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DIABETIC RETINOPATHY: CLINICAL FINDINGS AND MANAGEMENT

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Abstract
Diabetic retinopathy (DR) is a global source of visual loss and a consequence of diabetes. One of the most common metabolic disorders (DMs), diabetes mellitus (DM) affects people's ability to lead healthy lives worldwide and can have mild to severe secondary effects. Diabetic retinopathy (DR), which damages the retina and may result in blindness, is one of the secondary consequences of diabetes mellitus (DM). Although there may be other ocular problems associated with diabetes, diabetic retinopathy and cataracts are the primary causes of vision loss in DM. Patients with diabetes frequently experience severe retinal capillary aneurysms, haemorrhage, and edoema. These conditions might eventually result in diabetic macular edoema (DME) and non-proliferative or proliferative diabetic retinopathy (NPDR or PDR).Our goal in this study is to give a thorough overview of the epidemiology, pathophysiology, and potential treatment options of diabetic retinopathy, as well as how it progresses.

Keywords: diabetic retinopathy (DR), diabetic mellitus (DM), vision loss, edema, complications.

Introduction

Micro vascular disease is the cause of diabetic retinopathy. One of the main causes of vision impairment is diabetes mellitus (DM). It has a variety of structural and functional effectson the majority of the ocular and visual systems. Although there are undoubtedly other issues, diabetic retinopathy and diabetic cataract are the primary causes of visual impairment in DM. It is still often believed that having great blood glucose control lowers the likelihood of developing problems from diabetes (1).

The body's vasculature is compromised by the unbalanced metabolism of carbohydrates brought on by diabetes, which is typified by hyperglycaemias and the ensuing disruptions of protein and lipid synthesis. When examined in isolation, the intricate array of events that makes up the retina's distinctive reaction to diabetes appears normal. The integrity of the body's vasculature is harmed by the imbalance in glucose metabolism that arises from diabetes, which is marked by hyperglycaemia and related disruptions in lipid and protein production. Globally, DR is a major factor in blindness and vision impairment among working-class people (ages 20 to 65).

Hyperglycaemia is the cause of 2.6% of blindness worldwide3. Although some research has shown a strong correlation with sleep pane, post-translational modifications of histones within chromatins, and methylation of DNA and non-coding RNAs, its pathophysiology is likewise unknown. Non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR) are the two stages of Receding to the Vision Loss Expert Group (VLEG), DR was responsible for 1.07% of blindness and 1.25% of moderate to severe vision impairment.

1.1 : Incidence of Diabetic Retinopathy

Makes blindness 2s5 times more frequent in diabetics than no diabetics. Retinopathy may exist in some diabetic patients without visual impairment; however, 15% of diabetics have a level of retinopathy more likely to show market visual impairment if left untreated.

For instance, instance, in 1930 instance, in 1930 of new cases of blindness in the United States were associated with diabetes, by 1960, with advanced level of diabetic care which increased life expectancy and the associated long-term complications, this figure had risen to around 15%.

Crosby reported a lower rate of 7% of 10,000 new blind registrations for the period 1958–1962 whereas in the

following interval, 1963–1968, this increased to 13% of male and 18.2% of female blind registrations. Palmer found that diabetic retinopathy to be responsible for about 10% of the new blind registrations at all ages, about 20% of who were between the ages of 45–74 years (2).

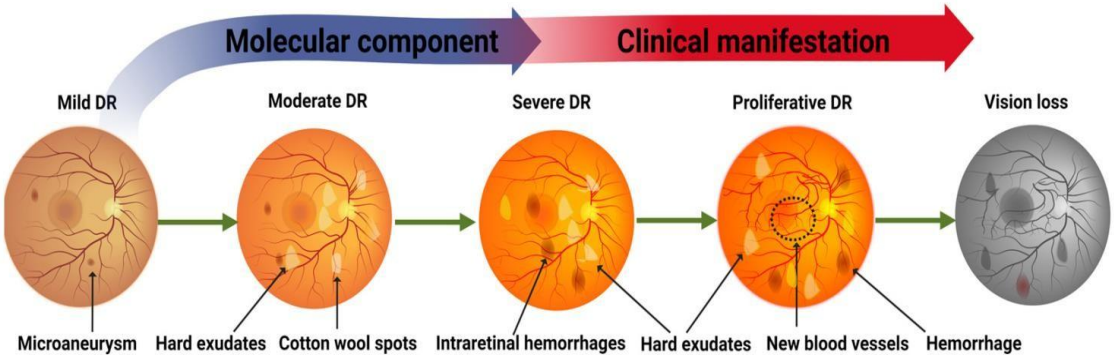

Fig no; 1 Incidence of diabetic retinopathy

2. Classification

It is known that a set of factors contribute to the clinical characteristics of diabetic retinopathy (3). All of these components show up in varying forms according on how diabetic retinopathy develops and progresses. According to the literature review, the following classification is most appropriate for diabetic retinopathy.

