A REVIEW ON DIABETIC RETINOPATHY

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ABSTRACT
Diabetes mellitus is a chronic disease which is difficult to cure. Management concentrates on keeping blood sugar levels as close to normal as possible without presenting undue patient danger. This can usually be with close dietary management, exercise and use of appropriate medications. Insulin can is used only in the case of type 1 diabetes mellitus. Oral medications may be used in the case of type 2 diabetes, as well as insulin. This review focused on progressive of retinopathy and managements of Retinopathy. Retinopathy is a general term that refers to some form of non-inflammatory damage to the retina of the eye. Retinopathy is an ocular manifestation of systemic disease. This review concluded many types of retinopathy are progressive and may result in blindness or severe vision loss or impairment, particularly if the macula becomes affected.

Keywords: Diabetes mellitus, Retinopathy, Managements of Retinopathy.

INTRODUCTION
Diabetes is a group of metabolic diseases in which a person has high blood sugar, either because the body does not produce enough insulin, or because cells do not respond to the insulin that is produced. This high blood sugar produces the classical symptoms of polyuria (frequent urination), polydipsia (increased thirst) and polyphagia (increased hunger). The term diabetes usually refers to diabetes mellitus, which roughly translates to excessive sweet urine known as glycosuria. The most common of these is diabetes insipidus, in which large amounts of urine are produced (polyuria).

There are three main types of diabetes:
- Type 1 diabetes: Results from the body's failure to produce insulin, and presently requires the person to inject insulin. It also referred to as insulin-dependent diabetes mellitus, IDDM for short, and juvenile diabetes.
- Type 2 diabetes: Results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency.
- Gestational diabetes: When pregnant women, who have never had diabetes before, have a high blood glucose level during pregnancy. It may precede development of type 2 DM.

Diabetes without proper treatments can cause many complications. Acute complications include hypoglycemia, diabetic ketoacidosis, or nonketotic hyperosmolar coma. Serious long-term complications include cardiovascular disease, chronic renal failure and retinal damage. Adequate treatment of diabetes is thus important, as well as blood pressure control and lifestyle factors such as smoking cessation and maintaining a healthy body weight [1].
CAUSES

The cause of diabetes depends on the type. Type 2 diabetes is due primarily to lifestyle factors and genetics. Type 1 diabetes is also partly inherited and then triggered by certain infections, with some evidence pointing at Coxsackie B4 virus. There is a genetic element in individual susceptibility to some of these triggers which has been traced to particular HLA. However, even in those who have inherited the susceptibility, type 1 diabetes mellitus seems to require an environmental trigger.

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Retinopathy

Retinopathy is a general term that refers to some form of non-inflammatory damage to the retina of the eye. Retinopathy is an ocular manifestation of systemic disease. Many types of retinopathy are progressive and may result in blindness or severe vision loss or impairment, particularly if the macula becomes affected.

Causes of retinopathy are diabetes - diabetic retinopathy, arterial hypertension - hypertensive retinopathy, prematurity of the newborn - retinopathy of prematurity, sickle cell disease, ciliopathy, direct sunlight exposure - solar retinopathy and retinal vein or artery occlusion [2].

Diabetic Retinopathy

Diabetic retinopathy is the leading cause of preventable blindness. Diabetic retinopathy is retinopathy (damage to the retina) caused by complications of diabetes mellitus, which can eventually lead to blindness. It is an ocular manifestation of systemic disease which affects up to 80% of all patients who have had diabetes for 10 years or more.

Diabetic retinopathy is the most frequent cause of new cases of blindness among adults aged 20–74 years. During the first two decades of disease, nearly all patients with type 1 diabetes and >60% of patients with type 2 diabetes have retinopathy. In the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR), 3.6% of younger-onset patients (type 1 diabetes) and 1.6% of older-onset patients (type 2 diabetes) were legally blind. In the younger-onset group, 86% of blindness was attributable to diabetic retinopathy. In the older-onset group, in which other eye diseases were common, one-third of the cases of legal blindness were due to diabetic retinopathy.

