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ASSESSMENT OF DIABETIC RETINOPATHY AND ITS ASSOCIATED RISK FACTORS AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT

Diabetic retinopathy (DR) is one of the most significant microvascular complications of type 2 diabetes mellitus (T2DM) and a leading cause of preventable blindness worldwide. The present study aims to assess the prevalence of diabetic retinopathy and identify its associated risk factors among patients with type 2 diabetes mellitus. This study focuses on key clinical, demographic, and biochemical parameters such as duration of diabetes, glycemic control, hypertension, dyslipidemia, obesity, and lifestyle factors. Early detection of DR and timely management of modifiable risk factors are crucial in preventing vision loss. The findings highlight the importance of regular ophthalmic screening and integrated diabetes care to reduce the burden of diabetic eye disease.

Keywords: - Diabetic Retinopathy, Type 2 Diabetes Mellitus, Risk Factors, Glycemic Control, Hypertension

I. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from insulin resistance and relative insulin deficiency. It has emerged as one of the most significant global public health challenges due to its rapidly increasing prevalence, long-term complications, and associated economic burden on healthcare systems. The condition is strongly influenced by genetic predisposition, sedentary lifestyle, unhealthy dietary habits, obesity, and advancing age. According to global estimates, the number of individuals affected by diabetes continues to rise sharply, particularly in developing countries, where urbanization and lifestyle transitions have contributed significantly to disease burden. Among the various complications associated with T2DM, microvascular complications such as diabetic retinopathy (DR), diabetic nephropathy, and diabetic neuropathy are particularly important because they directly affect the quality of life and may lead to permanent disability if not diagnosed and managed early.

Diabetic retinopathy is one of the most common and vision-threatening complications of diabetes mellitus. It is a progressive retinal microvascular disorder caused primarily by chronic hyperglycemia, which leads to damage of the retinal blood vessels. Over time, sustained high blood glucose levels result in biochemical and structural changes in the retinal capillaries, including basement membrane thickening, pericyte loss, endothelial dysfunction, and microaneurysm formation. These pathological changes contribute to retinal ischemia, increased vascular permeability, hemorrhages, and in advanced stages, neovascularization. If left untreated, diabetic retinopathy can progress to diabetic macular edema and proliferative diabetic retinopathy, both of which are major causes of irreversible visual impairment and blindness worldwide.

The burden of diabetic retinopathy is increasing in parallel with the rising prevalence of diabetes mellitus. It is estimated that a significant proportion of patients with long-standing diabetes develop some degree of retinal involvement during their disease course. The risk of developing diabetic retinopathy increases with the duration of diabetes, poor glycemic control, and the presence of associated systemic conditions such as hypertension and dyslipidemia. The condition often remains asymptomatic in its early stages, which makes regular screening and early detection extremely important. Many patients are diagnosed only when the disease has progressed to an advanced stage, at which point treatment options may be limited and visual prognosis becomes poor.

Several risk factors have been identified in the development and progression of diabetic retinopathy. The most important among these is the duration of diabetes, as longer exposure to hyperglycemia significantly increases retinal damage. Glycemic control, commonly assessed by glycated hemoglobin (HbA1c), plays a critical role, as poor glycemic control is strongly associated with both the onset and severity of retinopathy. Hypertension is another major risk factor that accelerates microvascular damage in the retina by increasing capillary pressure and promoting vascular leakage. Dyslipidemia, characterized by elevated serum cholesterol and triglyceride levels, contributes to the formation of hard exudates and retinal edema. In addition, factors such as obesity, smoking, physical inactivity, and genetic predisposition further increase the risk of diabetic retinopathy. The interaction of these risk factors determines the overall progression and severity of the disease.

