8 | 8 | 4 |
| 4 | Veerammal | 53 | 2 | 270 | 253 | 40 | 84 | 3 | 0 | Y | N | 130 | 90 | 29 | 0.8 | 1 | 15 | 12 | 17 |
| 5 | Pitchai | 54 | 1 | 289 | 263 | 23 | 160 | 0 | 3 | N | Y | 160 | 100 | 38 | 2 | 1 | 10 | 8 | 8 |
| 6 | Murugan | 60 | 1 | 288 | 266 | 34 | 164 | 6 | 3 | Y | N | 110 | 70 | 27 | 0.8 | 1 | 15 | 7 | 7 |
| 7 | Patiyammal | 68 | 2 | 303 | 277 | 12 | 191 | 3 | 4 | N | Y | 140 | 90 | 29 | 1 | 1 | 13 | 5 | 9 |
| 8 | Bakiyam | 60 | 2 | 290 | 267 | 30 | 269 | 3 | 3 | Y | Y | 148 | 84 | 23 | 0.8 | 2 | 11 | 0 | 8 |
| 9 | subathal | 47 | 2 | 300 | 278 | 35 | 180 | 2 | 4 | N | Y | 130 | 90 | 25 | 0.7 | 2 | 12 | 0 | 6 |
| 10 | Ramasamy | 48 | 1 | 302 | 289 | 22 | 166 | 3 | 5 | Y | Y | 150 | 92 | 26 | 1 | 1 | 15 | 12 | 7 |
| 11 | Damodaran | 51 | 1 | 277 | 256 | 31 | 130 | 3 | 2 | Y | N | 164 | 94 | 29 | 1.1 | 2 | 10 | 0 | 12 |
| 12 | Sundari | 65 | 2 | 403 | 378 | 35 | 184 | 6 | 6 | Y | Y | 190 | 110 | 34 | 1.9 | 2 | 10 | 0 | 0 |
| 13 | Gurusami | 75 | 1 | 279 | 254 | 26 | 166 | 6 | 4 | Y | Y | 120 | 80 | 22 | 0.7 | 2 | 0 | 0 | 8 |
| 14 | Madheena | 53 | 2 | 290 | 245 | 41 | 140 | 3 | 1 | Y | N | 160 | 90 | 28 | 0.9 | 1 | 12 | 5 | 8 |
| 15 | Thangammal | 87 | 2 | 311 | 278 | 28 | 174 | 5 | 2 | Y | Y | 164 | 90 | 30 | 1.7 | 1 | 14 | 5 | 10 |
| 16 | Perumal | 60 | 1 | 356 | 289 | 30 | 221 | 6 | 4 | Y | Y | 180 | 110 | 32 | 1.9 | 2 | 10 | 0 | 5 |
| 17 | Thangammal | 73 | 2 | 374 | 312 | 35 | 194 | 6 | 6 | Y | Y | 150 | 92 | 27 | 1 | 1 | 11 | 11 | 1 |
| 18 | Chellamal | 56 | 2 | 288 | 267 | 30 | 171 | 3 | 1 | Y | N | 120 | 80 | 23 | 0.7 | 2 | 0 | 0 | 0 |
| 19 | Arumugam | 49 | 1 | 356 | 300 | 23 | 214 | 5 | 4 | Y | Y | 160 | 90 | 27 | 1.1 | 1 | 15 | 13 | 15 |
| 20 | Arokiyasamy | 60 | 1 | 287 | 254 | 20 | 199 | 3 | 2 | N | Y | 170 | 100 | 28 | 1.3 | 1 | 12 | 7 | 9 |
| 21 | Nagendran | 48 | 1 | 315 | 276 | 38 | 240 | 4 | 3 | N | Y | 150 | 90 | 25 | 1.2 | 1 | 10 | 2 | 6 |
| 22 | chinnasamy | 65 | 1 | 325 | 269 | 54 | 196 | 4 | 3 | Y | N | 148 | 90 | 28 | 1 | 2 | 6 | 0 | 6 |
| 23 | Masilamani | 58 | 2 | 315 | 296 | 36 | 248 | 4 | 3 | Y | Y | 150 | 88 | 27 | 1.1 | 2 | 13 | 0 | 10 |
| 24 | Mani | 61 | 1 | 289 | 245 | 35 | 154 | 3 | 2 | Y | N | 148 | 78 | 24 | 0.7 | 2 | 18 | 0 | 15 |
| 25 | Palaniyammal | 57 | 2 | 278 | 250 | 42 | 201 | 3 | 2 | Y | N | 168 | 88 | 23 | 0.7 | 2 | 0 | 0 | 3 |
| 26 | Sundaram | 68 | 1 | 290 | 234 | 26 | 140 | 3 | 0 | Y | N | 140 | 90 | 24 | 1 | 1 | 12 | 2 | 7 |
| 27 | Ravisankar | 65 | 1 | 303 | 276 | 19 | 402 | 6 | 2 | Y | N | 110 | 72 | 22 | 0.7 | 1 | 13 | 1 | 2 |
| 28 | Kuppamal | 52 | 2 | 346 | 243 | 19 | 122 | 6 | 2 | Y | N | 180 | 100 | 21 | 0.9 | 2 | 15 | 0 | 17 |