EPIDEMIOLOGY

In 2000, according to the World Health Organization, at least 171 million people worldwide suffer from diabetes, or 2.8% of the population. Its incidence is increasing rapidly, and it is estimated that by 2030, this number will almost double. Diabetes mellitus occurs throughout the world, but is more common (especially type 2) in the more developed countries. The greatest increase in prevalence is, however, expected to occur in Asia and Africa, where most patients will probably be found by 2030. The increase in incidence of diabetes in developing countries follows the trend of urbanization and lifestyle changes, perhaps most importantly a "Western-style" diet. This has suggested an environmental (i.e., dietary) effect, but there is little understanding of the mechanism(s) at present, though there is much speculation, some of it most compellingly presented [3].

For at least 20 years, diabetes rates in North America have been increasing substantially. In 2008 there were about 24 million people with diabetes in the United States alone, from those 5.7 million people remain undiagnosed. Other 57 million people are estimated to have pre-diabetes.

The Centers for Disease Control has termed the change an epidemic. The National Diabetes Information Clearinghouse estimates that diabetes costs $132 billion in the United States alone every year. About 5%–10% of diabetes cases in North America are type 1, with the rest being type 2. The fraction of type 1 in other parts of the world differs. Most of this difference is not currently understood. The American Diabetes Association cite the 2003 assessment of the National Center for Chronic Disease Prevention and Health Promotion (Centers for Disease Control and Prevention) that 1 in 3 Americans born after 2000 will develop diabetes in their lifetime.

According to the American Diabetes Association, approximately 18.3% of Americans age 60 and older have diabetes. Diabetes mellitus prevalence increases with age, and the numbers of older persons with diabetes are expected to grow as the elderly population increases in number. The National Health and Nutrition Examination Survey demonstrated that, in the population over 65 years old, 18% to 20% have diabetes, with 40% having either diabetes or its precursor form of impaired glucose tolerance.

Indigenous populations in first world countries have a higher prevalence and increasing incidence of diabetes than their corresponding non-indigenous populations. In Australia the age-standardised prevalence of self-reported diabetes in Indigenous Australians is almost 4 times that of non-indigenous Australians. Preventative community health programs such as Sugar Man (diabetes education) are showing some success in tackling this problem.
CAUSES

Every person who has diabetes, whether it's type 1 or type 2, is at risk of developing retinopathy. The disease is more prevalent and tends to be more severe, though, among those with type 1 disease. As with all chronic disorders, some risk factors lie outside your control. Others can be managed with lifestyle modifications and medical intervention. The important causes of diabetic retinopathy can be classified into two. They are non-modifiable factors and controllable or partially controllable factors.

Non-Modifiable factors

- Genetic variation. The abnormal development of new blood vessels that occurs in diabetic retinopathy is regulated by a protein called vascular endothelial growth factor A (VEGF-A). This protein carries out orders from a gene called, fittingly enough, the VEGF-A gene. Scientists have discovered a variation in the sequence of this gene that's associated with the development of severe diabetic retinopathy.

- Advancing age. Diabetic retinopathy is rare among children age 10 and younger. About 10 percent of teens with diabetes ages 15 to 19 have diabetic retinopathy. The proportion rises from 10 percent to 40 percent between ages 20 and 29. By age 30, about 60 percent of people with diabetes have diabetic retinopathy, and by age 45 the figure rises to 70 percent.

- Ethnicity. Diabetic retinopathy is more common among some ethnic groups than others. For example, African Americans and Latino Americans with type 2 diabetes have a greater prevalence and severity of the disease than non-Hispanic whites. Researchers have been unable to explain why Latino Americans are disproportionately affected. African Americans, though, tend to have more risk factors for diabetes and thus may develop diabetic retinopathy as a complication of the disease.

- Duration of diabetic disease and age at diagnosis. The longer you've had diabetes, the more likely you are to develop diabetic retinopathy.

- Severity of diabetic disease. The more severe your diabetes is, the more likely you are to have diabetic retinopathy and the more likely it is to threaten your vision.

- Gender. Men with diabetes are at higher risk for developing diabetic retinopathy and for having more severe retinal disease. Researchers continue to seek an explanation for this puzzling gender difference [4].

