

Dyslipidemia-Induced Oxidative Stress and its Implications in Diabetic Retinopathy

Ekta Chitkara¹, Dr. Juhi Aggarwal², Dr. Urvashi Midha³

¹Professor, Department of Biochemistry, ITS Dental College, Greater Noida U.P.

²Professor and Head Department of Biochemistry, Santosh Medical College and Hospital
Santosh Deemed to be University Ghaziabad U.P.

³Phd scholar Department of Biochemistry, Santosh Medical College and Hospital, Santosh Deemed to be University Ghaziabad U.P.

HOW TO CITE:

Ekta Chitkara, Juhi Aggarwal, Urvashi Midha (2026).
Dyslipidemia-Induced Oxidative Stress and its Implications in Diabetic Retinopathy.
International Journal of Special Education, 41(24s), 745-753

ABSTRACT:

Background: Diabetic retinopathy (DR) is one of the most common microvascular complications of diabetes mellitus and a leading cause of preventable blindness worldwide. Dyslipidemia and oxidative stress have been implicated in the pathogenesis of DR by promoting endothelial dysfunction, inflammation, and retinal vascular damage. The present study aimed to evaluate the association between dyslipidemia, oxidative stress, and diabetic retinopathy in patients with type 2 diabetes mellitus.

Objectives: To assess lipid profile abnormalities and oxidative stress markers in patients with type 2 diabetes mellitus with and without diabetic retinopathy and to determine their association with the development of diabetic retinopathy.

Materials and Methods: A hospital-based case-control study was conducted on 100 participants aged 30–65 years, including 50 patients with type 2 diabetes mellitus and diabetic retinopathy (cases) and 50 patients with type 2 diabetes mellitus without diabetic retinopathy (controls). Fasting blood glucose (FBG), postprandial blood glucose (PPBG), glycated hemoglobin (HbA1c), lipid profile [total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C)], malondialdehyde (MDA), and superoxide dismutase (SOD) were estimated using standard laboratory methods. Statistical analysis was performed using Student's *t*-test and Pearson's correlation coefficient. A *p*-value <0.05 was considered statistically significant.

Results: The mean age of the cases was significantly higher than that of the controls (57.66 ± 6.07 vs. 45.00 ± 8.01 years, $p < 0.0001$). Patients with diabetic retinopathy exhibited significantly higher fasting blood glucose (180.08 ± 35.24 vs. 114.54 ± 28.57 mg/dL), postprandial blood glucose (248.16 ± 63.08 vs. 167.16 ± 24.37 mg/dL), and HbA1c (9.09 ± 2.35 vs. $5.66 \pm 0.38\%$; $p < 0.0001$ for all). Triglycerides (225.48 ± 144.69

COPYRIGHT STATEMENT:

Copyright: © 2026 Authors.
Open access publication under the
terms and conditions
of the Creative Commons Attribution
(CC BY)
license (<http://creativecommons.org/licenses/by/4.0/>).

vs. 155.04 ± 60.27 mg/dL; $p=0.002$) and VLDL-C (45.54 ± 18.11 vs. 34.70 ± 18.22 mg/dL; $p=0.0036$) were significantly elevated, whereas HDL-C was significantly reduced (38.14 ± 11.94 vs. 48.98 ± 12.11 mg/dL; $p<0.0001$) in cases. Total cholesterol and LDL-C did not differ significantly between the groups. Serum MDA levels were significantly higher in cases (3.81 ± 0.83 vs. 2.50 ± 1.35 ; $p<0.0001$), indicating increased oxidative stress, while SOD activity also differed significantly between the groups (179.72 ± 54.21 vs. 152.18 ± 52.96 ; $p=0.0117$). A strong positive correlation between MDA and SOD was observed in controls ($r=0.610$, $p<0.001$), whereas the correlation in cases was weak and not statistically significant ($r=0.262$, $p=0.664$).

Conclusion: Patients with diabetic retinopathy demonstrated significant dyslipidemia characterized by elevated triglycerides and VLDL-C, reduced HDL-C, poor glycemic control, and increased oxidative stress. These findings suggest that dyslipidemia-induced oxidative stress plays an important role in the pathogenesis of diabetic retinopathy. Early monitoring and management of lipid abnormalities and oxidative stress may help reduce the risk and progression of diabetic retinopathy in patients with type 2 diabetes mellitus.

