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Diabetes and Diabetic Retinopathy

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Revised May 2026

Diabetes is a condition where the body has trouble regulating blood sugar. Over time, persistently elevated blood sugar can damage blood vessels and organs, including the eyes, kidneys, nerves, and heart. There are two main types of diabetes. Type 1 diabetes typically begins in childhood or early adulthood and usually requires insulin therapy from the time of diagnosis. Type 2, or adult-onset, diabetes develops later in life and is often managed with diet alone or in combination with oral medications; however, some individuals eventually require insulin therapy as the disease progresses.

Diabetic retinopathy is characterized by damage to the retinal blood vessels and impaired delivery of oxygen and nutrients to the areas they supply. It is one of the leading causes of blindness in the United States and across developed countries worldwide. Thirty-seven million Americans have diabetes; only about half are aware that they have it. Almost everyone who has diabetes for 20 or more years will develop some degree of diabetic retinopathy. In most patients, the initial form of retinopathy is called “background” diabetic retinopathy, characterized by microaneurysms and the development of retinal hemorrhages, exudates, and edema. With increasing deprivation of oxygen and nutrients, new blood vessels may form in the retina and extend into the vitreous. This phase is called “proliferative” diabetic retinopathy. Although it might seem that new blood vessels would be beneficial by improving blood and oxygen supply to ischemic retinal areas, these vessels are abnormal, fragile, and prone to leakage and rupture. As a result, they often cause hemorrhage and can lead to significant vision loss. Proliferative diabetic retinopathy occurs in approximately 25% and 50% of patients with insulin-dependent diabetes after 15 and 20 years of disease duration, respectively. Additionally, even in those patients with Type 2 diabetes not taking insulin, a quarter will develop proliferative diabetic retinopathy at about the 25-year anniversary of their diabetes.

In addition to disease duration, the degree of glycemic control is a key factor influencing the development of diabetic retinopathy, the better and more consistent the control, the

lower the risk of proliferative disease. Accordingly, prevention may be possible through rigorous daily glycemic management. Other risk factors for retinal and vascular disease, such as hypertension, hyperlipidemia, and smoking, may also contribute and warrant careful attention.

Treatment of diabetic retinopathy depends on its severity and the presence of diabetic macular edema, which does not always correlate with the degree of retinopathy. Management has evolved substantially with the introduction of intravitreal corticosteroid or anti–vascular endothelial growth factor (anti-VEGF) therapies over the past 20 years.

For background diabetic retinopathy without macular edema, observation with follow-up at 3- to 12-month intervals is recommended, depending on disease severity. The presence of diabetic macular edema warrants treatment with intravitreal corticosteroid and anti-VEGF injections; in patients who do not respond adequately, laser therapy may be considered.

In proliferative diabetic retinopathy without diabetic macular edema, retinal laser photocoagulation remains the mainstay of therapy with durable effect. However, intravitreal anti-VEGF injections are an effective alternative, particularly in cases with media opacities (i.e., cataract and vitreous hemorrhage) that may preclude laser treatment. In his eyes with coexisting diabetic macular edema, initial treatment with intravitreal anti-VEGF injections is generally preferred.

Finally, pars plana vitrectomy may be required in patients who develop complications of proliferative diabetic retinopathy, such as retinal detachment, epiretinal membrane, or vitreous hemorrhage.