Controllable or Partially Controllable factors

- Poorly controlled blood sugar. Research indicates a direct correlation between the degree of blood sugar control and the likelihood of onset or progression of diabetic retinopathy.

- High blood pressure. If you've had diabetes for 10 years or longer and have high blood pressure, you're at risk for diabetic retinopathy that's more destructive and progresses more quickly.

- Obesity. Body mass index (BMI) is a measure of body weight adjusted for height. People with diabetes who are in the top quartile (that is, the highest 25 percent) of BMI compared with their peers are at twice the risk of developing retinopathy.

- Illness. People who have other health conditions, such as high blood pressure, heart disease, diabetic kidney disease, and high cholesterol, are at increased risk of developing diabetic retinopathy.

Types of Diabetic Retinopathy

There are two types of diabetic retinopathy:

- Nonproliferative diabetic retinopathy (NPDR)
  Early stage diabetic retinopathy

- Proliferative diabetic retinopathy (PDR)
  Later stage diabetic retinopathy

Nonproliferative diabetic retinopathy

Nonproliferative diabetic retinopathy also called as background diabetic retinopathy. This is the earliest stage of diabetic retinopathy. Damaged blood vessels fluid from blood plasma and small amounts of red blood cells will drain into the retina. Cholesterol, triglycerides, and proteins from blood may leak into the retina forming hard exudates.

Central vision is affected by any of the following:

- Cholesterol and protein deposits in the central retina (macula)
- Microaneurysms (small bulges in blood vessels of the retina may leak)
- Retinal hemorrhages (tiny spots of blood that may form in the central macula)
- Macular edema (swelling/thickening of macula)
- Macular ischemia (closing of small blood vessels or capillaries).
Proliferative diabetic retinopathy (PDR)

Proliferative diabetic retinopathy is the later stage of diabetic retinopathy. Abnormal blood vessels begin to grow on surface of retina or optic nerve (neovascularization) and they do not provide retina with normal blood flow. As these vessels grow a scar-like tissue (fibrovascular proliferation) develops which can lead to retinal detachment.

Vision is affected when any of the following:
- Vitreous hemorrhage (new abnormal blood vessels bleed into the vitreous gel, preventing light rays from reaching the retina).
- Traction retinal detachment (new abnormal blood vessels mature into scar-like membranes and tug on the retina potentially leading to retina detachment).
- Neovascular glaucoma (neovascularization occurs in the iris and nearby structures causing pressure to build up in the eye which may damage the optic nerve).

SYMPTOMS
Since symptoms during the early stages of diabetic retinopathy may be absent or subtle, everyone with diabetes should have a comprehensive dilated eye examination at least once a year. Of course, any flecks, spots, or blurs in your visual field should prompt an immediate visit to your vision care provider. Symptoms may not be noticeable but can including the following blurry vision, halos or flashing lights, double vision, dark spots or floaters, pain or a sensation of pressure, diminished peripheral (side) vision, poor night vision.

Early signs of diabetic retinopathy include leaking blood vessels, swelling (edema), fatty deposits, and damaged nerve tissue. But only your vision care specialist can diagnose those signs. Your eye doctor will use several tests to determine whether you have diabetic retinopathy and, if so, how to proceed based on the stage of the disease.

Physiology of eye
The Eyeball
To understand vision, first we need to know about the anatomy of the eye. Think of the human eye, with its remarkable anatomical design, as a miniature theater in which the process of vision takes place.

The eyeball, or globe, is spherical and measures about 2.5 cm across. It sits in a cone-shaped pocket within the skull called the orbital cavity, or “eye socket.” This bony orbit is cushioned by fatty tissue and protects the eyeball from trauma, such as injuries that may occur during car accidents, falls, and assaults. The eyes are also shielded by the eyelids, which help keep the eyes moist.

Layers of the Eye
Each layer and internal structure of the human eye performs a distinct function. The eye has three primary layers:

i. Sclera, ii. Choroid, iii. Retina.