| 29 | Kaliyammal | 71 | 2 | 359 | 300 | 22 | 192 | 6 | 5 | Y | Y | 120 | 84 | 34 | 1.6 | 2 | 14 | 0 | 2 |
| 30 | Karuppayammal | 60 | 2 | 423 | 256 | 35 | 110 | 5 | 1 | Y | N | 170 | 110 | 26 | 0.7 | 1 | 16 | 13 | 6 |
| 31 | Ahmed | 57 | 1 | 345 | 267 | 39 | 140 | 4 | 3 | Y | Y | 166 | 98 | 29 | 0.8 | 1 | 10 | 3 | 8 |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 32 | Manikandan | 54 | 1 | 325 | 300 | 22 | 220 | 3 | 3 | Y | Y | 150 | 96 | 26 | 1 | 1 | 14 | 13 | 10 |
| 33 | Vellamal | 80 | 2 | 430 | 320 | 35 | 90 | 6 | 4 | Y | N | 170 | 100 | 30 | 1.1 | 2 | 14 | 0 | 10 |
| 34 | Pandidurai | 70 | 1 | 378 | 265 | 25 | 308 | 4 | 3 | Y | Y | 148 | 96 | 25 | 0.9 | 2 | 10 | 0 | 5 |
| 35 | Durairaj | 63 | 1 | 311 | 268 | 24 | 219 | 3 | 3 | Y | N | 130 | 80 | 27 | 0.7 | 2 | 0 | 0 | 0 |
| 36 | Deivathal | 62 | 2 | 287 | 235 | 28 | 193 | 2 | 3 | N | Y | 140 | 88 | 24 | 1 | 2 | 10 | 0 | 8 |
| 37 | Subbaiya | 55 | 1 | 411 | 345 | 36 | 130 | 6 | 6 | Y | Y | 166 | 96 | 27 | 1.1 | 1 | 16 | 14 | 12 |
| 38 | Subbalakhmi | 44 | 2 | 356 | 312 | 40 | 161 | 5 | 5 | Y | Y | 170 | 100 | 30 | 1.8 | 1 | 18 | 12 | 16 |
| 39 | Theresa | 66 | 2 | 356 | 298 | 24 | 143 | 4 | 2 | Y | N | 160 | 100 | 28 | 0.9 | 2 | 16 | 0 | 10 |
| 40 | Ponnammal | 60 | 2 | 243 | 199 | 30 | 196 | 3 | 1 | N | Y | 164 | 90 | 29 | 1 | 1 | 12 | 4 | 15 |
| 41 | Kanagambal | 40 | 2 | 296 | 248 | 27 | 148 | 2 | 3 | Y | N | 168 | 90 | 28 | 0.7 | 2 | 13 | 0 | 8 |
| 42 | Manivannan | 60 | 1 | 340 | 312 | 31 | 300 | 5 | 4 | Y | N | 140 | 86 | 27 | 0.8 | 2 | 10 | 0 | 6 |
| 43 | Jankai | 65 | 2 | 356 | 325 | 30 | 212 | 5 | 6 | N | Y | 158 | 100 | 29 | 0.9 | 1 | 18 | 15 | 12 |
| 44 | Chellappa | 50 | 1 | 290 | 210 | 28 | 196 | 1 | 3 | N | Y | 158 | 88 | 29 | 1.1 | 1 | 10 | 3 | 9 |
| 45 | Palanisamy | 54 | 1 | 314 | 287 | 44 | 88 | 3 | 3 | N | Y | 150 | 90 | 25 | 0.7 | 2 | 14 | 0 | 8 |
| 46 | Karuppusamy | 48 | 1 | 378 | 324 | 23 | 200 | 4 | 3 | Y | N | 130 | 92 | 25 | 0.7 | 2 | 18 | 0 | 6 |
| 47 | Periyasamy | 64 | 1 | 346 | 333 | 30 | 150 | 6 | 6 | Y | N | 150 | 86 | 28 | 0.8 | 1 | 20 | 3 | 5 |
| 48 | Nesamalar | 72 | 2 | 330 | 287 | 22 | 184 | 3 | 2 | N | Y | 148 | 84 | 26 | 0.7 | 2 | 16 | 0 | 15 |
| 49 | kaliyammal | 74 | 2 | 298 | 247 | 17 | 211 | 3 | 0 | Y | N | 130 | 90 | 28 | 1 | 2 | 0 | 0 | 0 |
| 50 | Veeramani | 60 | 1 | 379 | 335 | 21 | 194 | 6 | 5 | Y | Y | 120 | 70 | 26 | 0.9 | 2 | 0 | 0 | 8 |
| 51 | Ragasamy | 65 | 1 | 390 | 337 | 30 | 195 | 5 | 3 | N | Y | 130 | 90 | 26 | 0.8 | 1 | 12 | 3 | 2 |
| 52 | Sreenivasan | 58 | 1 | 288 | 234 | 32 | 187 | 4 | 3 | N | Y | 156 | 96 | 24 | 1 | 2 | 10 | 0 | 8 |