A. Background or non-proliferative diabetic retinopathy

B. Pre proliferative diabetic retinopathy Macular degeneration:

i. Ischemic

ii. Cystoid

iii. Focal

C. Proliferative diabetic retinopathy

D. Advanced diabetic ocular disease:

i. Vitreous haemorrhage

ii. Detachment traction

iii. Opaque membrane formation

iv. Neo vascular glaucoma {NVG}

It is also regarded as the diabetic patient's most well-documented micro vascular hazard. Aside from these effects, insufficient analysis or delayed therapeutic intervention may cause vision impairment, partial blindness, and ocular problems. (4) Moreover, hard exudates accumulate at the macula's core due to both vascular leakage and prolonged DME. Depending on when the problems start and how long they last, these designs can be serious or severe11. Biomolecules include lipoprotein-PLA2, secretory phospholipase A2 IIA, and pro-inflammatory factors are responsible for the vascular edema.

3. Background diabetic retinopathy:

Background retinopathy is characterized by capillary blockage and lessoning of micro aneurysms, haemorrhages, and exudates. The inner form and inner nuclear layer cytokines, along with the region temporally adjacent to the macular centre, are the most frequently reported alterations in the diabetic fundus (5,6).

4. Proliferative Diabetic Retinopathy

Once there is arterial and artery participation within the capillaries, it is analysed. The following characteristicsof the medical imaging for this stage of diabetic retinopathy:

4:1: -Proliferative Diabetic Retinopathy

The most mutually dangerous stage of diabetic retinopathy is represented by such alterations, which occur in 5%–6% of cases. It commonly manifests in type I diabetes and happens betweenthe ages of 11 and 18 after developing diabetes12. Proliferative diabetic retinopathy was shown to occur at a rate of 67% in type I diabetics within 35 years of the onset of the disease, but it was 15.5% in type II diabetics with adoration of 15 years or more. Hypoxia of the tissue is caused by micro vascular disease in the retina with capillary closure. In order to provide better oxygenation of the retinal soft tissue, hypoxia causes proliferative factors to be released,

which in turn stimulates the formation of new blood vessels (7).

These budding blood vessels have the potential to bleed and cause food to leak into the vitreous.It appears that the development of new vessels follows three steps. Protease-induced extracellular matrix degradation occurs first, and endothelial cell migration occurs at the tip of the newlyformed capillary. Cell division behind the apex, which is crucial to the new vessel elongation, isthe last stage (8).

4:2: -Non-proliferative Diabetic Retinopathy

At this stage, the retina develops internal lesions that include micro aneurysms, small "splinter" and "dot and blot" haemorrhages, intraregional micro vascular abnormalities (IRMA), and "cotton wool" acne. The degreeto which these grazes are present defines the classification of the NPDR as "mild," "moderate," "severe," or "very severe."

4:2.1Mild Non-Proliferative Diabetic Retinopathy

At least one micro aneurysm, and also dot, blots or flame-shaped haemorrhages in all four fundus quadrants**4:2.2 Moderate Non-Proliferative Diabetic Retinopathy**

Intraregional micro aneurysms and dot and blot haemorrhages of better sternness, in one to three quadrants. Cotton wool spots, venous calibre variations with venous beading, and intraregional micro vascular abnormalities are current but slight (9,10).

4:2.3 Very Severe Non-Proliferative Diabetic Retinopathy

Two or more of the criteria for severe non-proliferative diabetic retinopathy, but without any proliferative diabetic retinopathy.