Keywords: Diabetic retinopathy, Dyslipidemia, Oxidative stress, Malondialdehyde, Superoxide dismutase, Lipid profile, Type 2 diabetes mellitus.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It has emerged as one of the fastest-growing global health challenges, with an estimated 537 million adults living with diabetes worldwide in 2021, a number projected to increase substantially over the coming decades (1). Long-standing diabetes is associated with several microvascular and macrovascular complications, among which diabetic retinopathy (DR) remains the leading cause of preventable blindness among the working-age population. The prevalence and severity of diabetic retinopathy increase with the duration of diabetes, poor glycemic control, hypertension, and metabolic abnormalities (2).

Diabetic retinopathy is a progressive neurovascular disorder characterized by retinal microvascular damage, capillary basement

membrane thickening, pericyte loss, endothelial dysfunction, retinal ischemia, increased vascular permeability, and pathological neovascularization in advanced stages (3). Although chronic hyperglycemia is the primary pathogenic factor, growing evidence suggests that diabetic retinopathy is a multifactorial disease involving dyslipidemia, oxidative stress, chronic inflammation, genetic susceptibility, and endothelial dysfunction (4).

Dyslipidemia is a common metabolic abnormality in patients with type 2 diabetes mellitus and is characterized by elevated triglycerides, increased very low-density lipoprotein (VLDL), increased small dense low-density lipoprotein (LDL), and reduced high-density lipoprotein (HDL) cholesterol (5). Besides contributing to cardiovascular morbidity, dyslipidemia has been increasingly recognized as an important risk factor for the development and progression of diabetic retinopathy. Elevated

serum triglycerides and cholesterol have been associated with retinal hard exudates, diabetic macular edema, and worsening retinal vascular damage (6).

One of the principal mechanisms linking dyslipidemia with diabetic retinopathy is oxidative stress. Oxidative stress occurs due to an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense system. The retina is particularly vulnerable to oxidative damage because of its high metabolic activity, increased oxygen consumption, and abundance of polyunsaturated fatty acids. Persistent hyperglycemia enhances ROS production through several biochemical pathways, including mitochondrial dysfunction, activation of the polyol pathway, protein kinase C activation, advanced glycation end-product (AGE) formation, and the hexosamine pathway (7). Excessive ROS production leads to lipid peroxidation, DNA damage, protein oxidation, endothelial dysfunction, and apoptosis of retinal cells, thereby accelerating the progression of diabetic retinopathy (8).

Lipid peroxidation is considered one of the earliest biochemical events in oxidative stress. Malondialdehyde (MDA), a stable end-product of lipid peroxidation, is widely used as a reliable biomarker for assessing oxidative stress. Elevated serum MDA levels have been reported in patients with diabetic retinopathy, indicating increased oxidative damage to retinal tissues (9). Conversely, superoxide dismutase (SOD), an important enzymatic antioxidant, protects retinal cells by catalyzing the conversion of superoxide radicals into hydrogen peroxide and molecular oxygen. Altered SOD activity reflects impaired antioxidant defense mechanisms and has been implicated in the progression of diabetic retinopathy (10).

Recent studies suggest that dyslipidemia and oxidative stress act synergistically in the pathogenesis of diabetic retinopathy. Lipid abnormalities increase oxidative stress through enhanced lipid peroxidation, while oxidative damage further modifies lipoproteins, perpetuating endothelial dysfunction,

inflammation, capillary occlusion, and retinal neurodegeneration. This vicious cycle contributes significantly to retinal vascular damage and disease progression (11,12).

Despite considerable advances in understanding the molecular mechanisms of diabetic retinopathy, the relationship between dyslipidemia-induced oxidative stress and retinal damage remains incompletely understood, particularly in the Indian population. Identification of biochemical markers associated with oxidative stress may facilitate early diagnosis, improve risk stratification, and aid in the development of targeted therapeutic interventions. Therefore, the present study was undertaken to evaluate the association between dyslipidemia-induced oxidative stress and diabetic retinopathy by assessing lipid profile parameters and oxidative stress biomarkers, namely malondialdehyde (MDA) and superoxide dismutase (SOD), in patients with type 2 diabetes mellitus with and without diabetic retinopathy.