| 53 | Aiyaavoo | 61 | 1 | 309 | 278 | 28 | 115 | 3 | 3 | Y | Y | 160 | 100 | 29 | 1.8 | 1 | 15 | 7 | 0 |
| 54 | Lakshmi | 57 | 2 | 367 | 327 | 40 | 167 | 6 | 4 | Y | N | 156 | 96 | 25 | 1.2 | 2 | 17 | 0 | 5 |
| 55 | Moideen | 68 | 1 | 276 | 235 | 31 | 160 | 4 | 3 | N | Y | 148 | 90 | 28 | 1.3 | 2 | 12 | 0 | 6 |
| 56 | Shanmughasundaram | 65 | 1 | 343 | 312 | 35 | 223 | 3 | 3 | Y | Y | 120 | 90 | 32 | 1.4 | 2 | 10 | 0 | 10 |
| 57 | Veerapandi | 52 | 1 | 365 | 318 | 36 | 235 | 2 | 3 | N | Y | 134 | 80 | 28 | 0.7 | 2 | 12 | 0 | 5 |
| 58 | Devammal | 65 | 2 | 427 | 356 | 60 | 311 | 6 | 6 | Y | Y | 120 | 90 | 23 | 0.7 | 2 | 18 | 0 | 8 |
| 59 | Thirumathal | 58 | 2 | 387 | 335 | 56 | 324 | 5 | 5 | N | Y | 130 | 90 | 26 | 0.8 | 2 | 10 | 0 | 9 |
| 60 | Radhamani | 61 | 2 | 267 | 212 | 53 | 156 | 4 | 3 | N | Y | 134 | 94 | 23 | 0.7 | 1 | 13 | 0 | 8 |
| 61 | Badrammal | 57 | 2 | 317 | 267 | 29 | 212 | 3 | 3 | N | Y | 146 | 90 | 22 | 1.1 | 2 | 15 | 0 | 4 |
| 62 | Kanganbal | 68 | 2 | 341 | 300 | 42 | 254 | 2 | 3 | N | Y | 140 | 90 | 22 | 1 | 2 | 10 | 0 | 12 |
| 63 | Badrasami | 65 | 1 | 305 | 265 | 40 | 234 | 5 | 4 | Y | Y | 168 | 70 | 34 | 1.7 | 1 | 17 | 4 | 12 |
| 64 | Kaliyappan | 52 | 1 | 411 | 356 | 36 | 318 | 5 | 6 | Y | Y | 140 | 92 | 24 | 0.9 | 1 | 10 | 8 | 10 |
| 65 | Chinnappasamy | 60 | 1 | 267 | 200 | 25 | 217 | 1 | 3 | N | Y | 148 | 92 | 28 | 1.1 | 2 | 0 | 0 | 7 |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 66 | Jameela | 68 | 2 | 284 | 217 | 86 | 225 | 3 | 3 | Y | N | 130 | 90 | 23 | 0.9 | 1 | 10 | 1 | 10 |
| 67 | Kathirvel | 60 | 1 | 267 | 166 | 53 | 200 | 4 | 3 | Y | N | 146 | 80 | 24 | 0.7 | 2 | 14 | 0 | 1 |
| 68 | Mayilsamy | 47 | 1 | 348 | 243 | 111 | 287 | 6 | 6 | Y | N | 160 | 92 | 30 | 1.2 | 2 | 15 | 0 | 6 |
| 69 | Arokyasamy | 48 | 1 | 376 | 306 | 32 | 243 | 3 | 3 | Y | Y | 154 | 94 | 32 | 1.1 | 1 | 12 | 0 | 8 |
| 70 | Kannamal | 51 | 2 | 351 | 267 | 35 | 235 | 3 | 3 | Y | Y | 156 | 92 | 26 | 0.7 | 2 | 10 | 10 | 8 |
| 71 | Ponnappan | 65 | 1 | 349 | 278 | 25 | 342 | 4 | 3 | Y | N | 150 | 90 | 25 | 0.7 | 2 | 14 | 0 | 5 |
| 72 | Chidambaram | 75 | 1 | 448 | 380 | 31 | 338 | 3 | 3 | Y | Y | 136 | 82 | 26 | 1.2 | 2 | 11 | 0 | 1 |
| 73 | Vellamal | 48 | 2 | 329 | 254 | 36 | 245 | 4 | 3 | Y | Y | 146 | 90 | 23 | 0.8 | 1 | 14 | 7 | 8 |
| 74 | Kuppusamy | 65 | 1 | 297 | 219 | 33 | 244 | 4 | 3 | Y | N | 164 | 100 | 24 | 0.8 | 1 | 12 | 5 | 12 |
| 75 | Jayaraj | 58 | 1 | 414 | 214 | 26 | 300 | 6 | 6 | N | Y | 150 | 80 | 23 | 0.8 | 1 | 19 | 15 | 12 |
| 76 | Radha | 61 | 2 | 140 | 125 | 12 | 122 | 3 | 1 | Y | N | 134 | 86 | 22 | 0.9 | 1 | 9 | 2 | 10 |