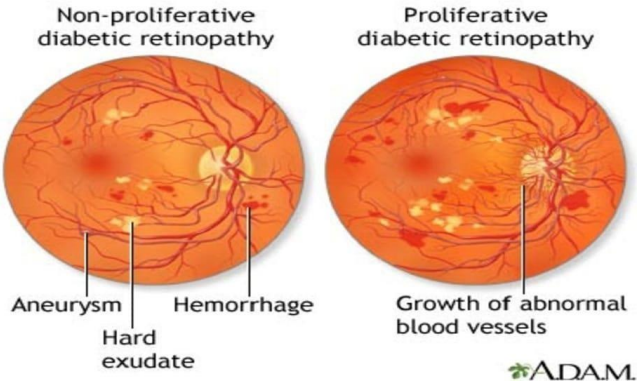

Fig no;2 Non proliferative and proliferative

4. Pre-Proliferative Diabetic Retinopathy

As soon as there is an arterial and artery contribution within the capillaries, a diagnosis is made. The followingcharacteristics describe the scientific picture for this stage of diabetic retinopathy:

4.1 : - Widespread hemorrhages: These are characteristic hallmarks of wide-ranging ischemia.

4.2 : - Cotton wool exudates: These designate micro infarcts in the nerve fibre film secondary to alteration ofthe axon plasmin movement (11). At the start these lacerations appear greyish in colour, later changing to white colour giving the arrival of cotton wool. When these grazes are present, they are an indication of a quickly failing retinopathy.

4.3 : - Intraregional micro vascular abnormalities: These any indicate an intraregional propagation or a dilation of capillaries due to enlarged hydrostatic pressure related with capillary leakage.

4.4 : - Focal diabetic Maculopathy: The key feature is focal dilatation of the vessels and the escape from the dilated abnormal capillaries with evidence of exudates. It is the most mutual type, and the visual loss is permanent once the leakage reaches the macular15. The recognition of the prominence of vascular endothelialgrowth factor (VEGF) in both proliferative retinopathy and macular enema has led to diagnosis with intra virtual injection of agents which block the effects of VEGF (12).

5. Advanced haemorrhages

The haemorrhages resemble boats, featuring a horizontal fluid level and a curving lowest. They cause retinaldetachment or significantly alter vision, obstructing broadcasts. The fibro vascular tissue will also be lifted off if grouping develops in the posterior hyaloids sheath because it has connected to this membrane. Condensation of the Diabetic Retinopathy

5.1 : Vitreous

vitreous strands and shrinking of the central vitreous will result from haemorrhaging into the vitreous along with the breakdown of collagen organization in the vitreous gel.

5.2 : - Detachment

There will be haemorrhages and eventually retinal detachment if vitreous shrinkage increases and vitriol retinal traction becomes extreme. Even though a haemorrhage that breaks into the vitreous gel will likely stay for a long time, it may be quickly absorbed if it is in the retina virtual space.

5.3 : - Opaque membrane

Advanced instances need vasectomy to remove membranes, fibrin, and blood; Ringer's solution is then used to replace the lost materials to restore clarity and shape. It aids in the retreat and cessation of bleeding in the newly formed vessels. Only eyes with vitreous opacities can benefit from this surgical procedure, and the likelihood of a favourable outcome is generally decreased in situations with prior retinal ischemia, degeneration, or fibrosis. Nevertheless, between 68% and 80% of cases show improvement in vision followingvasectomy.

5.4 : - Neo vascular glaucoma

It falls under the category of secondary glaucoma. There have been several names for it, including diabetic haemorrhagic glaucoma, thrombotic glaucoma, congestive glaucoma, and robotic glaucoma. A delayed diagnosis or inadequate treatment may cause total blindness or even the globe's eventual loss. It is essential to diagnose the illness quickly and to treat it aggressively and gradually. Treating the increased intraocular pressure as well as the underlying cause of the disease is crucial to controlling NVG.

6. Risk Factors of Diabetic Retinopathy

The length of the condition, the presence of neuropathy, diabetic nephropathy, foot ulcers, amputations, hypertension, serum cholesterol and triglyceride levels, fasting blood glucose, HbA1c, and the patient's age are risk factors for diabetic retinopathy18. The development of hypertension and an inappropriate glycaemia regulator (high HbA1c) are the main risk factors for the progression of diabetic retinopathy.