From a clinical perspective, diabetic retinopathy is classified into non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR). NPDR is the early stage of the disease and is characterized by microaneurysms, retinal hemorrhages, and hard exudates, whereas PDR represents an advanced stage marked by abnormal neovascularization and fibrous tissue formation. The progression from NPDR to PDR is associated with an increased risk of severe vision loss due to vitreous hemorrhage and retinal detachment. Another important complication is diabetic macular edema, which can occur at any stage of retinopathy and is a major cause of central vision impairment.

The global burden of diabetic retinopathy highlights the need for effective screening programs and preventive strategies. Early detection through regular fundoscopic examination or retinal imaging techniques such as fundus photography and optical coherence tomography (OCT) plays a crucial role in preventing disease progression. Timely intervention, including laser photocoagulation, intravitreal injections, and strict metabolic control, has been shown to significantly reduce the risk of vision loss. However, in many low- and middle-income countries, lack of awareness, limited access to eye care services, and inadequate screening programs contribute to delayed diagnosis and poor outcomes.

In addition to clinical factors, socioeconomic and healthcare-related determinants also influence the burden of diabetic retinopathy. Lack of awareness regarding diabetes complications, poor adherence to treatment, and inadequate follow-up care further exacerbate disease progression. Patients from rural and underserved populations are particularly at higher risk due to limited access to specialized ophthalmic services. Therefore, public health interventions aimed at increasing awareness, improving screening coverage, and integrating eye care into diabetes management programs are essential for reducing the burden of diabetic retinopathy.

Given the increasing prevalence of type 2 diabetes mellitus and its complications, it is important to understand the pattern and determinants of diabetic retinopathy in affected populations. Assessment of risk factors not only helps in identifying high-risk individuals but also aids in developing preventive strategies to delay or prevent the onset of vision-threatening complications. Studies focusing on the relationship between clinical, biochemical, and lifestyle factors with diabetic retinopathy are essential for improving patient outcomes and guiding healthcare policies.

II. PATHOPHYSIOLOGY OF RETINAL DAMAGE IN DIABETES

Diabetic retinopathy (DR) is a complex microvascular complication of diabetes mellitus that develops primarily due to chronic hyperglycemia and its metabolic consequences. The pathophysiology of retinal damage in diabetes involves a cascade of biochemical, cellular, and hemodynamic alterations that progressively disrupt the normal structure and function of the retinal microvasculature. These changes begin early in the course of diabetes and may remain clinically silent for years before becoming visually significant. The retina, being one of the most metabolically active tissues in the human body, is highly sensitive to fluctuations in glucose levels, making it particularly vulnerable to diabetic damage.

The initial trigger in the development of diabetic retinopathy is persistent hyperglycemia. Elevated blood glucose levels activate multiple metabolic pathways that contribute to vascular injury. One of the most important mechanisms is the polyol pathway, in which excess glucose is converted into sorbitol by the enzyme aldose reductase. Sorbitol accumulates within retinal cells, leading to osmotic stress, cellular swelling, and dysfunction of pericytes and endothelial cells. This process weakens the structural integrity of retinal capillaries and contributes to early microvascular damage.

Another important mechanism is the formation of advanced glycation end products (AGEs). In chronic hyperglycemia, glucose molecules bind irreversibly to proteins, lipids, and nucleic acids, forming AGEs. These compounds alter the structure and function of vascular basement membranes, increasing their thickness and reducing elasticity. AGEs also interact with specific receptors (RAGE) on endothelial cells, triggering inflammatory responses, oxidative stress, and the release of cytokines. This inflammatory environment further damages retinal microvessels and promotes vascular dysfunction.

Oxidative stress plays a central role in the progression of diabetic retinopathy. Hyperglycemia increases the production of reactive oxygen species (ROS) in retinal cells, overwhelming the antioxidant defense system. Excess ROS leads to damage of cellular proteins, lipids, and DNA, resulting in endothelial dysfunction and apoptosis of pericytes. Pericyte loss is one of the earliest histopathological features of diabetic retinopathy and contributes to capillary instability, microaneurysm formation, and increased vascular permeability.