Outer layer: The sclera and cornea

The outer layer of the eye is made up largely of an envelope of tissue called the sclera—the part most of us call “the white of the eye.” The sclera extends from the cornea, at the front of the eye, to the optic nerve, at the back of the eye, providing a sheath of protection for the eye’s internal structures. The sclera and cornea are known as the fibrous tunic because together they form a snug-fitting jacket that cloaks the eyeball, concealing its amazing contents from would-be intruders, such as bacteria, viruses, and other pathogens. The transparent, dome-like window at the front of the eye is called the cornea. Similar in appearance to a contact lens, this membrane covers the iris—the pigmented, or colored, part of the eye—and the pupil, a round opening in the center of the iris. The pupil appears to be black because most of the light that passes through it is absorbed by pigmented cells in the tissue at the back of the eye [6]. Although the cornea may seem no more substantial than a sheet of plastic wrap, it’s actually a highly specialized, finely constructed network of cells and proteins. In addition to serving as a barrier against harmful microbes (germs) and dirt, the cornea has a second important function. It refracts, or bends, light waves to project them onto the lens of the eye, as we’ll explain in a moment. Because the cornea must remain perfectly clear in order to refract light properly, it’s the only tissue in the body that contains no blood vessels.

Middle layer: The choroid

Beneath the sclera lies a rich network of blood vessels called the choroid. It nourishes and supplies oxygen to the retina and other internal structures of the eye. The choroid layer measures approximately 1/2 mm (1/20 of an inch) thick, about the same as a stack of five sheets of paper. The choroid contains light-absorbing pigment cells that minimize reflections in the retina. These cells are responsible for the red-eye effect you sometimes see in snapshots taken with a flashbulb [7].

Inner layer: The retina

The retina lies below the choroid and makes up the innermost layer, or lining, of the eye. It contains light-sensitive rod cells and cone cells, known as photoreceptor cells because they capture electrical impulses and transmit images along the optic nerve to the brain. Each eye contains an astonishing number of these cone cells—about 6 million of them. They’re concentrated in the macula, the central part of the retina, especially in the fovea, a tiny pit at the center of the macula. This area is responsible for producing color and sharp-detail vision.
The rod cells populate the fringes of the retina. They’re even more numerous than the cone cells, numbering perhaps 100 million. Rod cells are active in darkness and in dim light, although your eyesight in these conditions is monochromatic (that is, not in color) and has poor resolution. However, you can still distinguish shape, size, and movement with some clarity—it’s the rod cells that keep you from tripping over your cocker spaniel when you get up in the middle of the night [8].

Mechanism of vision
To illustrate the process of vision, let’s follow an image as it travels through the eye.

Path of light through the eye
As you look at something, for instance—light rays pass through the eye to the retina. Several structures help to refract the light so that it focuses properly. The first of these structures, at the front of the eye, is the cornea. The next is a clear fluid called the aqueous humor, which circulates in the chamber behind the cornea and helps to nourish it and maintain its curvature.

As the light continues on its pathway, it passes through the pupil. The iris is a muscle that controls the size of the pupil. It’s made up of specialized fibers that are able to constrict the pupil to about 1 mm or dilate it to about 10 mm (1 mm is about the thickness of a U.S. dime). This narrowing and widening of the pupil, known as the light reflex, regulates the amount of light that enters the eye [9].

The light rays then reach the crystalline lens, a transparent, elastic disc that’s about the same size (1 cm in diameter) and shape (round and slightly flattened) as a piece of M&Ms candy. The lens is suspended by muscles that can pull taut or slacken like a drawstring pouch, changing the shape of the lens. This adjustment process, known as accommodation, sharpens visual clarity by helping to project light properly onto the retina.

After passing through the lens, light moves through a large chamber filled with clear, viscous matter called the vitreous humor. This gelatin-like substance makes up 80% of the mass of the eyeball and allows it to maintain its spherical shape. The retina marks the finale of light’s movement through the eye. There, visual images are registered by the rod and cone cells, which convert light into electrical impulses and transmit them to the brain via the optic nerve.

Accessory structure of vision
The process of vision is not complete until the brain interprets the chorus of electrical impulses transmitted by the retina. These signals are conducted through the optic nerve, an elegant bundle of more than a million long, slender retinal fibers. Together, these specialized cells, called ganglion cells, carry visual images from the retina to the brain.