Throughout the clinical trial, a thorough diagnosis of hyperglycaemia decreased the risk of developing DR by76% and 54%, respectively. Between 1977 and 1997, the UKPDS examined 5102 people with type 2 diabetesand discovered that, in contrast to standard medication, appropriate blood glucose management reduced DR by 25%.

6.1 : -Genetic Risk Factors of Retinopathy

DR is categorized as a polygenic, genetically inherited illness based on many twin studies, with researchers finding a clear familial clustering. According to initial estimates, DR and PDR are 27% and 52% heritable, respectively19. According to studies, a character's risk of developing DR was raised by about two to three times if their family had a history of the disease. A close relationship was found between DR and the intragenic single-nucleotide polymorphism rs476141, according to a genome-wide meta-analysis.

7. Pathophysiology

The ploy route, increased glucose flow, AGE-product addition, edema, and protein kinase C (hexamine pathway) activation are among the biochemical mechanisms linked tohyperglycaemia-induced vascular damage.

Hyperglycaemia causes superoxide to build up in the mitochondria, which causes oxidative stress. Oxidative stress is a stressor that connects all of these metabolic pathways. Multiple early clinical characteristics of DR, including as a thicker basement sheath, parricide apoptosis, andmitochondrial dysfunction, are caused by oxidative stress and ultimately lead to BRB breakdown.

One of the earliest clinically recognized diseases of DR, intravascular immune response leucocytosis, grow thicker in the retina due to BRB deficiency. It results in the adhesion of white blood cells (WBCs) to the blood vessel endothelium, which affects capillary function and vascular leakage.

7.1 : -Pathological Targets

One of the key players in the pathophysiology of retinal micro vascular injury, which suggests diabetic retinopathy, is thought to be hyperglycaemia. Among the metabolic pathways taken into account during hyperglycaemia-induced vascular damage include the build-up of advanced gyration end products (AGEs), the ploy and hex examine route, and the protein kinase C (PKC) pathway (13).

Furthermore, in diabetic individuals, the first responses to hyperglycaemia from retinal blood vessels are alterations in blood flow and blood vessel dilatation. Additional barriers to the early stages of diabetic reticulum development include endothelial cell death, basement membrane condensing, and parricides loss brought on by elevated glucose concentration, which results in capillary wall pouching and the production ofmicro aneurysms.

Subsequently, a substantial loss of endothelial cells and parricides together impairs the BRB and causes ischemia and capillary blockage. Hypoxia-inducible factor 1 (HIF-1) is activated in response to retinal ischemia/hypoxia, and antigenic factors like vascular endothelial growth factor (VEGF) and angiopoietins (Ang-1, Ang-2) are upregulated.

7.2 : - DR—an Inflammatory Disease

At first, DR was thought to be a micro vascular only condition. Presently, persistent, low-grade swelling is a major factor in the pathophysiology of diabetic retinal disease (DR), which results in changes in the microcirculation of the retina. This swelling has been observed at various stages of diabetic DR in both diabeticanimal models and diabetic patients' retinas. This pathology affects the retina's neuronal and vascular components. In addition, it exhibits some characteristics of chronic inflammatory infections, such as the infiltration of inflammatory cells, the expression of various effectors, including cytokines that damage the retina, enema, neovascularization, or tissue destruction.

8.3. Role of Inflammatory Chemokine’s in DR

It has been shown that chemokine’s play a role in the ethology of DR. Recent research has indicated that individuals with

diabetes have higher levels of macrophage inflammatory protein-1 alpha (MIP-1a) and monocyte chemotactic protein-1 (MCP-1). Furthermore, diabetic individuals with DR had elevated levels of various inflammatory cytokines, including TNF-α, IL-6, IL-8, and interleukin 1 (IL-1). Furthermore, there is a connection between inflammation in DR patients and the high concentrations of growth factors and inflammatory cytokines in their eye fluids. Increased vascular permeability, parricide loss, and the development of micro aneurysms are all brought on by DR.

Reactive oxygen species (ROS) and free electrons are elevated during the inflammatory process in DR, which results in the release of harmful lipids, proteins, and oxidized mitochondrial DNA into the cytosol. This is because the retina uses large amounts of glucose and oxygen to generate energy through the mitochondrial electron transport chain (ETC)

8. Screening of Diabetic Retinopathy

When diabetic retinopathy is treated effectively in its early stages, vision is not compromised. The primary diagnosis of asymptomatic illness by fundus examination is necessary to prevent blindness due to retinopathy. Ophthalmoscopy, which uses a slit lamp and a contact lens or 78D lens, or retinal photography, which uses conservative film or a digital camera, are two methods for examining the fundus. Fundus photography has been demonstrated to be the most accurate method of detecting retinopathy. An ophthalmologist can quickly check a large number of eyes thanks to the pictures.