Aim

To evaluate the association between **dyslipidemia-induced oxidative stress and diabetic retinopathy** in patients with type 2 diabetes mellitus by assessing lipid profile parameters and oxidative stress biomarkers.

Objectives

Primary Objectives

1. To compare the lipid profile parameters [Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), and Very Low-Density Lipoprotein (VLDL)] between patients with type 2 diabetes mellitus with diabetic retinopathy and those without diabetic retinopathy.
2. To estimate and compare serum oxidative stress biomarkers, namely **Malondialdehyde (MDA)** and **Superoxide Dismutase (SOD)**, in both study groups.

3. To evaluate the association between dyslipidemia and oxidative stress in patients with diabetic retinopathy.

Secondary Objectives

4. To compare glycemic parameters (Fasting Blood Sugar, Postprandial Blood Sugar, and HbA1c) between diabetic patients with and without retinopathy.
5. To determine the correlation between serum MDA and SOD levels among diabetic patients with and without retinopathy.

MATERIALS AND METHODS

Study Design

The present study was a **hospital-based observational case-control study** conducted to evaluate the association between dyslipidemia-induced oxidative stress and diabetic retinopathy in patients with type 2 diabetes mellitus.

Study Setting

The study was carried out in the **Department of Biochemistry** in collaboration with the **Department of Ophthalmology**, Santosh Medical College and Hospital, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.

Study Duration

The study was conducted over a period of **12 months**.

Study Population

Patients diagnosed with **Type 2 Diabetes Mellitus (T2DM)** attending the Ophthalmology Outpatient Department (OPD) were recruited after obtaining written informed consent.

Sample Size

A total of **100 participants** were enrolled in the study.

- **Cases:** 50 patients with Type 2 Diabetes Mellitus having diabetic retinopathy.
- **Controls:** 50 patients with Type 2 Diabetes Mellitus without diabetic retinopathy.

Inclusion Criteria

Cases

- Patients aged **30–65 years**.
- Diagnosed cases of Type 2 Diabetes Mellitus.
- Presence of diabetic retinopathy confirmed by ophthalmological examination.
- Patients willing to participate and provide written informed consent.

Controls

- Patients aged **30–65 years**.
- Diagnosed cases of Type 2 Diabetes Mellitus.
- No evidence of diabetic retinopathy on ophthalmological examination.
- Written informed consent obtained.

Exclusion Criteria

Patients with any of the following were excluded:

- Type 1 Diabetes Mellitus.
- Age below 30 years or above 65 years.
- Pregnancy.
- Chronic kidney disease.
- Chronic liver disease.
- Malignancy.
- Autoimmune disorders.
- Acute or chronic inflammatory diseases.
- History of ocular diseases other than diabetic retinopathy.
- Patients receiving antioxidant supplementation or lipid-lowering therapy during the previous three months.
- Patients unwilling to participate.

Clinical Assessment

A detailed clinical history including age, sex, duration of diabetes, medication history, and

anthropometric measurements (height, weight, and body mass index) was recorded.

Sample Collection

Following an overnight fasting period of **8–10 hours**, approximately **5 mL of venous blood** was collected under aseptic precautions.

- **2 mL** was collected in fluoride vials for glucose estimation.
- **2 mL** was collected in EDTA vials for HbA1c estimation.
- **Remaining blood** was collected in plain tubes for serum separation and biochemical investigations.

Serum was separated by centrifugation at **3000 rpm for 10 minutes** and analyzed immediately or stored at **–20°C** until analysis.

Laboratory Methods

Parameter	Method
Fasting Blood Sugar	Glucose Oxidase–Peroxidase (GOD-POD) Method
Postprandial Blood Sugar	Glucose Oxidase–Peroxidase (GOD-POD) Method
HbA1c	High Performance Liquid Chromatography (HPLC)
Total Cholesterol	Enzymatic CHOD-POD Method
Triglycerides	Enzymatic GPO-POD Method
HDL-Cholesterol	Direct Homogeneous Enzymatic Method
LDL-Cholesterol	Friedewald Formula/Direct Method
VLDL-Cholesterol	Calculated as TG/5
MDA	Thiobarbituric Acid Reactive Substances (TBARS) Assay
SOD	Pyrogallol Autoxidation Inhibition Method

