

Diabetes and Diabetic Retinopathy

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Diabetic retinopathy is a leading cause of blindness in the United States, and, indeed, in all developed countries. Sixteen million Americans have diabetes; only about half are aware that they have it. One type, Type 1, has its onset in childhood or young adulthood, and it typically requires insulin therapy nearly from the beginning of its onset. Type 2, adult onset diabetes, often can be treated with diet or diet plus oral medications, but a significant number of people with Type 2 diabetes also evolve into the need for insulin therapy.

Diabetic retinopathy is characterized by blood vessel damage and poor delivery of oxygen and nutrients to areas previously supplied by the damaged blood vessels. Almost everyone who has diabetes for 20 or more years will develop some degree of diabetic retinopathy. Typically the type of retinopathy which develops in most patients, at least in the beginning, is called "background" retinopathy, with the development of small aneurysms in blood vessels, and the development of hemorrhages and exudates and edema in the retina. As areas of the retina become more and more starved for oxygen, these changes in the retina increase, and eventually new blood vessel formation develops, the phase of diabetic retinopathy known as "proliferative" diabetic retinopathy. Ordinarily, one might imagine that such new blood vessels would be good, given the fact that perhaps they could supply more blood and oxygen to the oxygen-starved areas of retina. However, these blood vessels are not normal, are incredibly delicate, typically leak, and frequently rupture and hemorrhage, producing pronounced degrees of vision loss as a result of these consequences.

This form of advanced, proliferative diabetic retinopathy occurs in about 25% of patients with insulin dependence for 15 years or more. 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 the duration of diabetes, the "tightness" of blood sugar control is a clear additional factor related to the likelihood of onset of diabetic retinopathy; the better and more faithful the blood sugar control, the less likely proliferative diabetic retinopathy is to occur.

Prevention, therefore, may be possible through very vigorous blood sugar control each day. Additional risk factors for retinal and blood vessel disease (high blood pressure, high blood fat levels, smoking, etc.) may also be influential and are worthy of attention.

Treatment of diabetic retinopathy, once it develops, revolves around laser photocoagulation treatments (in addition, of course, to proper blood sugar control). Patients who develop mild to moderate "background" retinopathy with clinically significant edema of the retina may require simple "spot" treatment with laser, while those who develop more severe "background" retinopathy, with or without clinically significant macula edema, will probably require much more extensive (so called "pan retinal") laser treatment. Patients with proliferative diabetic retinopathy will usually require pan retinal photocoagulation and sometimes will require additional, spot treatments near the macula. Finally, vitrectomy surgery will be required for those patients who develop proliferative retinopathy with hemorrhage into the vitreous.