| 77 | Kamaraj | 57 | 1 | 160 | 145 | 35 | 90 | 5 | 4 | Y | N | 146 | 90 | 22 | 0.7 | 1 | 9 | 4 | 2 |
| 78 | Sivagami | 68 | 2 | 418 | 183 | 26 | 287 | 3 | 5 | N | Y | 118 | 72 | 21 | 0.7 | 2 | 5 | 0 | 0 |
| 79 | Eswari | 65 | 2 | 288 | 193 | 36 | 196 | 4 | 3 | N | Y | 170 | 100 | 34 | 2.2 | 2 | 0 | 0 | 5 |
| 80 | Mani | 52 | 1 | 312 | 165 | 42 | 200 | 4 | 3 | N | Y | 150 | 86 | 26 | 1 | 1 | 7 | 1 | 6 |
| 81 | Ramathal | 71 | 2 | 289 | 175 | 29 | 214 | 4 | 3 | Y | Y | 156 | 88 | 29 | 0.7 | 1 | 8 | 1 | 10 |
| 82 | Kamaraj | 60 | 1 | 251 | 180 | 113 | 30 | 3 | 2 | Y | N | 164 | 110 | 28 | 0.9 | 2 | 7 | 0 | 8 |
| 83 | Senthil | 57 | 1 | 310 | 182 | 24 | 246 | 3 | 4 | N | Y | 164 | 90 | 29 | 0.9 | 2 | 0 | 0 | 5 |
| 84 | Chinnammal | 50 | 2 | 287 | 166 | 21 | 221 | 3 | 0 | Y | N | 140 | 80 | 27 | 0.9 | 2 | 5 | 0 | 9 |
| 85 | Gunasekaran | 54 | 1 | 385 | 264 | 36 | 268 | 5 | 4 | Y | N | 150 | 96 | 32 | 1 | 2 | 9 | 0 | 7 |
| 86 | Narayanasamy | 48 | 1 | 229 | 189 | 26 | 164 | 5 | 6 | N | Y | 120 | 70 | 26 | 1.3 | 2 | 8 | 0 | 8 |
| 87 | Vellamal | 64 | 2 | 430 | 320 | 31 | 117 | 5 | 3 | Y | Y | 170 | 100 | 30 | 1.1 | 2 | 14 | 0 | 10 |
| 88 | Pandidurai | 72 | 1 | 378 | 265 | 25 | 168 | 3 | 3 | Y | Y | 148 | 96 | 25 | 0.9 | 2 | 10 | 0 | 5 |
| 89 | Durairaj | 74 | 1 | 311 | 268 | 22 | 153 | 4 | 3 | Y | N | 130 | 80 | 27 | 0.7 | 2 | 13 | 0 | 6 |
| 90 | Deivathal | 60 | 2 | 287 | 235 | 19 | 121 | 3 | 3 | N | Y | 140 | 88 | 24 | 1 | 2 | 10 | 0 | 8 |
| 91 | Kalimuthu | 65 | 1 | 347 | 168 | 35 | 224 | 3 | 3 | Y | Y | 130 | 92 | 25 | 0.7 | 2 | 0 | 0 | 6 |
| 92 | Chandrasekhar | 58 | 1 | 233 | 168 | 22 | 179 | 2 | 3 | N | Y | 150 | 86 | 28 | 0.8 | 1 | 20 | 18 | 5 |
| 93 | Muniyandi | 61 | 1 | 288 | 195 | 143 | 26 | 0 | 4 | N | Y | 148 | 84 | 26 | 0.7 | 2 | 16 | 0 | 15 |
| 94 | Venugopal | 57 | 2 | 248 | 167 | 22 | 166 | 6 | 4 | Y | Y | 130 | 90 | 28 | 1 | 2 | 12 | 0 | 1 |
| 95 | Pechiyammal | 68 | 1 | 193 | 136 | 35 | 120 | 3 | 4 | Y | N | 120 | 70 | 26 | 0.9 | 2 | 10 | 0 | 8 |
| 96 | Kandhasamy | 65 | 2 | 329 | 263 | 196 | 110 | 3 | 3 | Y | N | 130 | 90 | 26 | 0.8 | 1 | 12 | 9 | 2 |
| 97 | Dhanabakyiam | 56 | 2 | 190 | 130 | 30 | 130 | 3 | 2 | Y | N | 156 | 96 | 24 | 1 | 2 | 0 | 0 | 8 |
| 98 | Marathal | 62 | 1 | 142 | 123 | 12 | 122 | 3 | 5 | Y | N | 160 | 100 | 29 | 1.8 | 1 | 15 | 13 | 0 |
| 99 | Marappan | 54 | 1 | 518 | 318 | 34 | 402 | 6 | 3 | N | Y | 156 | 96 | 25 | 1.2 | 1 | 12 | 12 | 2 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 100 | Senthinathan | 67 | 1 | 307 | 267 | 25 | 243 | 3 | 4 | Y | Y | 164 | 100 | 28 | 1 | 1 | 6 | 5 | 4 |
| 101 | Vetrivel | 57 | 1 | 112 | 104 | 35 | 60 | 2 | 1 | N | N | 130 | 86 | 26 | 0.8 | 1 | 8 | 5 | 7 |
