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Diabetic retinopathy: A review

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Abstract
Diabetic retinopathy (DR) is a vascular disease of the retina which affects patients with diabetes mellitus. It is the number one cause of blindness in people between the ages of 20-64 in the United States. It is, therefore, a worthwhile topic for all homoeopathy students to review. Diabetes mellitus is extremely common, so it is not surprising that DR affects 3.4 percent of the population (4.1 million individuals) of the millions of people with diabetic retinopathy, nearly one fourth have vision-threatening disease.

Keywords: Diabetic, retinopathy, homoeopathy

Introduction
Diabetes Mellitus (DM) and the eye diseases associated with it comprise a set of complex disorder with multi-factorial etiology, where both genetic and environmental factors play an active role. It is a major cause of avoidable blindness in both the developed and developing countries. Newly diagnosed diabetic cases are increasing at an alarming rate in the developing countries like India due to better life style and the demographic right shift of the population, urbanization and disparities in the access to the health care system.
Approximately 382 million people across the world have been estimated to have diabetic mellitus in 2013 and if no action is taken this number will rise to 592 million by 2035. WHO estimates that 19% of the world's diabetic population lives in India and 80 million people in India will have diabetes by the year 2030. Diabetic retinopathy (DR) is the most common complication of diabetes mellitus. It has been seen that patients having Diabetic retinopathy are 25 times more at risk of blindness than a non diabetic individual. Timely diagnosis with the help of better screening and referral facilities, strict control of systemic parameters and timely intervention in the form of medical and surgical intervention can delay the sight threatening complication of diabetic retinopathy [1].

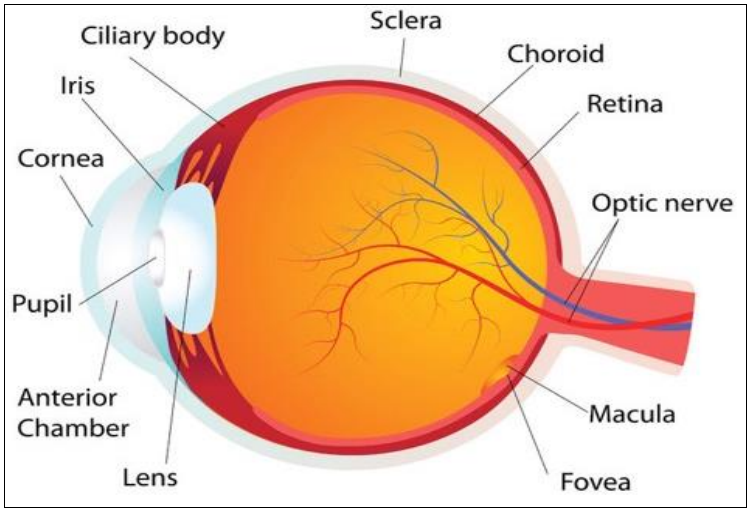

Fig 1: Anatomy of the Eye

Epidemiology
It has been estimated that 30% of people with diabetes mellitus have diabetic retinopathy worldwide. A pooled analysis of 35 studies showed that the overall prevalence of diabetic retinopathy of any severity is 34.6% and the prevalence of proliferative diabetic retinopathy

(PDR) and diabetic macular edema (DME) is 6.96% and 6.81% respectively. The presence of diabetic retinopathy is directly proportional to the duration of diabetes mellitus. **S**eoul metropolitan city diabetes prevention programme study estimated that 55.2% patient had diabetic retinopathy with more than 10 yrs of diabetes mellitus as compared to 12.6% with less than 10 yrs of diabetes mellitus with an approximately three fold increase in the vision threatening diabetic retinopathy in patients with more than 10 years of diabetes mellitus [2]. The prevalence of diabetic retinopathy in India is approximately 5.6 million. There may be 2.9 million people with mild non proliferative diabetic retinopathy (NPDR), 2.2 million people with moderate NPDR, 111,258 people with severe NPDR and 296,688 people with proliferative diabetic retinopathy [3].

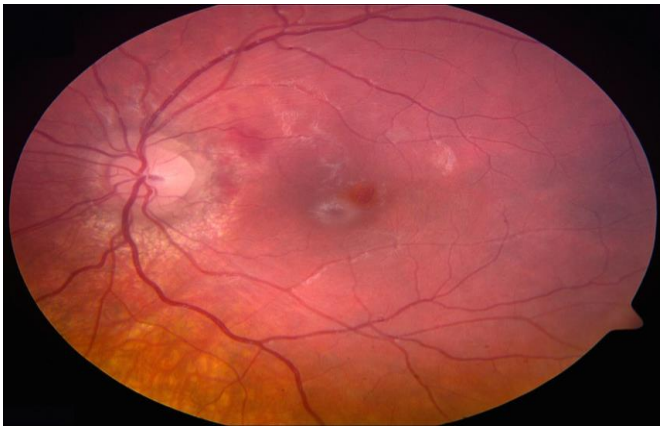

Fig 2: Normal Fundus

The prevalence rate of diabetes retinopathy in type 2 diabetes mellitus was reported as 34.1% from south India. Chennai urban rural epidemiology study (CURES) estimated that the overall prevalence of diabetic retinopathy in urban population to be 17.6%6. Several reports suggest that Indians with type 2 diabetes mellitus may differ from their European counterparts in many aspects, including younger age of onset, obesity, insulin resistance and genetic predisposition.