Biochemical Investigations

Glycemic Parameters

- Fasting Blood Sugar (FBS)
- Postprandial Blood Sugar (PPBS)
- Glycated Hemoglobin (HbA1c)

Lipid Profile

- Total Cholesterol (TC)
- Triglycerides (TG)
- High-Density Lipoprotein Cholesterol (HDL-C)
- Low-Density Lipoprotein Cholesterol (LDL-C)
- Very Low-Density Lipoprotein Cholesterol (VLDL-C)

Oxidative Stress Markers

- Serum Malondialdehyde (MDA)
- Serum Superoxide Dismutase (SOD)

Diagnosis of Diabetic Retinopathy

Diabetic retinopathy was diagnosed by an experienced ophthalmologist using **fundus examination** following pupillary dilatation.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using **Statistical Package for the Social Sciences (SPSS) version 25.0**.

- Continuous variables were expressed as **Mean $\pm$ Standard Deviation (SD)**.
- Categorical variables were expressed as **frequency and percentage**.
- Student's independent **t-test** was used for comparison between two groups.

- Pearson's correlation coefficient was used to assess the correlation between serum MDA and SOD levels.
- A **p-value <0.05** was considered statistically significant.

Ethical Considerations

The study was conducted after obtaining approval from the **Institutional Ethics**

Committee, Santosh Medical College and Hospital, Ghaziabad. Written informed consent was obtained from all study participants before enrolment. Confidentiality of participants' information was maintained throughout the study, and the study procedures complied with the ethical principles of the **Declaration of Helsinki**.

Results and Discussion

Parameters	Cases (DM with Retinopathy) (n=50)	Controls (DM without Retinopathy) (n=50)	p-value
Age (years), Mean $\pm$ SD	57.66 $\pm$ 6.07	45.00 $\pm$ 8.01	<0.0001
Weight (kg), Mean $\pm$ SD	70.41 $\pm$ 13.93	70.31 $\pm$ 8.21	0.4130
Height (cm), Mean $\pm$ SD	164.10 $\pm$ 7.93	163.52 $\pm$ 5.84	0.6780
BMI (kg/m ²), Mean $\pm$ SD	26.19 $\pm$ 4.77	27.14 $\pm$ 3.70	0.2685
Fasting Blood Sugar (mg/dL), Mean $\pm$ SD	180.08 $\pm$ 35.24	114.54 $\pm$ 28.57	<0.0001
Post-prandial Blood Sugar (mg/dL), Mean $\pm$ SD	248.16 $\pm$ 63.08	167.16 $\pm$ 24.37	<0.0001
HbA1c (%), Mean $\pm$ SD	9.09 $\pm$ 2.35	5.66 $\pm$ 0.38	<0.0001
Total Cholesterol (mg/dL), Mean $\pm$ SD	192.74 $\pm$ 61.37	189.94 $\pm$ 68.42	0.3701
Triglycerides (mg/dL), Mean $\pm$ SD	225.48 $\pm$ 144.69	155.04 $\pm$ 60.27	0.0020
HDL (mg/dL), Mean $\pm$ SD	38.14 $\pm$ 11.94	48.98 $\pm$ 12.11	<0.0001
LDL (mg/dL), Mean $\pm$ SD	98.70 $\pm$ 33.79	105.02 $\pm$ 34.43	0.3565
VLDL (mg/dL), Mean $\pm$ SD	45.54 $\pm$ 18.11	34.70 $\pm$ 18.22	0.0036
MDA, Mean $\pm$ SD	3.81 $\pm$ 0.83	2.50 $\pm$ 1.35	<0.0001
SOD, Mean $\pm$ SD	179.72 $\pm$ 54.21	152.18 $\pm$ 52.96	0.0117

A total of **100 participants** were enrolled in the study, comprising **50 patients with diabetic retinopathy (cases)** and **50 diabetic patients without retinopathy (controls)**. The mean age of the cases (**57.66 $\pm$ 6.07 years**) was significantly higher than that of the controls

(**45.00 $\pm$ 8.01 years**) (**p < 0.0001**). No significant differences were observed in weight, height, or BMI between the two groups (**p > 0.05**). Patients with diabetic retinopathy exhibited significantly higher fasting blood glucose, postprandial blood glucose, HbA1c, triglycerides, VLDL, and MDA

levels compared with controls. HDL cholesterol was significantly lower in cases, whereas total cholesterol and LDL cholesterol did not differ significantly between the two groups. Serum SOD activity also showed a statistically significant difference between cases and controls.