Some ganglion cells are particularly sensitive to movement and contrast, whereas others are more responsive to shape and detail. Still others relay information about color. Depth perception—the ability to tell how far away objects are from you and from one another—occurs as the brain compares parallel signals transmitted by each eye in perfect synchrony.

In a specialized area called the visual cortex, the brain reconstitutes this fanfare of electrical impulses into fully formed images. It’s a process more exquisite and mysterious than the most brilliant symphony ever written. What’s more, this flawless performance is given by our eyes continuously every day, without a single rehearsal or false note. Now, that deserves a standing ovation [10].

PATHOGENESIS
Diabetic retinopathy is the result of microvascular retinal changes. Hyperglycemia-induced intramural pericyte death and thickening of the basement membrane lead to incompetence of the vascular walls. These damages change the formation of the blood-retinal barrier and also make the retinal blood vessels become more permeable.

The pericyte death is caused when hyperglycemia persistently activates protein kinase C- (PKC-, encoded by Prkcd) and p38 mitogen-activated protein kinase (MAPK) to increase the expression of a previously unknown target of PKC- signaling, Src homology-2 domain-containing phosphatase-1 (SHP-1), a protein tyrosine phosphatase. This signaling cascade leads to PDGF receptor-dephosphorylation and a reduction in downstream signaling from this receptor, resulting in pericyte apoptosis.

Small blood vessels such as those in the eye are especially vulnerable to poor blood sugar (blood glucose) control. An over accumulation of glucose and/or fructose damages the tiny blood vessels in the retina. During the initial stage, called nonproliferative diabetic retinopathy (NPDR), most people do not notice any change in their vision.

Some people develop a condition called macular edema. It occurs when the damaged blood vessels leak fluid and lipids onto the macula, the part of the retina that lets us see detail. The fluid makes the macula swell, which blurs vision [11].

Vision Loss
Uncontrolled blood glucose diminishes vision by three mechanisms:
Macular edema. Fluid can leak into the macula, the central part of the retina that provides sharp central vision and allows us to see fine detail. This fluid accumulation causes the macula to swell, thickening it and elevating it from its normal position. Macular edema can occur during any stage of diabetic retinopathy, but it is more likely to accompany proliferative retinopathy. In fact, about half of those with proliferative retinopathy also have macular edema.

Proliferation of blood vessels. Damaged blood vessels can’t adequately nourish the retina with oxygen and nutrients, so the retina begins to grow new vessels in an effort to meet its needs. These new vessels, however, are improperly formed and fragile. They can leak blood into the jelly-like vitreous humor inside the eyeball and into the retina itself. These hemorrhages cause vision loss and permanent low vision.

Retinal detachment. The new vessels that form in the vitreous humor are often accompanied by scar tissue that can contract, tugging on the margins of the retina and forcing it to detach.

DIAGNOSIS

Early signs of diabetic retinopathy include leaking blood vessels, swelling (edema), fatty deposits, and damaged nerve tissue. Your vision care specialist will use several tests to determine whether you have diabetic retinopathy and, if so, how to proceed based on the stage of the disease [12].

Schedule an eye exam

• If you were 30 years old or younger when your diabetes was first detected, you should have your first eye exam within five years after that diagnosis.
• If you were 30 years old or older, your first exam should be within a few months of the diabetes diagnosis.
• If you are pregnant, you should have an exam during the first trimester.
• If you already have experienced a high-risk condition, such as kidney failure or amputation related to diabetes, schedule an eye exam immediately.

Optical Coherence Tomography (OCT)

• OCT is a diagnostic tool for diseases of the macula (central retina) and optic nerve.
• OCT uses non-invasive coherent light (in which the electromagnetic waves maintain a fixed phase relationship with each other) to create a microscopic cross sectional image of the macula and optic nerve head capable of resolution to between 8 and 10 microns.
• In diabetics, OCT can “map” areas of macular edema (“swelling”) thus facilitating fluorescein angiography in guiding laser treatment of the macula. Repeat OCT studies may assist the ophthalmologist in assessing response to treatment and recurrence of macular edema [13].