Pathophysiology

The vascular disruption of DR is characterised by abnormal vascular flow, disruption in permeability and closure or non perfusion of capillaries. Microvascular leakage and microvascular occlusion are the two main pathological processes leading to the changes in diabetic retinopathy. The earliest change seen in diabetes before development of retinopathy is the breakdown of the blood retinal barrier (BRB). Aldose reductase present in the pericytes alters the cellular metabolism, resulting in the loss of pericytes. This disrupts the autoregulation and lead to breakdown of BRB and leakage of plasma. Loss of pericytes also leads to saccular out pouching of the capillary walls known as microaneurysms. These microaneurysms tend to gradually enlarge, thickening and hyalinization of the walls takes place, which eventually lead to auto occlusion by encroachment of the thickened wall into the lumen. Other factors responsible for microvascular occlusion are capillary endothelial cell damage and proliferation; change in red blood cells leading to defective oxygen transport and increased stickiness and aggregation of platelets [3].

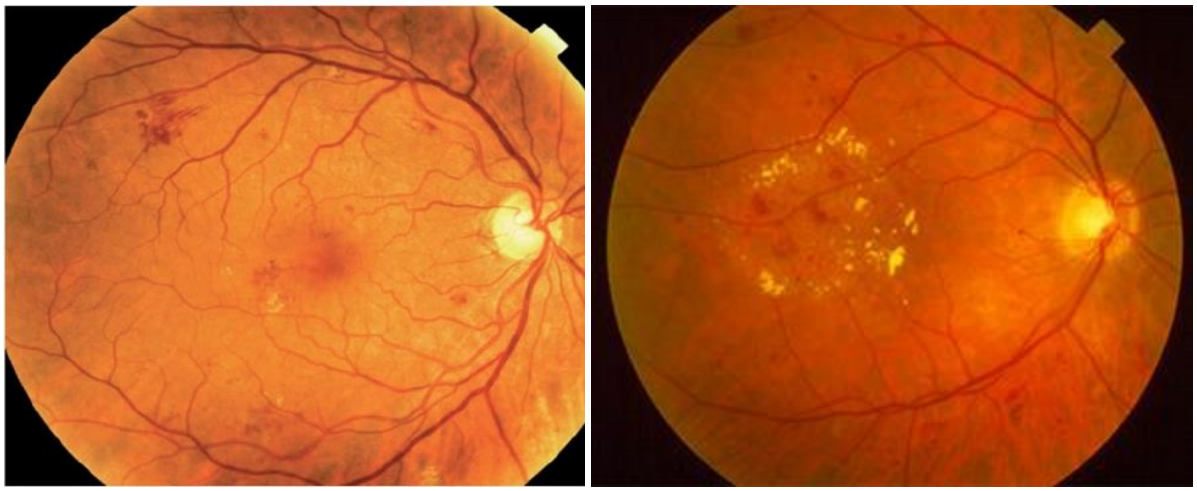

Fig 3: Nonproliferative Diabetic Retinopathy (NPDR)

Aetiology

The primary cause of diabetic retinopathy is diabetes-a condition in which the levels of glucose (sugar) in the blood are too high. Elevated sugar levels from diabetes can damage the small blood vessels that nourish the retina and may, in some cases, block them completely. When damaged blood vessels leak fluid into the retina it results in a condition known as diabetic macular edema which causes swelling in the center part of the eye (macula) that provides the sharp vision needed for reading and recognizing faces. Prolonged damage to the small blood vessels in the retina results in poor circulation to the retina and macula prompting the development of growth factors that cause new abnormal blood vessels (neovascularization) and scar tissue to grow on the surface of the retina [4]. This stage of the disease is known as proliferative diabetic retinopathy (PDR). New vessels may bleed into the middle of the eye, cause scar tissue formation, pull on the retina, cause retinal detachment, or may cause high pressure and pain if the blood vessels grow on the iris, clogging the drainage system of the eye-all of this can cause vision loss [5].