4. Results And Discussion

4.1 Age and Anthropometric Characteristics

The present study included 100 participants, comprising 50 patients with type 2 diabetes mellitus and diabetic retinopathy (cases) and 50 patients with type 2 diabetes mellitus without diabetic retinopathy (controls). The mean age of the cases (57.66 ± 6.07 years) was significantly higher than that of the controls (45.00 ± 8.01 years) ($p < 0.0001$). These findings suggest that increasing age is associated with a greater risk of developing diabetic retinopathy.

The higher age observed among patients with diabetic retinopathy is consistent with previous studies demonstrating that increasing age and longer duration of diabetes are major risk factors for retinal microvascular damage (13,14). Chronic exposure to hyperglycemia over time promotes endothelial dysfunction, oxidative stress, and progressive retinal capillary damage, thereby increasing the likelihood of retinopathy.

No statistically significant differences were observed in weight, height, or BMI between the two groups ($p > 0.05$). Similar observations have been reported by previous investigators, suggesting that obesity alone may not independently predict diabetic retinopathy after the onset of diabetes (14).

4.2 Glycemic Parameters

In the present study, fasting blood glucose, postprandial blood glucose, and HbA1c levels were significantly higher in patients with diabetic retinopathy than in diabetic patients without retinopathy ($p < 0.0001$). These findings indicate poor glycemic control among patients with retinal complications.

Persistent hyperglycemia activates several biochemical pathways, including the polyol

pathway, protein kinase C activation, advanced glycation end-product formation, and mitochondrial overproduction of reactive oxygen species, ultimately leading to retinal endothelial dysfunction and capillary cell apoptosis (15,16). These mechanisms are recognized as central to the development and progression of diabetic retinopathy.

4.3 Lipid Profile

The present study demonstrated significantly higher serum triglyceride and VLDL concentrations together with significantly lower HDL cholesterol concentrations among patients with diabetic retinopathy, whereas total cholesterol and LDL cholesterol did not differ significantly between the two groups.

These observations support the concept that diabetic dyslipidemia contributes to retinal microvascular injury primarily through elevated triglyceride-rich lipoproteins and reduced HDL cholesterol (17). Elevated triglycerides promote lipid deposition within retinal capillaries and increase vascular permeability, whereas HDL normally exerts antioxidant and anti-inflammatory effects. Reduced HDL therefore enhances oxidative damage and endothelial dysfunction.

The findings of the present study are also comparable with those reported by Kumari et al., who demonstrated a positive relationship between dyslipidemia and oxidative stress in diabetic retinopathy (18).

4.4 Oxidative Stress Markers

A major finding of the present study was the significantly elevated serum MDA concentration in patients with diabetic retinopathy compared with controls ($p < 0.0001$). MDA is a well-established biomarker of lipid peroxidation and reflects increased oxidative stress.

Hyperglycemia-induced oxidative stress accelerates lipid peroxidation of cellular membranes, producing MDA, which further damages retinal endothelial cells, pericytes, and retinal pigment epithelial cells (15,19). Several clinical studies and a recent meta-analysis have consistently demonstrated significantly higher

circulating MDA levels in patients with diabetic retinopathy than in diabetic patients without retinopathy.

Serum SOD activity also differed significantly between the two groups. Cases Showed higher levels than Controls $p = 0.0117$. Although several studies have reported reduced SOD activity in diabetic retinopathy, the present study demonstrated significantly higher SOD levels among cases. This may represent a compensatory increase in antioxidant enzyme activity in response to increased oxidative stress during the earlier stages of retinal damage.

Alterations in SOD activity reflect disruption of the endogenous antioxidant defense system. Experimental and clinical studies have shown that impaired antioxidant enzyme activity contributes to progressive retinal oxidative damage (19,20).

4.5 Correlation Between MDA and SOD

The present study demonstrated a weak positive but statistically non-significant correlation between MDA and SOD among patients with diabetic retinopathy ($r = 0.262$; $p = 0.664$). In contrast, diabetic patients without retinopathy exhibited a strong positive correlation ($r = 0.610$; $p < 0.001$).