Hemodynamic changes are also significant in the pathophysiology of diabetic retinal damage. Diabetes leads to impaired autoregulation of retinal blood flow, resulting in abnormal capillary perfusion. Initially, there is hyperperfusion, followed by areas of non-perfusion and ischemia as the disease progresses. Capillary basement membrane thickening and endothelial cell damage contribute to reduced oxygen delivery to retinal tissues. This ischemic state stimulates the release of hypoxia-inducible factors (HIFs), which promote the production of vascular endothelial growth factor (VEGF).

VEGF is a key mediator in the progression of advanced diabetic retinopathy. It increases vascular permeability, leading to leakage of plasma proteins and fluids into the retinal tissue, resulting in retinal edema. VEGF also stimulates the formation of new, fragile blood vessels (neovascularization) in response to ischemia. These newly formed vessels are structurally weak and prone to rupture, causing vitreous hemorrhage and further vision loss. Neovascularization is a hallmark of proliferative diabetic retinopathy, the most severe stage of the disease.

Inflammation is another critical component of retinal damage in diabetes. Chronic hyperglycemia induces the activation of inflammatory pathways, including the release of cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and adhesion molecules. These inflammatory mediators promote leukocyte adhesion to the vascular endothelium, a process known as leukostasis. Leukostasis leads to capillary occlusion, further ischemia, and breakdown of the blood-retinal barrier. The disruption of this barrier is responsible for fluid leakage and the development of diabetic macular edema, a major cause of vision impairment.

The blood-retinal barrier (BRB) consists of tight junctions between endothelial cells in retinal vessels and retinal pigment epithelial cells. In diabetes, this barrier becomes compromised due to endothelial dysfunction, inflammation, and VEGF overexpression. The breakdown of the BRB results in increased vascular permeability, leakage of plasma constituents, and accumulation of fluid in the retinal layers. This contributes to retinal thickening and distortion of the macular architecture, ultimately impairing central vision.

As diabetic retinopathy progresses, structural changes in the retina become more pronounced. Capillary dropout leads to areas of non-perfusion, while microaneurysms and hemorrhages become more frequent. In advanced stages, fibrous tissue formation accompanies neovascularization, which may lead to tractional retinal detachment. The combination of hemorrhage, edema, and retinal detachment significantly increases the risk of irreversible vision loss.

III. OPHTHALMIC EXAMINATION METHODS

Ophthalmic examination plays a crucial role in the early detection, classification, and monitoring of diabetic retinopathy (DR) in patients with type 2 diabetes mellitus. Since diabetic retinopathy is often asymptomatic in its early stages, systematic and comprehensive eye examinations are essential for timely diagnosis and prevention of vision-threatening complications. Various diagnostic techniques are used in clinical practice, ranging from basic visual assessment methods to advanced retinal imaging technologies. These methods help in evaluating retinal structure, identifying microvascular changes, and determining the severity of disease progression.

The initial step in ophthalmic evaluation is visual acuity testing, which is commonly performed using a Snellen chart or LogMAR chart. This test measures the clarity or sharpness of vision and helps in identifying any reduction in visual function. Although visual acuity may remain normal in early diabetic retinopathy, a decline in vision may indicate the presence of macular edema or advanced retinal involvement. Therefore, visual acuity assessment serves as a baseline tool in all diabetic patients undergoing eye examination.

Another fundamental component of ophthalmic evaluation is pupil examination and anterior segment assessment, which is performed using a slit lamp biomicroscope. This examination allows detailed visualization of the cornea, anterior chamber, iris, and lens. In diabetic patients, the slit lamp is also used to detect associated complications such as cataracts, which are more common in individuals with long-standing diabetes. Although diabetic retinopathy primarily affects the posterior segment, anterior segment examination helps in ruling out other ocular conditions that may contribute to visual impairment.