Classification

Diabetic retinopathy falls into two main classes: nonproliferative and proliferative. The word “proliferative” refers to whether or not there is neovascularization (abnormal blood vessel growth) in the retinaEarly disease without neovascularization is called nonproliferative

diabetic retinopathy (NPDR). As the disease progresses, it may evolve into proliferative diabetic retinopathy (PDR), which is defined by the presence of neovascularization and has a greater potential for serious visual consequences.

Nonproliferative Diabetic Retinopathy

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Proliferative Diabetic Retinopathy

As mentioned earlier, the retina has a high metabolic requirement, so with continued ischemia, retinal cells respond by releasing angiogenic signals such as vascular endothelial growth factor (VEGF). Angiogenic factors, like VEGF, stimulate growth of new retinal blood vessels to bypass the damaged vessels. This is referred to as neovascularization. In PDR, the fibrovascular proliferation extends beyond the ILM. This may sound like a good idea, but the new vessels are leaky, fragile, and often misdirected. They may even grow off the retina and into the vitreous. As the vitreous shrinks with age, it pulls on these fragile vessels and can cause them to tear, resulting in a vitreous hemorrhage and sudden vision loss. These vessels may also scar down, forming strong anchors between the retina and vitreous causing traction on the retina. If enough force is created, a tractional retinal detachment may occur. This is another mechanism by which DR can cause sudden vision loss. If the retina is not re-attached soon, especially if the macula is involved, vision may be permanently compromised [6]. While the effects of neovascularization in PDR can be devastating, the most common cause of vision loss in diabetics is macular edema. Macular edema can occur in NPDR, but it is more common in more severe cases of DR due to the leakiness of the new blood vessels [7].

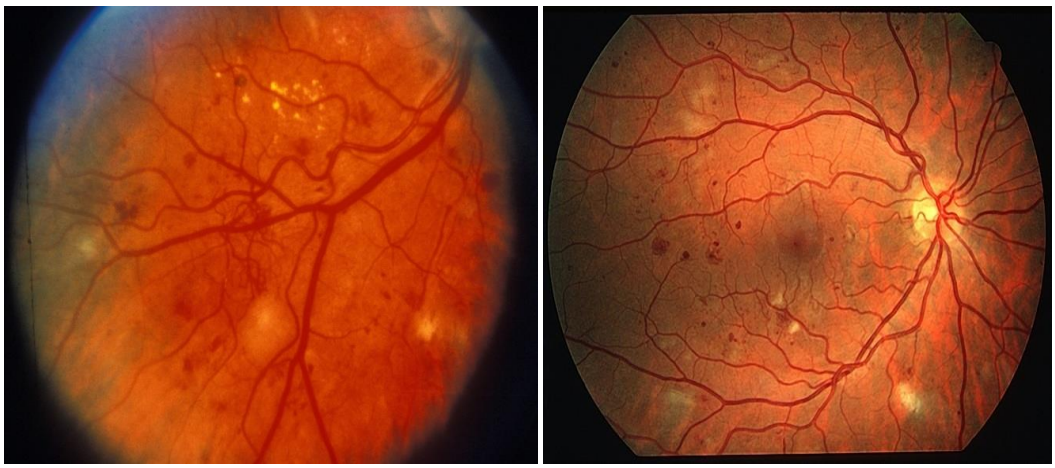

Fig 4: Proliferative Diabetic Retinopathy (PDR)

Diabetics can also have problems located more anteriorly in the eye [8]. The angiogenic molecules that are produced by the retina may float anteriorly, causing neovascularization of the iris. These vessels can grow into the angle of the anterior chamber where the trabecular meshwork, the drain of the eye, resides. This can obstruct outflow of aqueous fluid, raising intraocular pressure and causing acute glaucoma [9, 10].

Symptoms

- Blurred or double vision
- Difficulty reading [11].
- The appearance of spots-commonly called "floaters"-in your vision
- A shadow across the field of vision
- Eye pain or pressure
- Difficulty with colour perception [12, 13, 14].

Diagnosis

The best way to diagnose diabetic retinopathy is a dilated eye exam. During this exam, the physician places drops in the eyes to make the pupils dilate (open widely) to allow a better view of the inside of the eye, especially the retinal tissue [15].

Regular dilated eye exams by an ophthalmologist are important, especially for those who are at a higher risk for diabetic retinopathy or diabetes. If you are over age 50, an exam every 1 to 2 years is a good idea so the physician can look for signs of diabetes or diabetic retinopathy before any vision loss has occurred. In addition to checking for signs of diabetic eye disease, a comprehensive dilated eye exam will evaluate your vision/need for corrective lenses, eye pressure (looking for glaucoma), the "front" of the eye (eyelids, cornea, checking for dry eye), lens (looking for cataracts), as well as a complete exam of the retina and vitreous. In addition to this exam, physicians use other tests to detect and manage diabetic retinopathy: An optical coherence tomography (OCT) test provides highly detailed cross-sectional images of the retina that show its thickness, helping determine whether fluid has leaked into retinal tissue [16].