| 102 | Karupayee | 75 | 2 | 156 | 124 | 39 | 108 | 3 | 2 | N | N | 124 | 80 | 18 | 0.6 | 1 | 7 | 5 | 6 |
| 103 | Chinnavel | 55 | 1 | 148 | 118 | 45 | 82 | 0 | 2 | N | N | 130 | 90 | 24 | 0.9 | 2 | 5 | 0 | 8 |
| 104 | Ganeshan | 65 | 1 | 129 | 84 | 48 | 68 | 1 | 0 | N | N | 120 | 80 | 36 | 0.9 | 2 | 9 | 0 | 10 |
| 105 | Saraswathi | 54 | 2 | 143 | 93 | 45 | 74 | 2 | 2 | N | N | 124 | 90 | 42 | 1.4 | 2 | 0 | 0 | 2 |
| 106 | Chellamal | 70 | 2 | 246 | 170 | 40 | 112 | 4 | 3 | N | N | 130 | 74 | 54 | 2.4 | 1 | 6 | 6 | 12 |
| 107 | Krishnan | 60 | 1 | 400 | 212 | 43 | 156 | 3 | 2 | N | N | 140 | 100 | 70 | 4.3 | 1 | 8 | 4 | 10 |
| 108 | Nallathambi | 62 | 1 | 312 | 194 | 42 | 190 | 3 | 2 | N | N | 170 | 106 | 63 | 2.6 | 1 | 6 | 4 | 7 |
| 109 | Kaliyammal | 58 | 2 | 134 | 78 | 47 | 84 | 4 | 2 | N | N | 140 | 90 | 29 | 1.1 | 1 | 8 | 8 | 8 |
| 110 | Masilamai | 67 | 2 | 116 | 101 | 45 | 73 | 0 | 1 | N | N | 120 | 80 | 32 | 1 | 2 | 8 | 0 | 58 |
| 111 | Fathima | 45 | 2 | 188 | 112 | 41 | 121 | 4 | 2 | N | N | 140 | 94 | 48 | 1.2 | 1 | 5 | 1 | 5 |
| 112 | Thirumalasamy | 52 | 1 | 163 | 83 | 40 | 108 | 2 | 2 | N | N | 134 | 80 | 33 | 0.8 | 1 | 7 | 2 | 11 |
| 113 | Venkatachalam | 61 | 1 | 148 | 99 | 46 | 183 | 1 | 0 | N | N | 130 | 80 | 24 | 0.8 | 1 | 9 | 4 | 5 |
| 114 | Kuppusamy | 68 | 1 | 180 | 104 | 45 | 124 | 2 | 3 | N | N | 126 | 76 | 19 | 0.9 | 2 | 4 | 0 | 4 |
| 115 | Sellamal | 42 | 2 | 174 | 143 | 41 | 93 | 2 | 2 | N | N | 120 | 80 | 29 | 0.8 | 1 | 7 | 3 | 6 |
| 116 | Kalavathi | 58 | 2 | 116 | 97 | 40 | 54 | 2 | 0 | N | N | 130 | 90 | 36 | 0.9 | 2 | 0 | 0 | 0 |
| 117 | Shanthi | 47 | 2 | 412 | 338 | 42 | 123 | 3 | 2 | N | N | 170 | 100 | 63 | 4.3 | 2 | 8 | 0 | 8 |
| 118 | Micheal John | 61 | 1 | 158 | 153 | 44 | 74 | 4 | 3 | N | N | 140 | 90 | 21 | 0.8 | 2 | 6 | 0 | 6 |
| 119 | Ramasamy | 72 | 1 | 143 | 133 | 50 | 70 | 2 | 2 | N | N | 130 | 80 | 24 | 0.8 | 2 | 8 | 0 | 6 |
| 120 | Muthammal | 58 | 2 | 151 | 126 | 53 | 81 | 3 | 2 | N | N | 134 | 84 | 32 | 0.7 | 1 | 7 | 2 | 7 |
| 121 | Jeyakodi | 50 | 2 | 284 | 214 | 51 | 126 | 0 | 2 | N | N | 140 | 100 | 43 | 2.1 | 2 | 5 | 0 | 5 |
| 122 | Vasantha | 70 | 2 | 177 | 168 | 56 | 110 | 3 | 3 | N | N | 120 | 80 | 36 | 0.9 | 1 | 7 | 3 | 7 |
| 123 | Sayeeda Beegum | 49 | 2 | 164 | 143 | 59 | 103 | 2 | 3 | N | N | 124 | 80 | 24 | 1.2 | 2 | 0 | 0 | 0 |
| 124 | Mahalingam | 56 | 1 | 183 | 162 | 44 | 116 | 0 | 2 | N | N | 130 | 86 | 25 | 1.1 | 2 | 4 | 0 | 1 |
| 125 | Nagarathinam | 59 | 2 | 310 | 211 | 45 | 110 | 3 | 2 | N | N | 160 | 100 | 51 | 4 | 1 | 6 | 6 | 5 |
| 126 | Kasi | 65 | 1 | 180 | 165 | 48 | 101 | 3 | 4 | N | N | 118 | 82 | 29 | 1.1 | 1 | 8 | 6 | 4 |