At first, digital fundus photography is exclusive, but because it doesn't require film or developing the images, it has very low operating expenses. The pictures are immediately accessible. Digital images are not as excellent as traditional film in this regard. They do, however, work rather well for retinal screening. There aren't enough ophthalmologists

in the majority of developing nations for each diabetic to receive a yearly examination. The diabetic doctor, an optometrist, or an ophthalmic assistant may check the fundus if retinal imaging is not an option.

Diabetic retinopathy screening is only useful if a high coverage rate (at least 80% of known diabetics) is reached. It is imperative that diabetes patients have the most convenient screening procedure possible. It oughtto be free as well (14).

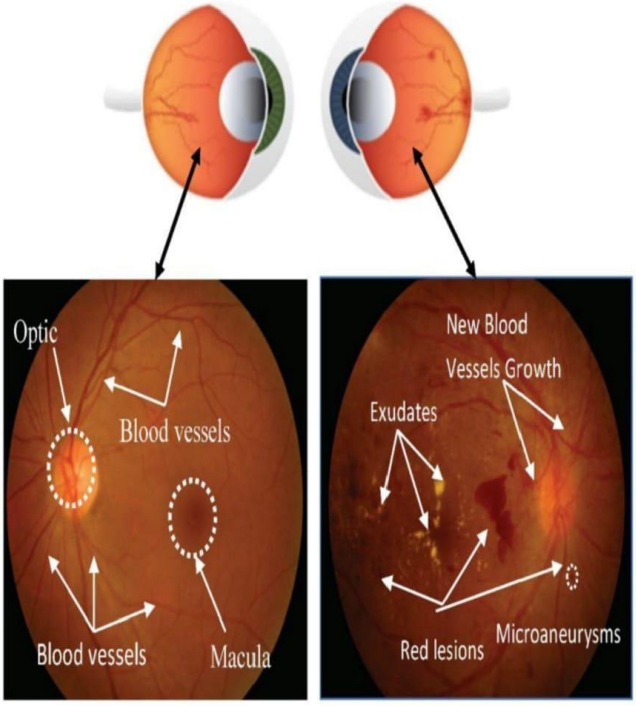

Fig no; 3 Screening of Diabetic Retinopathy

9.1. Insulin dependent

- Expanded fundus examination every year commencement 5 years after analysis, from puberty onwards.
- Examinations more frequently once diabetic retinopathy is diagnosed.

9.2: -Non-Insulin Dependent

- Dilated fundus examination every year once diabetes analysed.
- Analysis more repeatedly once diabetic retinopathy diagnosed. Diabetics are at significantly increased risk of cataract.

Every diabetic should get an annual evaluation of their visual acuity. Individuals who have vision lessthan 6/18 in one or both eyes should be evaluated thoroughly since they may be suffering from glaucoma, cataracts, or refractive errors.

9. Diabetic Maculopathy

Diabetic Maculopathy, which can cause vision impairment, is defined as diabetic retinopathy located in and around the

macula. The aforementioned diabetic retinal abnormalities are all caused by disease occurring at the micro vascular level of the retina. This includes capillary dilatation, capillary wall breakdown, and capillary closure, which result in micro infarcts and hypoxia.

Based on the presence or absence of "clinically significant macular oedema," patients receiving macular focused laser therapy were categorized by the Early Treatment of Diabetic Retinopathy Study (ETDRS) as follows:

- Retinal condensing at or within 500μ (one third of the diameter of the optic disc) at the centre of the macula.
- Hard exudates at or within 500μ of the midpoint of the macula, if there is stiffening of the adjacent retina.
- An area of retinal stiffening greater than one optic disc area in size, at least a part of which is within onedisc width of the centre of the macula.