The most important part of diabetic retinopathy evaluation is the fundus examination, which allows direct visualization of the retina, optic disc, macula, and retinal vasculature. This can be performed using direct ophthalmoscopy, indirect ophthalmoscopy, or slit lamp biomicroscopy with a fundus lens. Fundus examination helps in identifying characteristic signs of diabetic retinopathy such as microaneurysms, retinal hemorrhages, hard exudates, cotton wool spots, venous beading, and neovascularization. Based on these findings, diabetic retinopathy is classified into non-proliferative and proliferative stages.

Fundus photography is another widely used method for documenting retinal changes. It involves capturing high-resolution images of the retina, which can be stored for comparison over time. Fundus photography is highly useful in screening programs as it allows for remote evaluation and teleophthalmology applications. It also provides objective documentation of disease progression and response to treatment.

One of the most advanced and sensitive imaging techniques used in diabetic retinopathy assessment is Optical Coherence Tomography (OCT). OCT is a non-invasive imaging modality that provides cross-sectional, high-resolution images of the retina. It is particularly useful in detecting and quantifying macular edema, which is a major cause of vision loss in diabetic patients. OCT helps in measuring retinal thickness and identifying fluid accumulation within retinal layers, even before clinical symptoms appear.

In addition to OCT, fluorescein angiography (FA) is an important diagnostic tool used to evaluate retinal blood flow and vascular abnormalities. In this procedure, a fluorescent dye is injected into the bloodstream, and sequential retinal images are captured as the dye circulates through retinal vessels. FA helps in identifying areas of capillary non-perfusion, microaneurysms, leakage, and neovascularization. It is particularly useful in planning laser photocoagulation therapy in patients with proliferative diabetic retinopathy.

Color fundus imaging systems and digital retinal screening cameras are increasingly being used in population-based screening programs. These devices allow quick, non-invasive retinal imaging and can be used in primary healthcare settings. The images can be reviewed by ophthalmologists remotely, improving access to eye care services, especially in rural and underserved regions.

Another supportive method is tonometry, which measures intraocular pressure (IOP). Although not directly related to diabetic retinopathy, it is important in ruling out coexisting conditions such as glaucoma, which may further compromise vision in diabetic patients.

In clinical research and epidemiological studies, grading of diabetic retinopathy is often performed using standardized classification systems such as the Early Treatment Diabetic Retinopathy Study (ETDRS) scale or the International Clinical Diabetic Retinopathy Disease Severity Scale. These grading systems ensure uniformity in diagnosis and help in comparing disease severity across different populations.

IV. ASSOCIATION BETWEEN RISK FACTORS AND DR

Diabetic retinopathy (DR) is a multifactorial microvascular complication of diabetes mellitus, and its development is strongly influenced by a combination of systemic, metabolic, and lifestyle-related risk factors. The association between these risk factors and DR has been extensively studied, and evidence consistently shows that both modifiable and non-modifiable factors contribute to the onset, progression, and severity of retinal damage in patients with type 2 diabetes mellitus (T2DM). Understanding these associations is essential for early identification of high-risk individuals and for implementing preventive strategies aimed at reducing vision loss.

One of the most significant and consistently reported risk factors for diabetic retinopathy is the duration of diabetes mellitus. Numerous clinical studies have demonstrated a strong positive correlation between the length of time a patient has had diabetes and the likelihood of developing retinopathy. Patients with a shorter duration of diabetes (less than five years) generally show a lower prevalence of DR, whereas those with more than ten to fifteen years of disease duration exhibit a substantially higher risk. This relationship is primarily due to prolonged exposure to hyperglycemia, which leads to cumulative damage to retinal microvasculature over time. The longer the exposure, the greater the likelihood of structural and functional retinal changes.

Another critical factor associated with diabetic retinopathy is glycemic control, commonly assessed using glycated hemoglobin (HbA1c). Poor glycemic control is one of the strongest predictors of both the onset and progression of DR. Elevated HbA1c levels reflect chronic hyperglycemia, which triggers multiple biochemical pathways such as oxidative stress, formation of advanced glycation end products, and activation of inflammatory responses. These processes contribute to endothelial dysfunction, capillary basement membrane thickening, and pericyte loss in the retina. Patients with persistently high HbA1c levels are significantly more likely to develop moderate to severe forms of diabetic retinopathy compared to those maintaining optimal glycemic control.