| 127 | Bagyalakshmi | 62 | 2 | 137 | 113 | 41 | 56 | 0 | 2 | N | N | 120 | 84 | 34 | 0.8 | 2 | 7 | 0 | 6 |
| 128 | Angammal | 53 | 2 | 145 | 126 | 48 | 78 | 2 | 0 | N | N | 130 | 86 | 19 | 0.9 | 2 | 8 | 0 | 8 |
| 129 | Adachiyammal | 56 | 2 | 192 | 197 | 40 | 111 | 2 | 3 | N | N | 120 | 80 | 21 | 1.1 | 1 | 7 | 5 | 7 |
| 130 | Devadas | 77 | 1 | 142 | 156 | 38 | 83 | 4 | 3 | N | N | 130 | 84 | 40 | 1.3 | 1 | 4 | 2 | 5 |
| 131 | Murugan | 70 | 1 | 124 | 117 | 40 | 66 | 2 | 2 | N | N | 124 | 80 | 33 | 0.9 | 1 | 7 | 1 | 4 |
| 132 | Dhanam | 51 | 2 | 185 | 173 | 35 | 104 | 3 | 2 | N | N | 134 | 86 | 24 | 0.8 | 2 | 0 | 0 | 5 |
| 133 | Rajammal | 56 | 2 | 144 | 132 | 40 | 71 | 0 | 2 | N | N | 110 | 70 | 28 | 1 | 2 | 7 | 0 | 8 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 134 | Velathai | 50 | 2 | 204 | 146 | 41 | 140 | 3 | 2 | N | N | 114 | 78 | 17 | 0.7 | 2 | 8 | 0 | 10 |
| 135 | Mary | 59 | 2 | 196 | 173 | 50 | 145 | 3 | 2 | N | N | 120 | 82 | 23 | 0.9 | 2 | 5 | 0 | 9 |
| 136 | Gomathi | 68 | 2 | 155 | 146 | 47 | 82 | 4 | 6 | N | N | 124 | 82 | 36 | 1.2 | 2 | 9 | 0 | 7 |
| 137 | Selvaraj | 63 | 1 | 183 | 167 | 45 | 114 | 0 | 2 | N | N | 130 | 80 | 40 | 1.4 | 2 | 8 | 0 | 8 |
| 138 | Chinnakannu | 56 | 1 | 147 | 138 | 48 | 190 | 4 | 2 | N | N | 130 | 86 | 26 | 0.9 | 2 | 11 | 0 | 1 |
| 139 | Papamal | 64 | 2 | 316 | 261 | 52 | 124 | 3 | 2 | N | N | 140 | 88 | 18 | 0.7 | 2 | 10 | 0 | 8 |
| 140 | Pandidurai | ~~48~~ | 1 | 182 | 165 | 54 | 102 | 2 | 0 | N | N | 134 | 82 | 32 | 1 | 2 | 5 | 0 | 10 |
| 141 | Radha | 55 | 2 | 156 | 148 | 57 | 84 | 3 | 0 | N | N | 124 | 84 | 19 | 1 | 1 | 9 | 5 | 8 |
| 142 | Pitchai | 53 | 1 | 214 | 193 | 51 | 86 | 0 | 2 | N | N | 130 | 80 | 27 | 1.1 | 2 | 0 | 0 | 3 |
| 143 | Singaram | 60 | 1 | 210 | 140 | 60 | 106 | 2 | 3 | N | N | 144 | 90 | 28 | 1 | 2 | 0 | 0 | 0 |
| 144 | Sethulakshmi | 64 | 22 | 190 | 170 | 67 | 102 | 2 | 0 | N | N | 146 | 86 | 23 | 0.7 | 1 | 6 | 4 | 4 |
| 145 | Chidambaram | 60 | 1 | 197 | 150 | 60 | 120 | 2 | 0 | N | N | 134 | 70 | 21 | 0.8 | 1 | 8 | 6 | 12 |
| 146 | Palaniyappan | 63 | 1 | 188 | 190 | 62 | 122 | 2 | 2 | N | N | 154 | 88 | 26 | 0.8 | 1 | 7 | 7 | 6 |
| 147 | Muniyandi | 59 | 1 | 176 | 200 | 56 | 194 | 4 | 3 | N | N | 130 | 92 | 25 | 1 | 1 | 5 | 2 | 5 |
| 148 | Subbaiyah | 53 | 1 | 210 | 205 | 53 | 60 | 3 | 2 | N | N | 144 | 84 | 27 | 0.9 | 1 | 7 | 1 | 7 |
| 149 | Noorjahan | 55 | 2 | 234 | 179 | 57 | 108 | 3 | 2 | N | N | 146 | 88 | 27 | 0.8 | 1 | 8 | 1 | 8 |
| 150 | Jamal | 48 | 1 | 196 | 248 | 47 | 82 | 4 | 6 | N | N | 144 | 84 | 26 | 0.7 | 2 | 6 | 0 | 6 |