References

- International Diabetes Federation. **IDF Diabetes Atlas**. 10th ed. Brussels, Belgium: International Diabetes Federation; 2021.
- Cheung N, Mitchell P, Wong TY. Diabetic retinopathy. **Lancet**. 2010;376(9735):745-753.
- Antonetti DA, Klein R, Gardner TW. Diabetic retinopathy. **N Engl J Med**. 2012;366(13):745-753.
- Kowluru RA, Mishra M. Oxidative stress and its regulation in diabetic retinopathy. **Antioxidants (Basel)**. 2023;12(8):1649.
- American Diabetes Association. Standards of Care in Diabetes—2024. **Diabetes Care**. 2024;47(Suppl 1):S1-S350.
- Sas KM, Karnovsky A, Michailidis G, Pennathur S. Metabolomics and dyslipidemia in diabetic retinopathy. **Front Endocrinol (Lausanne)**. 2021;12:620045.
- Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. **Diabetes**. 2005;54(6):745-753.
- Wu Y, Tang L, Chen B. Oxidative stress: implications for the development of diabetic retinopathy and antioxidant therapeutic perspectives. **Oxid Med Cell Longev**. 2014;2014:752387.
- Kumari S, Panda S, Mangaraj M, Mandal MK, Mahapatra PC. Plasma malondialdehyde and antioxidant vitamins in diabetic retinopathy. **Indian J Clin Biochem**. 2008;23(2):745-753.
- Kowluru RA, Chan PS. Oxidative stress and diabetic retinopathy. **Exp Diabetes Res**. 2007;2007:43603.

The preserved association between MDA and SOD in diabetic patients without retinopathy may represent an intact compensatory antioxidant response. However, the loss of this relationship in patients with diabetic retinopathy suggests exhaustion of antioxidant defense mechanisms due to prolonged oxidative stress (18,19). Similar alterations in oxidative stress biomarkers have been reported in previous clinical studies investigating diabetic retinopathy.

4.6 Overall Interpretation

Overall, the present study demonstrates that diabetic retinopathy is associated with poor glycemic control, dyslipidemia characterized by elevated triglycerides and VLDL together with reduced HDL cholesterol, and increased oxidative stress as evidenced by elevated serum MDA. These findings support the hypothesis that dyslipidemia-induced oxidative stress plays a significant role in the pathogenesis of diabetic retinopathy. Early identification and management of lipid abnormalities and oxidative stress may therefore help delay the onset and progression of retinal complications in patients with type 2 diabetes mellitus (15,17,19).

- Stitt AW, Curtis TM, Chen M, et al. The progress in understanding and treatment of diabetic retinopathy. **Prog Retin Eye Res.** 2016;51:745-753.
- Wong TY, Cheung CMG, Larsen M, Sharma S, Simó R. Diabetic retinopathy. **Nat Rev Dis Primers.** 2016;2:16012.
- Antonetti DA, Silva PS, Stitt AW. Current understanding of the molecular and cellular pathology of diabetic retinopathy. *Nat Rev Endocrinol.* 2021;17:195–206.
- Madsen-Bouterse SA, Kowluru RA. Oxidative stress and diabetic retinopathy: pathophysiological mechanisms and treatment perspectives. *Rev Endocr Metab Disord.* 2008;9:315–327.
- Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. *Diabetes.* 2005;54:1615–1625.
- Antonetti DA, Klein R, Gardner TW. Diabetic retinopathy. *N Engl J Med.* 2012;366:1227–1239.
- Sas KM, Karnovsky A, Michailidis G, Pennathur S. Emerging roles of dyslipidemia and hyperglycemia in diabetic retinopathy: molecular mechanisms and clinical perspectives. *Front Endocrinol.* 2021;12:620045.
- Kumari S, Panda S, Mangaraj M, Mandal MK, Mahapatra PC. Plasma MDA and antioxidant vitamins in diabetic retinopathy. *Indian J Clin Biochem.* 2008;23(2):158–162.
- Kowluru RA, Mishra M. Oxidative stress and its regulation in diabetic retinopathy. *Antioxidants (Basel).* 2023;12:1649.
- Malondialdehyde levels in diabetic retinopathy patients: a systematic review and meta-analysis. *Medicine (Baltimore).* 2023.