Retinal imaging

The Retasure retinal imaging device is a new tool that allows you to be screened for diabetic retinopathy at a primary care physician's office. The device takes a quick, painless digital photo of the macula and optic nerve. This photo is transmitted via a secure network to an ophthalmologist at an accredited reading center. Results are available within a few days, and you'll be referred to an ophthalmologist for a complete examination and possible treatment if signs of retinopathy are discovered.

However, the Retasure network is limited to several hundred primary care offices that subscribe to the service and have the necessary camera and hardware on the premises; unless you live in a rural area, far from a vision care provider's office, the system offers little advantage except that your pupils don't have to be dilated. For that very reason, though, the system could fail to discover any diseased areas on the periphery (edges) of the retina.

Comprehensive Examination

After asking about your symptoms, family history, medications, surgical and medical history, and general health, eye doctor will give an eye examination. A comprehensive examination to detect diabetic retinopathy and macular edema consists of three parts:

Visual acuity testing. Most of us are familiar with the “big E” eye chart, so named for the large block letter at the top. This visual acuity chart measures how well you see at various distances. From a specified distance, usually 20 feet, your vision care provider will ask you to read aloud progressively smaller rows of capital letters.

Ophthalmoscopy and slit-lamp examination. Your vision care provider will assess the retina using a hand-held, microscope-like viewing instrument called an ophthalmoscope to look for the following signs of diabetic retinopathy:

• Leaking blood vessels
• Swelling and inflammation (edema)
• Fatty yellow deposits called exudates
• Cotton-wool spots, so named for their fuzzy white appearance; they indicate areas where tissue has died and become opaque

First, eye drops will be instilled to dilate your pupils, revealing the internal structures of the eye. After this examination, a slit lamp, which is a high-intensity light source combined with a low-power microscope, will be used to examine the frontal structures of the eye.
Tonometry. A tonometer is an instrument that measures intraocular pressures (the pressure of fluids inside the eye) after anesthetic (numbing) drops have been instilled. This test is performed to rule out glaucoma.

Supplemental Testing

Optical coherence tomography

Optical coherence tomography (OCT) is a safe, painless test that produces high-resolution images of three-dimensional cross-sections of the retina, allowing its thickness to be measured. This test can also show abnormal fluid build-up in and under the retina. OCT works like ultrasound, except that it measures scattered light instead of sound waves [14,5].

Angiography

If you have macular edema, you will probably also undergo a fluorescein angiogram. Fluorescein is a yellow dye injected into a vein that's taken up by vessels of the eye, allowing your vision care provider to visualize any leaking vessels in your retina. A similar dye called indocyanine green may also be used to obtain additional information about the vasculature (vessel structure) of your eye. Eye diseases other than diabetic retinopathy can cause edema, so your vision care provider must carefully consider your signs and symptoms.

TREATMENT

During the nonproliferative stage of diabetic retinopathy, treatment consists of controlling glucose levels, keeping blood pressure within a desired range, and limiting blood cholesterol. An exception is if macular edema has developed, in which case focal laser therapy is often very effective in preserving vision. Once blood vessels begin to appear during the proliferative stage, treatments include scatter laser therapy and vitrectomy. Investigational treatments are being pursued as well.

Drug Therapy for Diabetic Retinopathy

Intravitreal (“inside the eye”) drug injections:

- Triamcinolone (a corticosteroid; mechanism of action unclear)
- Macugen, Lucentis, & Avastin (drugs primarily used to manage neovascular (“wet”) age-related macular degeneration (reduction in the severity of retinopathy by inhibiting VEGF). Macugen & Lucentis are FDA approved to treat “wet” AMD; none of these medications are yet FDA approved to treat diabetic retinopathy.
- Intravitreal drug injections are usually performed with laser procedures in order to enhance response to treatment.

Aspirin treatment

The Early Treatment Diabetic Retinopathy Study (ETDRS) investigated whether aspirin (650 mg/day) could retard the progression of retinopathy. After examining progression of retinopathy, development of vitreous hemorrhage, or duration of vitreous hemorrhage, aspirin was shown to have no effect on retinopathy. With these findings, there are no ocular contraindications to the use of aspirin when required for cardiovascular disease or other medical indications.