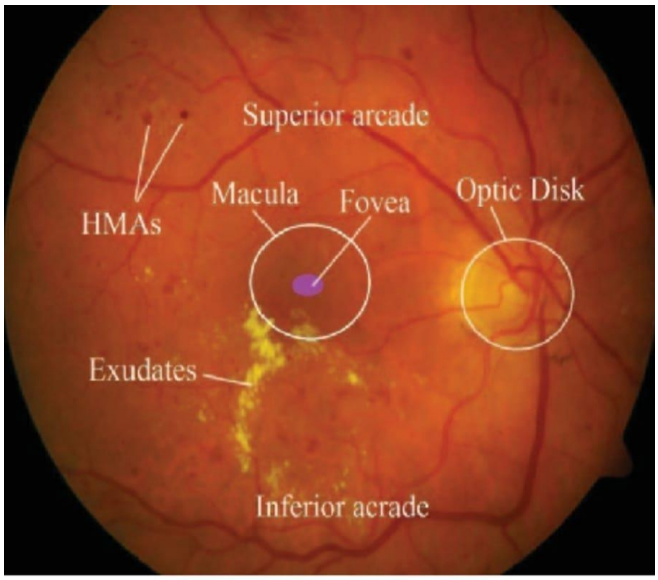

Fig no; 4. A sample fundus images

10. Treatment of Diabetic Retinopathy

❖ Age

There has been evidence of a positive association between the patient's age at the onset of diabetes and diabeticretinopathy. Proliferative retinopathy appears to be associated with a higher frequency of visual impairment in younger onset patients; older onset patients are more likely to experience maculopathy.

❖ Duration

The most significant correlation between proliferative diabetic retinopathy and retinopathy is diabetic duration. Evidence of retinopathy is common after ten years, although it is rarely observed in the first five to ten years. It was reported that the occurrence rate was 50%, and in young people who had had diabetes for 30years, this number was probably closer to 90%. The frequency of retinopathy generally tends to rise after 15–20 years of prolonged exposure;

But it reaches a threshold of twenty to thirty years. Up to 15–20 years of age, there is a typical trend toward an increase in the prevalence of retinopathy; nevertheless, it reaches a peak between 20 and 30 years. This extended duration may be caused by an expanding human population with more severe retinopathy. Accordingto reports, the prevalence rate of total retinopathy was 97.5% over a 15-year period, with a 40% risk of proliferative lesions after retina.

❖ Diabetic Control

As was already indicated, using an effective glycaemic controller lowers the likelihood that diabetic retinopathy will develop and worsen. It is important to stress the value of effective control. Patients on insulin therapy seem to experience diabetic retinopathy more frequently and with greater severity than those whose diabetes is managed solely by diet or medication. Patients on insulin were seen to have a higher developed level of fluorescein saturation through the blood retinal barrier compared to those on pills or diet.

❖ Blood pressure

Due to its relationship to nephropathy, the relationship between retinopathy and high blood pressure is complicated. The typical association between aging and hypertension adds even more complexity. It has been shown that retinopathy and hypertension share a major commonality. They found that diabetics with systolicblood pressure more than 145 mmHg had a

twice as high incidence of exudates compared to those with lowervalues.

❖ Pregnancy

Pregnancy is generally thought to have a negative impact on retinopathy. Pregnant IDDM women who have had diabetes for 15 years or more have been shown to have a 63%–82% risk of developing any type of retinopathy and an 18%–20% likelihood of developing proliferative retinopathy. 40% of the 234 pregnant diabetics at the Right Hospitalet in Oslo who were evaluated between 1970 and 1977showed signs of diabetic retinopathy.

Retinopathy seldom develops in pregnant women without it; however, some may show signs of background alterations. Individuals with pre-existing retinopathy have a 50% probability of their condition getting worse in the second trimester and maybe getting better in the late third trimester. A small percentage may also develop new blood vessels, and in 50% of individuals with existing new blood vessels, their retinopathy worsens30.