Risck Factors

Duration of diabetes mellitus**:** It has been reported that there a direct correlation between the duration of diabetes mellitus and severity of diabetic retinopathy. There is 99% risk of DR in patients of insulin depended diabetes mellitus of 20 years duration as compared to 60% risk of diabetes retinopathy in NIDDM patients with same duration [17].

Genetic predisposition: A lot of research is in process for understanding the genetic predisposition for diabetes retinopathy in diabetes mellitus. A large no of genes and genetic variants have been reported but till date no gene have been accepted as a high risk gene for diabetes retinopathy. Recently genetic association for susceptibility to diabetes retinopathy has been found in five chromosomal regions and PLXDC2 and ARHGAP22. The latter two genes are being implicated in the endothelial cell angiogenesis and increased capillary permeability in Taiwanese population [18].

Insulin resistance: Insulin resistance is an independent specific marker of proliferative retinopathy that may characterize patients at increased risk for blindness.

Treatment

Non proliferative diabetic retinopathy is typically managed by optimizing the patient's general health. The best treatment for diabetic retinopathy is prevention of its development and progression with tight glucose control. Patients should maintain a HbA1c ≤7%. Blood pressure management has also been shown to decrease disease progression (UKPDSG 1998) and patients should be counseled to stop smoking. Ophthalmologists should intervene if the patient has clinically significant macular edema (CSME) with Non proliferative diabetic retinopathy. The causative microaneurysms are localized, often using fluorescein angiography, and then directly treated with laser therapy. If the leakage is more diffuse, a grid of light laser burns can slow the edema. Finally, several off-label medical options are available, such as intravitreous injections of triamcinolone and antibodies against VEGF such as Lucentis or Avastin [19].

Once a patient has developed PDR, there are several treatment modalities available. Treating macular edema in proliferative diabetic retinopathy is similar to treating Non proliferative diabetic retinopathy. Proliferative diabetic retinopathy, however, also has additional therapy options aimed at taming the growth of new, problematic vessels. The mainstay of treatment is panretinal photocoagulation (PRP), in which portions of retina are destroyed using thousands of laser burns while sparing the macula. It is hypothesized that this may reduce the amount of ischemic retina, and thus, reduce the production of angiogenic molecules. The treatment may sound extreme, but actually causes surprisingly little vision loss. It has been found to be extremely effective, reducing the risk of severe vision loss by 50% and resulting in regression of neovascularization in 30-55% of patients.

Patients with non-resolving vitreous haemorrhages or severe traction causing retinal detachment may benefit from a vitrectomy. In this procedure, the vitreous gel and haemorrhage are removed from the eye and replaced with a saline solution.

Homoeopathy Medicines

With help of homoeopathic medicines can be prevent the diabetic retinopathy.

Sanicula

Lids agglutinated in morning. Lachrymation in cold open air or cold application. Eyes burn, exuding a sticky fluid. Dandruff of eyebrows. Sight dim. Cornea ulcerated. Photophobia.

Physostigma

Blood eyes with burning. Glaucoma. Contraction of pupils. Profuse lachymation. Increasing myopia. Eyelids tense, Night-blindness, Photophobia. Muscae volitantes, flashes of light. Increasing myopia. Vision trembling.

Phosphorus

Eyeballs feel large, stiff, Choroiditis. Fatigue of eye and head even without much use of eyes. Edema of lids and about eyes. Glaucoma. Cataract. Vitreous opacities. Atrophy of optic nerve. Black points seem to float before the eyes. Green halo about the candlelight. Letters appear red. Thrombosis of retinal vessels and degenerative changes in retina. Retinal trouble with the lights.

Lachesis

Defective visions after diphtheria, sensation as of eyes were drawn together by cords, which were tied in a knot at root of nose. Blindness with lung or heart disorders. Eyes watery from pain. Intra-ocular haemorrhages. Feels as if eyes were forced out on pressing the throat. Dim sight. Conjuctivitis with measles. The eyes water all the time. Acrid lachrymation, bland coryza. Thick, acrid, yellow discharge from the eyes. Profuse hot or acrid tears worse open air, Pressive, cutting pains in the eyes. Sticky mucus on cornea, must wink to remove it. Pressure in eyes. Little blister on cornea. Cataract with watery eyes.

Arnica

Bloodshot. Retinal haemorrhage. Black eye. Bruised, sore feeling in eyes after close work. Must keep eyes open. Dizzy on closing them. Feel tired and weary after sight seeing, moving pictures, etc. Photophobia. High objects appear to lean forward and about to fall [20].

Conclusion

Diabetic retinopathy is the most common complication of diabetes which may lead to legal blindness and is a major public health problem. Early detection through screening, educating the population and timely intervention may decrease the complications in the course of disease.

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