



Pathophysiology of Diabetic Retinopathy

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DESCRIPTION

Diabetic retinopathy, also known as diabetic eye disease, is a condition in which the retina is damaged due to true diabetes. This is the main cause of blindness in developed countries. Diabetic retinopathy affects up to 80 percent of people who have had diabetes for over 20 years. With proper eye care and monitoring, you can reduce at least 90% of new cases. The longer a person has diabetes, the more likely he or she will develop diabetic retinopathy. Diabetic retinopathy accounts for 12% of all new cases of blindness in the United States each year. It is also a major cause of blindness in people between the ages of 20 and 64.

Pathophysiology

The exact mechanism by which diabetes causes retinopathy remains unclear, but several theories have been put forward to explain the typical course and history of diabetes.

Growth hormone

Growth hormone seems to play a function for the improvement and development of diabetic retinopathy. Diabetic retinopathy has been proven to be reversible in ladies with postpartum hemorrhagic necrosis (Shehan's syndrome) of the pituitary gland. This caused the debatable exercise of pituitary ablation to deal with or save you from diabetic retinopathy withinside the 1950s. This method was changed into sooner or later deserted because of many systemic headaches and the invention of the effectiveness of laser treatment. It must be stated that diabetic retinopathy has additionally been pronounced in sufferers with hypopituitarism.

Platelets and blood viscosity

The style of hematologic abnormalities visible in diabetes, together with expanded erythrocyte aggregation, reduced crimson blood mobileular deformability, expanded platelet aggregation, and adhesion lead the affected person to slow circulation, endothelial damage, and focal capillary

occlusion. This results in retinal ischemia, which turn to contributes the improvement of diabetic retinopathy.

Aldose reductase and vasoproliferative factors

Fundamentally, diabetes mellitus reasons atypical glucose metabolism due to the reduced stages or pastime of insulin. Increased stages of blood glucose are concept to have a structural and physiologic impact on retinal capillaries inflicting them to be each functionally and anatomically incompetent.

A continual in blood glucose stages shunts extra glucose into the aldose reductase pathway in positive tissues, which converts sugars into alcohol. The pericytes withinside the wall of the retinal capillaries appear like suffering from this expanded sorbitol level, in the long run main to the lack of their important function. This results in weakening of the capillary wall and in the long run a saccular bulge. These microaneurysms are the earliest detectable symptoms and symptoms of DM retinopathy.

Nailfold video capillary angiography reveals a high prevalence of capillary changes in diabetic patients, especially those with retinal injury. This reflects the involvement of common microvessels in both type 1 and type 2 diabetes.

A ruptured microaneurysm causes retinal hemorrhage either superficially or in a deeper layer of the retina Increased permeability of those vessels effects leakage of fluid and proteinaceous material, which clinically seems as retinal clot and exudates. If the swelling and exudation contain the macula, a diminution in critical imaginative and prescient can be experienced.

Macular edema

Macular edema is the one of the unusual place imputes of imaginative and prescient loss sufferers with nonproliferative diabetic retinopathy. However, it isn't always completely visible in sufferers with NPDR; it could additionally complicate instances of proliferative diabetic retinopathy. Another theory that explains the development of macular edema focuses on

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elevated levels of diacylglycerols due to excess glucose moved. It is through the activate protein kinase C, which affects retinal hemodynamics, especially permeability and flow, causing fluid leakage and thickening of the retina.

Hypoxia

Ultimately, as the disease progresses, the retinal capillaries become obstructed, causing hypoxia. Infarction of the nerve fiber layer leads to the formation of cotton wool patches with stagnation associated with the flow of axoplasm. Wider range of retinal hypoxia causes the eye's compensatory mechanism to supply sufficient oxygen to tissues. Abnormal venous diameters such as venous beading, loops, and dilation indicate an increase in hypoxia and are most often found in the non-perfused areas of the capillaries. Intraretinal microvascular abnormalities represent either the growth of new blood vessels or the remodeling of existing blood vessels by the proliferation of endothelial cells in the retinal tissue, acting as a shunt through the non-perfused region.

Angiogenesis

Further increase in retinal ischemia causes the production of angioplasty factors that stimulate new angioplasty. The

extracellular matrix is first degraded by proteases, with new blood vessels originating primarily from the retinal venules penetrating the internal limiting membrane, forming a capillary network between the inner surface of the retina and the posterior vitreous surface.

In sufferers with proliferative diabetic retinopathy, nocturnal intermittent hypoxia/reoxygenation that effects from sleep-disordered respiration can be a threat element for iris and/or attitude neovascularization.

Neovascularization is maximum typically discovered on the borders of perfused and nonperfused retina and maximum typically takes place alongside the vascular arcades and on the optic nerve head. The new vessels destroy and develop alongside the floor of the retina and into the scaffold of the posterior hyaloid face. By themselves, those vessels hardly ever motive visible compromise, however they're fragile and pretty permeable. These sensitive vessels are disrupted without problems with the aid of using vitreous traction, which results in hemorrhage into the vitreous hollow space or the preretinal space.