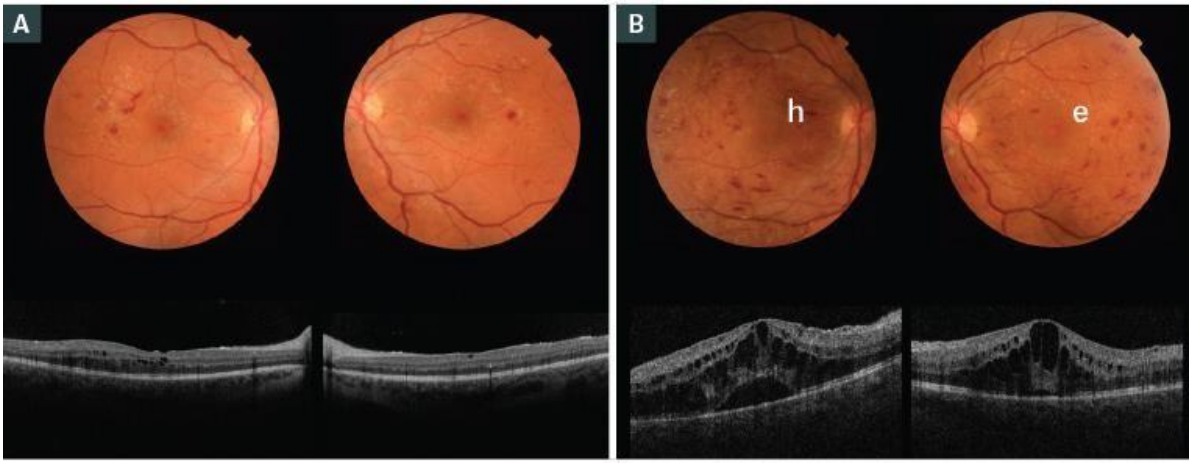

Fig no; 5. Ophthalmic Associations in pregnancy

❖ Prevention of Blindness due to Diabetic Retinopathy

The prevention of diabetic retinopathy-related blindness necessitates knowledge of the prevalence of the condition in the general population, identification of high-risk subgroups within the diabetic population, and the use of low-cost screening techniques like fundus photography or ophthalmoscopy. Photo coagulators are necessary for conduct facility centres. Remember that patients with diabetes in some groups may experience blindness or visual impairment from other causes, such as cataract or refractive error.

10.1 : -Diagnosis &Management

Identification In addition to their friends, family, and medical professionals, diabetic individuals need to be made aware of the importance of routine eye exams in order to detect diabetic retinopathy (DR) early on. Various diagnostic methods that could be applied for identification, examination, and diagnosis. Direct and indirect ophthalmoscopy, stereoscopic digital and fundus photography, my drastic or non-mydriaticdigital colour or monochromatic single-field photography, optic coherence tomography (OCT), optic coherence tomography-angiography (OCT-A), and fluorescein angiography are among the diagnostic techniques used to measure DR and treatment efficacy.

10.2 : -Therapeutic Concepts

10.2.1. Prevention

Clinical usage of metabolic regulators is recommended to prevent the escalation or advancement of DR. Controlling hyperglycaemia is the most important factor in preventing the onset of diabetic ketoacidosis (DR).Trials involving complex patients with type 1 and type 2 diabetes have demonstrated that strict management of low haemoglobin levels and rigorous glycaemic control can reduce the risk of DR by as much as20%. Statins and/or fibrates are used to patients with type 2 diabetes in order to significantly lower the risk and progression of DR. Additionally, the value of DR showing and surveillance is based on early identification, which is why yearly screenings for retinopathy are provided to diabetics. It is mandatory to take digital fundusphotos for all diabetic screening programs.

10.2.2. Specific Therapeutic Options

To complement the best medical management of blood pressure, serum cholesterol, and glucose levels, a number of intraocular interventions have become established treatments for diabetic retinopathy. By limiting the inflammatory pathway, ophthalmological treatment lowers vascular permeability, aids in the BRB's collapse, inhibits leukostasis, blocks the transcription and translation of the VEGF gene, and,ultimately, lowers the risk of vision loss in eyes with vision-threatening conditions. Anti-VEGF medications,corticosteroids, and laser therapy are part of the ocular therapy for diabetic retinopathy and Maculopathy, according to apparatuses.

10.2.3. DME Treatment

It is still unknown exactly what equipment is used in laser photocoagulation for DME treatment. It is proposedthat it combines

the destruction of ischemic areas of the retina with the blockage of leaky arteries, particularlymicro aneurysms. It lessens the release of cytokines and pro-antigenic substances while also increasing oxygenation in the areas close to the treated ones

Following laser treatment, problems include sub retinal fibrosis, growth of the laser scar, diminished visual field sensitivity, colour degradation, and night vision and sensitivity. Over the past few years, DME treatment paradigms have changed. Anti-VEGF intra viral injections have nearly eliminated the DME laser management.