Hypertension is another major systemic risk factor closely associated with diabetic retinopathy. High blood pressure exacerbates retinal vascular damage by increasing hydrostatic pressure within retinal capillaries, leading to endothelial injury and increased vascular permeability. The combination of hypertension and diabetes has a synergistic effect, accelerating the progression of retinopathy. Studies have shown that diabetic patients with uncontrolled hypertension are at a significantly higher risk of developing advanced stages of DR, including proliferative diabetic retinopathy and diabetic macular edema. Therefore, blood pressure control is considered an essential component in the prevention and management of DR.

Dyslipidemia, characterized by elevated levels of serum cholesterol, low-density lipoprotein (LDL), and triglycerides, also plays an important role in the development of diabetic retinopathy. Lipid abnormalities contribute to the formation of hard exudates in the retina due to leakage of lipoproteins from damaged retinal vessels. These exudates are commonly observed in patients with diabetic macular edema and are associated with visual impairment. Moreover, dyslipidemia promotes oxidative stress and endothelial dysfunction, further aggravating retinal vascular damage. Clinical evidence suggests that patients with poorly controlled lipid profiles are more likely to experience progression of diabetic retinopathy.

Obesity is another important risk factor linked to diabetic retinopathy. Obesity is strongly associated with insulin resistance, chronic low-grade inflammation, and metabolic dysfunction, all of which contribute to the pathogenesis of DR. Adipose tissue secretes inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukins, which promote vascular inflammation and endothelial damage. Additionally, obesity is often associated with poor glycemic control and hypertension, thereby indirectly increasing the risk of retinal complications.

Smoking is a well-established modifiable risk factor that has been shown to worsen diabetic complications, including retinopathy. Smoking induces oxidative stress, reduces oxygen delivery to retinal tissues, and promotes vascular constriction. These effects contribute to worsening retinal ischemia and accelerate microvascular damage. Smokers with diabetes are more likely to develop severe forms of retinopathy compared to non-smokers. Despite this strong association, smoking remains a preventable risk factor, highlighting the importance of lifestyle modification in diabetic patients.

Genetic predisposition also plays a role in the development of diabetic retinopathy. Although not fully understood, certain genetic factors may influence individual susceptibility to retinal microvascular damage. Variations in genes related to inflammatory pathways, angiogenesis, and oxidative stress have been implicated in increasing the risk of DR. However, genetic factors alone are not sufficient to cause retinopathy; they interact with environmental and metabolic factors to determine disease expression.

Physical inactivity is another contributing factor that indirectly increases the risk of diabetic retinopathy. Sedentary lifestyle leads to poor glycemic control, weight gain, and increased cardiovascular risk, all of which are associated with DR. Regular physical activity improves insulin sensitivity, helps control blood glucose levels, and reduces systemic inflammation, thereby lowering the risk of microvascular complications.

The association between risk factors and diabetic retinopathy is often multifactorial and interdependent. For example, a patient with long-standing diabetes is more likely to have poor glycemic control, hypertension, and dyslipidemia simultaneously, all of which collectively increase the risk of retinal damage. This clustering of risk factors creates a cumulative effect that significantly accelerates disease progression. Therefore, it is not a single factor but the interaction of multiple factors that determines the severity of diabetic retinopathy.

Clinical studies have consistently shown that patients with multiple uncontrolled risk factors are at the highest risk of developing proliferative diabetic retinopathy and diabetic macular edema. On the other hand, patients who maintain good glycemic control, normal blood pressure, and healthy lipid levels have a significantly reduced risk of disease progression. This clearly demonstrates the importance of comprehensive metabolic control in preventing diabetic eye disease.

