PERSPECTIVE

Diabetes Management

Short note on diabetic retinopathy

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Diabetic retinopathy often has no early caution signs. Even macular edema, which can effect rapid central visualization loss, may not have any caution signs for some time. In general, however, a person with macular edema is likely to have blurred visualization, making it hard to do things like read or drive. In some cases, the vision will get better or worse during the day.

Non-Proliferative Diabetic Retinopathy (NPDR) is the first stage, which has no symptoms. Patients with 20/20 vision may not notice the indications. Fundus examination with a direct or indirect ophthalmoscope by a trained ophthalmologist or optometrist is the only way to detect NPDR. Fundus photography can be used for objective documentation of the fundus findings, which can show microaneurysms (microscopic bloodfilled bulges in the artery walls). Fluorescein angiography can clearly indicate narrowed or obstructed retinal blood vessels if vision is impaired (lack of blood flow or retinal ischemia).

Macular edoema is a condition in which blood vessels leak their contents into the macular region. It can happen at any stage of the NPDR process. Blurred vision and darkened or distorted images that are not the same in both eyes are signs of this condition. Approximately 10% of diabetic people will experience vision loss due to macular edoema. Optical coherence is a term that refers to the ability of light to Tomography can reveal areas of retinal thickness caused by macular edoema fluid accumulation.

As part of Proliferative Diabetic Retinopathy (PDR), abnormal new blood vessels (neovascularisation) grow at the back of the eye; these might rupture and bleed (vitreous haemorrhage) and obscure vision since these new blood vessels are fragile.

Risk factors

All diabetics are at risk, whether they have Type I or Type II diabetes. The longer a person has diabetes, the more likely they are to develop an eye condition. Diabetic retinopathy affects 40 to 45 percent of people in the United States who have diabetes. Nearly all Type I diabetes patients and>60 percent of Type II diabetes patients had some degree of retinopathy after 20 years of diabetes; however, these results were published in 2002 using data from four years earlier, limiting the research's applicability. In the late 1970s, before contemporary fast-acting insulin and home glucose monitoring, the subjects would have been diagnosed with diabetes.

Prior studies had also assumed a clear glycemic threshold between people at high and low risk of diabetic retinopathy. Published rates vary between trials, the proposed explanation being differences in study methods and reporting of prevalence rather than incidence values.

Genetics also play a role in diabetic retinopathy. Genetic predisposition to diabetic retinopathy in type 2 diabetes consists of many genetic variants across the genome that are collectively associated with diabetic retinopathy (polygenic risk) and overlaps with genetic risk for glucose, low-density lipoprotein cholesterol, and systolic blood pressure.

Pathogenesis (Immunopathogenesis)

Diabetic retinopathy is caused by damage to the retina's tiny blood vessels and neurons. Narrowing of the retinal arteries, which leads to reduced retinal blood flow; dysfunction of the

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neurons of the inner retina, followed by changes in the function of the outer retina, which leads to subtle changes in visual function; and dysfunction of the blood-retinal barrier, which protects the retina from many substances in the blood (including toxins and immune cells), which leads to the leaking of blood. Later, the retinal blood vessel basement membrane thickens, capillaries deteriorate, and cells, particularly pericytes and vascular smooth muscle cells, are lost. This results in decreased blood flow and progressive ischemia, as well as microscopic aneurysms, which look as balloon-like structures protruding out from capillary walls and attract inflammatory cells; and progressed retinal neuron and glial cell malfunction and degeneration. The syndrome usually appears 10–15 years after being diagnosed with diabetes mellitus.