10.2.4. Surgical Treatment

The improvement of vitreous haemorrhages that do not resolve and slight retinal detachment caused by fibro vascular proliferation linking the macular region have led to the need for surgical procedures like vasectomy, pealing of epic-retinal membranes, undo-laser photocoagulation during surgery, and vitreous replacement with silicone oil or per fluorocarbons. Because of the less intrusive and safer nature of these techniques, patients with PDR may benefit from an early vasectomy in cases where the macula is reluctant to heal because of fibro vascular tissue covering it. Moreover, it has been demonstrated that these procedures are safer with anti- VEGF pre-treatment. Therefore, the early intervention has prolonged the signs of vasectomy for PDR treatment.

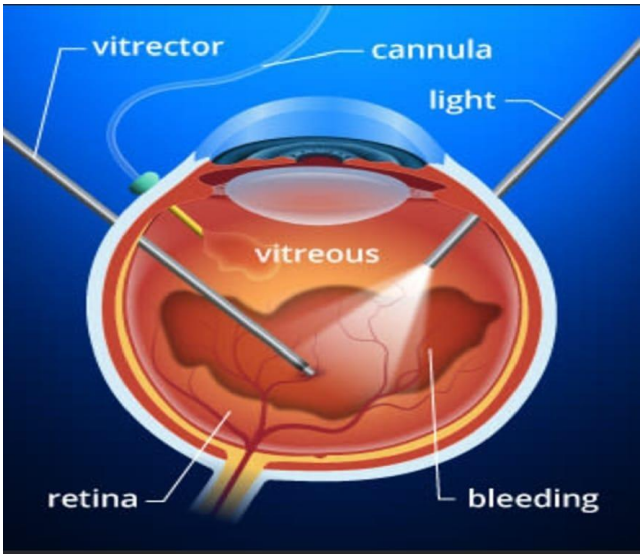

Fig no; 15 Surgical treatment

11. 2.6. Vasectomy

This method can be applied to PDR cases of mutual minor and rhegmatogenous retinal detachment, fractional retinal detachment, and long-standing vitreous haemorrhage (when visualization of the condition of the afterpole is too difficult).

11. 2.7. Cryotherapy

Cryotherapy may be used in place of laser photocoagulation in PDR when it is prohibited by an opaque medium, such as in cases of cataracts or vitreous haemorrhage. The Diabetes Regulator and Difficulties Trialdiscovered that patients with IDDM who underwent strict glucose control saw a reduction in the onset and advancement of diabetic retinopathy. Accepting that NIDDM operates on the same principles might make sense.

11.2.8. Pan Retinal Photocoagulation

To lower the oxygen demand of the hypoxic retina in diabetic retinopathy, photocoagulate the later 45°–60°of the retina, away from the vascular arcades of the macula, with graded burns. This converts the hypoxic zones of the retina into anoxic zones, which lowers the release of V a s o - proliferative factors (Figure 5). Asa result, PRPC stops new vasculature from growing and may cause old, original vessels on the retina or optic disc to revert (14).

Conclusion

In highly developed nations, diabetic retinopathy is a major cause of vision loss in patients of working age and a major contributor to blindness globally. The number of patients affected by this issue steadily rises.Our challenges include providing this patient with proper care, facilitating an early and simple diagnosis process, and improving results. Numerous investigations have provided evidence in Favour of the notion thatneuron degeneration is an early occurrence in the diabetic retina and that diabetic retinopathy is a neurovascular disease. This study summarized current treatments for DR and DME, including laser, anti-VEGF, steroid,and surgery. The suggested actions for PDR and DME are displayed in Figures 2 and 3. PRP is typically used to treat individuals with severe NPDR; however, TRP may also be available choice if the NPA can be evaluated in FA. PRP or anti-VEGF therapy may be necessary for PDRpatients, depending on their clinical and socioeconomic circumstances. Depending on whether the fovea is involved in the enema, there are

several approaches to treating DME. This full prescription is essential for preventing and delaying the development of DR. Furthermore, the therapies that are still available address vascular problems rather than cerebral ones. Thus,it is imperative to create neuron protection or preventive therapy as soon as possible for the early stages of DR.

Author contributions

All authors are contributed equally.

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Declaration of Competing Interest

The authors have no conflicts of interest to declare.

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