V. BIOLOGICAL EXPLANATION OF RISK FACTORS

Diabetic retinopathy (DR) is fundamentally a microvascular complication of diabetes mellitus that arises due to prolonged metabolic disturbances affecting the retinal tissue. The biological basis of risk factors associated with DR is rooted in complex interactions between hyperglycemia-induced biochemical pathways, vascular dysfunction, inflammation, and oxidative stress. These mechanisms collectively damage the retinal microvasculature and lead to progressive visual impairment. Each major risk factor contributes to retinal pathology through specific biological processes that accelerate disease onset and progression.

The most important biological trigger in diabetic retinopathy is chronic hyperglycemia, which acts as the central driving force behind all downstream pathological events. Persistently elevated blood glucose levels activate several damaging metabolic pathways, including the polyol pathway, protein kinase C (PKC) activation, hexosamine pathway flux, and formation of advanced glycation end products (AGEs). These pathways disrupt normal cellular metabolism in retinal endothelial cells, pericytes, and neurons. One of the earliest biological changes is the loss of pericytes, which are essential for maintaining capillary stability. Their degeneration leads to weakened capillary walls, microaneurysm formation, and increased vascular fragility.

Duration of diabetes is biologically significant because prolonged exposure to hyperglycemia results in cumulative cellular and structural damage to retinal tissues. Over time, repeated metabolic stress leads to progressive thickening of the capillary basement membrane, reduced capillary perfusion, and capillary dropout. The retina becomes increasingly ischemic due to loss of functional microvasculature, which triggers compensatory but pathological angiogenic responses. Thus, longer disease duration corresponds to greater accumulation of irreversible microvascular injury.

A key biological mechanism linking risk factors to diabetic retinopathy is oxidative stress.

Hyperglycemia increases the production of reactive oxygen species (ROS) within retinal cells, overwhelming the natural antioxidant defense systems. Excess ROS damages lipids, proteins, and DNA, leading to endothelial dysfunction and apoptosis of retinal cells. Oxidative stress also amplifies inflammatory signaling pathways, further worsening vascular injury. This mechanism is strongly influenced by poor glycemic control, making elevated HbA1c levels a major biological risk factor.

Advanced glycation end products (AGEs) play a central role in the biological progression of diabetic retinopathy. AGEs are formed when glucose molecules non-enzymatically bind to proteins and lipids over time. These compounds accumulate in retinal tissues and alter the structural integrity of blood vessels. AGEs bind to their receptors (RAGE) on endothelial cells, activating intracellular signaling pathways that promote inflammation, oxidative stress, and vascular permeability. This interaction leads to thickening of the basement membrane, reduced elasticity of capillaries, and breakdown of the blood-retinal barrier.

The role of hypertension in diabetic retinopathy can be explained through its effects on retinal hemodynamics. Elevated systemic blood pressure increases hydrostatic pressure within retinal capillaries, causing mechanical stress on endothelial cells. This leads to endothelial injury, increased vascular permeability, and leakage of plasma components into retinal tissues. Chronic hypertension also impairs autoregulation of retinal blood flow, resulting in fluctuating perfusion and increased susceptibility to ischemic damage. The combination of hypertension and hyperglycemia significantly accelerates microvascular degeneration.

Dyslipidemia contributes biologically to retinal damage through lipid deposition and inflammatory mechanisms. Elevated serum lipids, particularly LDL cholesterol and triglycerides, infiltrate damaged retinal vessels and accumulate as hard exudates. These lipid deposits disrupt retinal architecture and are commonly associated with macular edema. Additionally, oxidized lipids trigger inflammatory responses and endothelial dysfunction, further compromising vascular integrity. Dyslipidemia also enhances oxidative stress, thereby intensifying hyperglycemia-induced retinal injury.

