20 DIABETIC RETINOPATHY

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PATIENT STORY

A 38-year-old man saw a physician for the first time in 10 years after noticing visual loss in his left eye. His history revealed many risk factors for and symptoms of diabetes mellitus (DM). On an undilated funduscopic examination, his physician was able to see some hemorrhages and hard exudates. A fingerstick in the office showed a blood glucose level of 420 mg/dL. He was treated for DM and referred to an ophthalmologist to be evaluated for his diabetic retinopathy (Figure 20-1).

INTRODUCTION

Diabetic retinopathy (DR) is a leading cause of blindness in the United States. Nonproliferative DR is characterized by microaneurysms, macular edema, cotton-wool spots, superficial (flame) or deep (dot-blot) hemorrhages, and exudates. Proliferative DR also has neovascularization of the retina, optic nerve head, or iris. Because patients may be asymptomatic until vision loss occurs, screening is indicated in all diabetic patients. Excellent glycemic control lowers a patient’s risk of developing DR.

EPIDEMIOLOGY

- In developed nations, DR is the leading cause of blindness among people younger than age 40 years.
- In a community-based study, 29% of adults older than age 40 years with DM had DR. Prevalence in black patients was higher than in white patients (38.8% vs. 26.4%).
- Twenty-one percent of patients have retinopathy at the time type 2 diabetes is diagnosed.
- More than 60% of patients with type 2 DM have retinopathy within 20 years of diagnosis.
- After 40 years of type 1 DM, 84% of patients have retinopathy.

ETIOLOGY AND PATHOPHYSIOLOGY

- Hyperglycemia results in microvascular complications including retinopathy.
- Several biochemical pathways linking hyperglycemia and retinopathy have been proposed.
- In nonproliferative retinopathy, microaneurysms weaken vessel walls. Vessels then leak fluid, lipids, and blood resulting in macular edema, exudates, and hemorrhages (Figures 20-1 and 20-2).
- Cotton-wool spots result when small vessel occlusion causes focal ischemia to the superficial nerve fiber layer of the retina.
• In proliferative retinopathy, new blood vessels form in response to ischemia (Figure 20-3).

RISK FACTORS

• In type 1 DM, identified risk factors include: longer diabetes duration, high hemoglobin (Hgb) A1c, hypertension, smoking, and male gender.4,5
• In type 2 DM, identified risk factors include: longer diabetes duration, high HgbA1c, elevated systolic blood pressure, male gender, presence of albuminuria, and pharmacologic therapy.6

DIAGNOSIS

Definitive diagnosis is made by an eye specialist:
• Gold standard is grading of stereoscopic color fundus photographs in seven standard fields.3
• In comparison with the gold standard, a single monochromatic digital photo through a nondilated eye is sufficient to determine the presence or absence of DR with a sensitivity and specificity of 71% and 96%, respectively.7 SOR B

CLINICAL FEATURES

• Central vision loss as a result of macular edema or macular ischemia.
• Nonproliferative retinopathy—Microaneurysms are seen initially (mild), followed by macular edema, cotton-wool spots, superficial (flame) or deep (dot-blot) hemorrhages, and exudates (Figure 20-1 shows moderate, and Figure 20-2 shows severe).
• Proliferative retinopathy—Neovascularization, that is, growth of new blood vessels on the optic disc (Figure 20-3), the retina, or iris.

DIFFERENTIAL DIAGNOSIS

Retinopathy is also seen with other systemic illnesses and infections including:
• Hypertensive retinopathy—Arterial narrowing or atrioventricular nicking in addition to cotton-wool spots (see Chapter 21, Hypertensive Retinopathy).
• HIV retinopathy—Cotton-wool spots and infections such as Cytomegalovirus.

MANAGEMENT

Control diabetes and vascular risk factors:
• Glycemic control lowers the risk of retinopathy (35% risk reduction per 1 point HgbA1c reduction).3 SOR A
• Blood pressure control improves visual outcomes (34% risk reduction in retinopathy progression; 47% risk reduction for declines in visual acuity).3 SOR A

FIGURE 20-3 Proliferative diabetic retinopathy showing newly developed, porous, friable blood vessels. New vessels can be seen on the optic disc and peripheral retina. Panretinal photocoagulation may help prevent vitreous hemorrhage, retinal detachment, and neovascular glaucoma. (Courtesy of Paul D. Comeau.)

FIGURE 20-4 This vitreous hemorrhage occurred when friable neovascular membranes broke spontaneously. The patient described a “shower of red dots” obscuring the vision and then loss of vision in that eye. (Courtesy of Paul D. Comeau.)
Treatment with angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) in type 1 DM or an ARB in type 2 DM have been shown to reduce retinopathy progression independent of blood pressure control.\textsuperscript{8,9} SOR \textit{B}

Patients with high lipids have more hard exudates and a higher risk of vision loss, but it is unclear if lipid control changes outcomes. SOR \textit{C}

REFERRAL

Work with an ophthalmologist to prevent vision loss:

- Complications of DR are vitreous hemorrhage (Figure 20-4), retinal detachment, and neovascular glaucoma. Each of these complications can result in devastating vision loss.

- Ophthalmologists will determine when peripheral retinal photocoagulation is indicated (Figure 20-5). Photocoagulation reduces the risk of severe visual loss by more than 50% with side effects of peripheral and night vision loss.\textsuperscript{14} SOR \textit{A}

Other surgical treatments, including vitrectomy, have been less successful.\textsuperscript{3}

PREVENTION

Prevent DR by preventing development of type 2 DM or tightly controlling type 1 or type 2 DM.

Screen patients with DM for DR based on national recommendations.\textsuperscript{10}

- Type 1 DM—Adults and children older than age 10 years: Screen for retinopathy 5 years after diagnosis and at regular intervals as recommended by an eye specialist.

- Type 2 DM—Screen for retinopathy at diagnosis and then annually.

Patients can be referred to an eye specialist, or screened using telemedicine or retinal photographs taken during outreach screenings or in primary care offices.\textsuperscript{11,12}

Mathematical models are being developed to individualize screening frequency. In one study, screening intervals ranged from 6 to 60 months (mean: 29 months). This resulted in 59% fewer visits than with fixed annual screening without compromising safety.\textsuperscript{13}

FOLLOW-UP

Once DR is diagnosed, frequency of examination is set by the ophthalmologist.

PATIENT EDUCATION

Preventing retinopathy by controlling diabetes and hypertension leads to better vision outcomes than any available treatment.\textsuperscript{3,14}

PATIENT RESOURCES

- National Eye Institute—\url{http://www.nei.nih.gov/health/diabetic/}. 
REFERENCES