People with proliferative diabetic retinopathy can reduce their risk of blindness by 90% with appropriate treatment and follow-up. Few serious conditions have such controllable outcomes.

During the nonproliferative stage of diabetic retinopathy, treatment consists of controlling glucose levels, keeping blood pressure within a desired range, and limiting blood cholesterol. In the late non-proliferative stage, focal laser treatments may be used to seal leaking blood vessels. Once new blood vessels begin to appear in the proliferative stage, scatter laser treatment can be used to shrink them [15].

Laser Therapy

Laser therapy is usually an outpatient procedure requiring only a topical anesthetic. It aims to stabilize vision but generally does not restore sight that has already diminished.

Focal or pan retinal laser treatment for macular edema

Macular edema may occur in late-stage nonproliferative diabetic retinopathy. If it has not caused scarring, macular edema can be treated with a focal laser that seals off leaking blood vessels. This approach lowers the risk of vision loss by 20% in people with mild or moderate nonproliferative diabetic retinopathy. As the disease becomes more severe and extensive, a scatter laser can be used [5,10].

Scatter (pan retinal) laser treatment

Scatter laser photocoagulation (sealing off leaking vessels with a laser beam) has been shown to reduce the risk of blindness by as much as 90%. Few treatments for any disease can boast of such success. This therapy slows the progression to proliferative retinopathy and cuts in half the chance that vitrectomy will be needed in certain patients. Because the procedure destroys part of the retina, you risk some loss of central vision, night vision, and peripheral vision.

Surgery

Vitrectomy

The retina is a thin layer of tissue at the back of the eye. It’s sandwiched between the choroid and the vitreous humor, a jelly-like substance that fills the cavity of the eyeball. Abnormal vessels caused by diabetic retinopathy can hemorrhage into the vitreous humor. Blood in the vitreous can diminish vision by keeping light from reaching the retina, and it can also impede laser surgery.
When hemorrhaging occurs, the vitreous can be suctioned right out of its chamber in the eyeball. Usually under local anesthesia, your eye surgeon will make a small incision into the sclera (the "white" of the eye) to access the vitreous, which is replaced with a neutral saline solution to maintain the shape of the eyeball.

**EXPERIMENTAL TREATMENTS**

**C-peptide**

Though not yet commercially available, c-peptide has shown promising results in treatment of diabetic complications incidental to vascular degeneration. Once thought to be a useless byproduct of insulin production, it helps to ameliorate and reverse many symptoms of diabetes.

**Pine bark extract**

A pine bark extract of Oligomeric proanthocyanidins has been shown to improve microcirculation, retinal edema and visual acuity in the early stages of diabetic retinopathy.

**Precautions & care**

**Tightly control your blood sugar.** Tight glucose control is the linchpin of both prevention and management of diabetic retinopathy. Management is aimed is slowing disease progression in order to preserve vision.

**Keep blood pressure and cholesterol within a narrow range.** Controlling your blood pressure will reduce your risk of low vision and confer cardiovascular and other health benefits at the same time.

**Reduce body mass index by following healthy lifestyle recommendations.** Get moving! Aerobic physical activity doesn't have to involve leg warmers and Spandex. It just means you need to exercise at moderate intensity at least 2½ hours per week. Brisk walking, swimming, cycling, snowshoeing, or any other activity will do, as long as it's powered by you.

**Stop smoking or don't start.** Smoking has a deleterious effect on virtually every body system, and the eyes are no exception. The chemicals in tobacco are believed to keep your body from properly absorbing lutein, an antioxidant that helps shield the retina. An equally important consideration is that people with diabetes have a high risk of stroke and heart attack, and smoking markedly elevates that risk. If you're a smoker, speak with your doctor about your smoking cessation options [11,15].

**CONCLUSION**

**Current Research**

1. The National Eye Institute (NEI) is conducting and supporting research that seeks better ways to detect, treat, and prevent vision loss in people with diabetes. This research is conducted through studies in the laboratory and with patients.

2. For example, researchers are studying drugs that may stop the retina from sending signals to the body to grow new blood vessels. Someday, these drugs may help people control their diabetic retinopathy and reduce the need for laser surgery.
REFERENCES