The biological impact of obesity on diabetic retinopathy is primarily mediated through chronic low-grade inflammation and insulin resistance. Adipose tissue acts as an active endocrine organ, secreting pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and leptin. These inflammatory mediators contribute to systemic vascular inflammation and endothelial dysfunction. Obesity also worsens insulin resistance, leading to poor glycemic control, which indirectly accelerates retinal damage through hyperglycemia-related pathways.

Smoking has direct biological effects on retinal microvasculature. Nicotine and other toxic substances in tobacco induce vasoconstriction, reduce oxygen delivery, and increase oxidative stress. Carbon monoxide from smoking reduces oxygen-carrying capacity of blood, worsening retinal hypoxia. Chronic smoking also promotes endothelial dysfunction and increases platelet aggregation, leading to microvascular occlusion. These effects collectively exacerbate retinal ischemia and promote progression to proliferative diabetic retinopathy.

A critical biological pathway in diabetic retinopathy is vascular endothelial growth factor (VEGF) activation. Retinal ischemia, resulting from capillary non-perfusion, stimulates the release of hypoxia-inducible factors (HIFs), which in turn increase VEGF expression. VEGF enhances vascular permeability and promotes neovascularization. However, these newly formed blood vessels are structurally fragile and prone to leakage and rupture, leading to vitreous hemorrhage and severe vision loss. Risk factors such as poor glycemic control, hypertension, and dyslipidemia all contribute to increased VEGF expression through ischemic and inflammatory pathways.

Another important biological mechanism is the breakdown of the blood-retinal barrier (BRB). The BRB normally regulates fluid and molecular exchange between the bloodstream and retinal tissue. In diabetes, endothelial cell dysfunction, inflammation, and VEGF overexpression disrupt tight junctions, leading to increased permeability. This results in fluid leakage into the retinal layers and the development of diabetic macular edema, a major cause of vision impairment.

VI. CONCLUSION

Diabetic retinopathy remains one of the most serious and vision-threatening microvascular complications of type 2 diabetes mellitus, with a growing global burden due to the increasing prevalence of diabetes. The present study highlights that diabetic retinopathy is a multifactorial disease, where its development and progression are strongly influenced by both modifiable and non-modifiable risk factors. Among these, duration of diabetes, poor glycemic control, hypertension, dyslipidemia, obesity, smoking, and sedentary lifestyle emerge as the most significant contributors.

The findings indicate that prolonged exposure to hyperglycemia plays a central role in retinal damage through mechanisms such as oxidative stress, formation of advanced glycation end products, inflammation, and endothelial dysfunction. These biological processes lead to progressive breakdown of the blood-retinal barrier, capillary occlusion, retinal ischemia, and abnormal neovascularization, ultimately resulting in visual impairment if not detected and managed early.

It is also evident that systemic conditions such as hypertension and dyslipidemia significantly accelerate the progression of diabetic retinopathy by worsening vascular injury and increasing retinal leakage. Lifestyle-related factors such as obesity, smoking, and physical inactivity further compound the risk by promoting metabolic imbalance and chronic inflammation. The combined effect of these risk factors results in a higher likelihood of developing severe stages of diabetic retinopathy, including proliferative diabetic retinopathy and diabetic macular edema.

The study emphasizes the importance of early screening and regular ophthalmic examinations in all patients with type 2 diabetes mellitus, especially those with long-standing disease or poor metabolic control. Timely detection using appropriate diagnostic methods such as fundus examination, fundus photography, and optical coherence tomography can significantly reduce the risk of irreversible vision loss. Furthermore, strict glycemic control, effective management of blood pressure and lipid levels, and adoption of healthy lifestyle practices are essential preventive strategies.

In conclusion, diabetic retinopathy is a preventable but potentially blinding complication of diabetes mellitus. Its progression is closely linked to multiple interrelated risk factors that can be effectively controlled through comprehensive diabetes management. Strengthening awareness, improving screening programs, and ensuring multidisciplinary care are crucial steps toward reducing the burden of diabetic retinopathy and preserving vision in patients with type 2 diabetes mellitus.

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