| 151 | Valliyammal | 57 | 2 | 178 | 190 | 35 | 68 | 0 | 3 | N | N | 166 | 90 | 23 | 0.7 | 2 | 7 | 0 | 10 |
| 152 | Nagammal | 75 | 2 | 200 | 160 | 44 | 74 | 3 | 2 | N | N | 134 | 90 | 25 | 0.9 | 1 | 6 | 4 | 5 |
| 153 | Thomas | 55 | 1 | 203 | 158 | 64 | 201 | 2 | 2 | N | N | 146 | 88 | 24 | 1 | 1 | 6 | 5 | 6 |
| 154 | Motchamary | 65 | 2 | 236 | 163 | 55 | 240 | 2 | 0 | N | N | 140 | 90 | 22 | 0.7 | 2 | 8 | 0 | 3 |
| 155 | Perumal | 54 | 1 | 225 | 153 | 68 | 190 | 3 | 0 | N | N | 136 | 84 | 22 | 0.8 | 2 | 9 | 0 | 8 |
| 156 | Susheela | 70 | 2 | 218 | 210 | 47 | 84 | 0 | 2 | N | N | 168 | 98 | 24 | 0.7 | 2 | 6 | 0 | 10 |
| 157 | Dhandapani | 60 | 1 | 215 | 135 | 63 | 73 | 2 | 3 | N | N | 160 | 90 | 27 | 1 | 1 | 6 | 7 | 12 |
| 158 | Palanathal | 62 | 2 | 187 | 230 | 67 | 121 | 2 | 0 | N | N | 148 | 80 | 27 | 0.7 | 2 | 0 | 0 | 1 |
| 159 | Kathirvel | 58 | 1 | 212 | 167 | 42 | 108 | 0 | 2 | N | N | 170 | 100 | 32 | 2 | 2 | 7 | 0 | 5 |
| 160 | Ponnamal | 67 | 2 | 240 | 178 | 44 | 83 | 3 | 2 | N | N | 130 | 80 | 26 | 0.8 | 1 | 7 | 4 | 3 |
| 161 | Esari | 45 | 2 | 243 | 183 | 40 | 124 | 2 | 2 | N | N | 144 | 90 | 26 | 0.8 | 1 | 5 | 5 | 5 |
| 162 | Karuppan | 52 | 1 | 235 | 150 | 65 | 110 | 6 | 3 | N | N | 146 | 86 | 23 | 1.1 | 1 | 8 | 1 | 7 |
| 163 | Alima | 61 | 2 | 228 | 169 | 60 | 123 | 3 | 3 | N | N | 160 | 100 | 29 | 1 | 2 | 0 | 0 | 2 |
| 164 | Sivagnanam | 68 | 1 | 217 | 178 | 66 | 214 | 3 | 0 | N | N | 154 | 88 | 23 | 1 | 1 | 7 | 2 | 17 |
| 165 | Anbu | 42 | 2 | 191 | 185 | 54 | 132 | 3 | 4 | N | N | 136 | 80 | 23 | 0.7 | 1 | 8 | 3 | 7 |
| 166 | Palani | 58 | 1 | 189 | 164 | 46 | 122 | 1 | 0 | N | N | 146 | 92 | 26 | 0.8 | 1 | 4 | 1 | 2 |
| 167 | Aruchamy | 47 | 1 | 174 | 236 | 51 | 135 | 2 | 2 | N | N | 154 | 94 | 28 | 0.9 | 1 | 7 | 1 | 17 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 168 | Subban | 61 | 1 | 208 | 176 | 54 | 167 | 3 | 0 | N | N | 132 | 86 | 26 | 1.1 | 2 | 6 | 0 | 1 |
| 169 | Thangam | 72 | 2 | 217 | 156 | 59 | 123 | 0 | 2 | N | N | 110 | 74 | 29 | 1.7 | 1 | 8 | 4 | 5 |
| 170 | Raji | 58 | 2 | 240 | 180 | 64 | 112 | 0 | 2 | N | N | 120 | 80 | 23 | 0.9 | 2 | 0 | 0 | 3 |
| 171 | Muthu | 50 | 1 | 223 | 167 | 66 | 111 | 2 | 3 | N | N | 134 | 82 | 25 | 0.8 | 1 | 9 | 9 | 4 |
| 172 | Boominathan | 70 | 1 | 256 | 165 | 22 | 83 | 1 | 0 | N | N | 140 | 88 | 26 | 1 | 2 | 0 | 0 | 8 |
| 173 | Pushpa | 49 | 2 | 190 | 188 | 56 | 66 | 2 | 2 | N | N | 150 | 90 | 25 | 0.9 | 2 | 8 | 0 | 10 |
| 174 | Arumugham | 56 | 1 | 187 | 190 | 47 | 104 | 2 | 0 | N | N | 156 | 96 | 28 | 0.8 | 1 | 7 | 4 | 12 |
| 175 | Rangasamy | 59 | 1 | 213 | 201 | 43 | 71 | 3 | 0 | N | N | 110 | 70 | 27 | 1.8 | 2 | 4 | 0 | 0 |
| 176 | Panchavarnam | 65 | 2 | 209 | 165 | 47 | 140 | 2 | 3 | N | N | 158 | 98 | 24 | 2 | 2 | 6 | 0 | 5 |
| 177 | Selvakumar | 62 | 1 | 177 | 150 | 45 | 145 | 1 | 3 | N | N | 140 | 90 | 23 | 0.8 | 2 | 8 | 0 | 5 |
| 178 | Rajammal | 53 | 2 | 179 | 156 | 54 | 82 | 6 | 6 | N | N | 130 | 90 | 25 | 0.9 | 1 | 4 | 4 | 3 |
| 179 | Prema | 56 | 2 | 200 | 185 | 42 | 114 | 2 | 1 | N | N | 150 | 90 | 24 | 0.8 | 1 | 8 | 5 | 8 |
| 180 | Rajamani | 77 | 2 | 211 | 164 | 47 | 90 | 2 | 3 | N | N | 146 | 86 | 22 | 0.7 | 1 | 9 | 3 | 9 |
| 181 | Madhukumar | 70 | 1 | 210 | 140 | 45 | 169 | 0 | 1 | N | N | 130 | 80 | 27 | 1.1 | 2 | 0 | 0 | 7 |
| 182 | Marimuthu | 51 | 1 | 190 | 170 | 40 | 102 | 3 | 1 | N | N | 144 | 90 | 28 | 1 | 2 | 7 | 0 | 8 |
| 183 | Subramani | 56 | 1 | 197 | 150 | 53 | 84 | 3 | 4 | N | N | 146 | 86 | 23 | 0.7 | 2 | 9 | 0 | 8 |
| 184 | Ruckmani | 50 | 2 | 196 | 248 | 57 | 146 | 0 | 2 | N | N | 144 | 84 | 26 | 0.7 | 1 | 7 | 2 | 10 |
| 185 | Shanthi | 59 | 2 | 178 | 190 | 64 | 82 | 1 | 0 | N | N | 166 | 90 | 23 | 0.7 | 1 | 8 | 2 | 4 |
| 186 | Chandran | 68 | 1 | 200 | 160 | 66 | 93 | 2 | 3 | N | N | 134 | 90 | 25 | 0.9 | 1 | 5 | 1 | 1 |
| 187 | Mayangal | 63 | 2 | 203 | 158 | 61 | 54 | 3 | 4 | N | N | 146 | 88 | 24 | 1 | 2 | 7 | 0 | 5 |
| 188 | Philip | 56 | 1 | 236 | 163 | 54 | 119 | 2 | 3 | N | N | 140 | 90 | 22 | 0.7 | 1 | 8 | 6 | 6 |
| 189 | Kanappan | 64 | 1 | 225 | 153 | 43 | 74 | 3 | 2 | N | N | 136 | 84 | 22 | 0.8 | 1 | 8 | 8 | 6 |
| 190 | Ponraj | 48 | 1 | 218 | 210 | 46 | 70 | 1 | 3 | N | N | 168 | 98 | 24 | 0.7 | 2 | 0 | 0 | 8 |
| 191 | Murugesan | 55 | 1 | 215 | 135 | 43 | 81 | 2 | 0 | N | N | 160 | 90 | 27 | 1 | 1 | 9 | 5 | 7 |
| 192 | Kalavathi | 53 | 2 | 199 | 138 | 42 | 90 | 0 | 2 | N | N | 140 | 90 | 26 | 0.9 | 2 | 8 | 0 | 4 |
| 193 | Chinnamani | 60 | 2 | 177 | 205 | 46 | 110 | 3 | 2 | N | N | 144 | 84 | 24 | 0.7 | 1 | 4 | 1 | 7 |
| 194 | Anbmani | 64 | 2 | 187 | 230 | 49 | 103 | 0 | 2 | N | N | 148 | 80 | 27 | 0.7 | 2 | 7 | 0 | 8 |
| 195 | Balasubramaniyan | 60 | 1 | 212 | 167 | 50 | 116 | 2 | 3 | N | N | 170 | 100 | 32 | 2 | 2 | 0 | 0 | 5 |
| 196 | Rajamanikam | 63 | 1 | 240 | 178 | 62 | 112 | 2 | 0 | N | N | 130 | 80 | 26 | 0.8 | 2 | 8 | 0 | 6 |
| 197 | Palanisamy | 59 | 1 | 243 | 183 | 40 | 101 | 2 | 3 | N | N | 144 | 90 | 26 | 0.8 | 2 | 8 | 0 | 6 |
| 198 | Rangasamy | 53 | 1 | 235 | 150 | 42 | 56 | 3 | 2 | N | N | 146 | 86 | 23 | 1.1 | 2 | 5 | 0 | 7 |
| 199 | Muthammal | 55 | 2 | 228 | 169 | 44 | 78 | 2 | 2 | N | N | 160 | 100 | 29 | 1 | 2 | 4 | 0 | 10 |
| 200 | Rajalakshmi | 48 | 2 | 217 | 178 | 46 | 89 | 2 | 3 | N | N | 154 | 88 | 23 | 1 | 2 | 0 | 0 | 0 |