A 51-YEAR-OLD MAN WITH DIABETIC RETINOPATHY

Emily Y. Chew, MD

BACKGROUND
A 51-year-old man presented to the Clinical Center of the National Eye Institute/ National Institutes of Health (Bethesda, MD) with a history of central vision loss that had occurred gradually and painlessly over the past 2 years. The rate of his vision loss has accelerated over the past 6 months. His central visual acuity varies during the day and also from day to day. Initially, he had been seen by his general ophthalmologist who suggested that no further treatment could be administered.

MEDICAL HISTORY
This patient has had type 2 diabetes mellitus and hypertension for 15 years and was diagnosed with Crohn's disease 32 years ago.

REVIEW OF SYSTEMS
He has had difficulties maintaining good glycemic control for the past 5 years. He reports he has been experiencing lethargy for the past 6 months. The patient has no gastrointestinal symptoms. He has no history of renal disease, including diabetic neuropathy.

FAMILY HISTORY
His maternal grandfather, sister, and brother also had type 2 diabetes. His grandfather became blind as a result of diabetic retinopathy.

SOCIAL HISTORY
The patient is a former policeman who is on disability because of his poor vision. He is married and has 3 children. He does not smoke and occasionally has an alcoholic drink.

OCULAR EXAMINATION
On examination, the patient’s best-corrected visual acuity was 20/200 in the right eye and 20/400 in the left. The results of slit lamp biomicroscopy were unremarkable, showing no evidence of neovascularization on the iris and minimal lens opacities. Dilated funduscopic examination revealed severe bilateral clinically significant macular edema with marked ischemia, retinal hard exudate exceeding the most severe Modified Airlie House standard photograph of retinal hard exudate, and high-risk proliferative diabetic retinopathy (Figures 1A and 1B).

INVESTIGATIONS
Fluorescein angiography was performed to evaluate the health of the retinal capillaries, which are necessary to guide laser photocoagulation for the diabetic macular edema. Results of the angiography demonstrated capillary nonperfusion throughout the posterior poles, indicating marked ischemia. The late frames of the angiography image showed marked leakage of fluorescein associated with the macular edema and also from abnormal proliferating retinal vessels.

LABORATORY INVESTIGATIONS
The patient was referred to the Internal Medicine Service to improve his medical status, which may play an important role in his ocular disease. His serum lipid panel revealed the following values: total serum cholesterol level of 421 mg/dL, 10.9 mmol/L (normal <200 mg/dL, <5.17 mmol/L); triglycerides 1272 mg/dL, 14.4 mmol/L (normal <150 mg/dL, <1.69 mmol/L); low-density lipoprotein (LDL) 201 mg/dL, 5.2 mol/L (normal 65–129 mg/dL, 1.68–3.34 mmol/L); and high-density lipoprotein (HDL) 45 mg/dL, 1.17 mmol/L (normal ≥35 mg/dL, ≥0.91 mmol/L). A thyroid panel revealed a thyroid-stimulating hormone (TSH) level of 53.2 µU/mL (normal 0.43–4.6 µU/mL) and a free thyroxine (FT4) level of 0.5 ng/dL, 6.5 pmol/L (normal 0.9–1.6 ng/dL, 11.6–20.6 pmol/L). The hemoglobin A1c level was 10.3% (normal 4.8–6.4%).

TREATMENT
Both eyes were treated (one eye per session) with focal laser photocoagulation. Over a period of 2 months,
both eyes were treated with scatter (panretinal) photocoagulation. The patient’s medical treatment for glycemic control was intensified by increasing his insulin dose to 30 U daily, and he began a diet and exercise program. He also began taking a daily dosage of 20 mg simvastatin orally for elevated serum lipid levels and 150 mg of levothyroxine orally for hypothyroidism.

**FOLLOW-UP**

Three months after the initiation of medical intervention and laser treatment, the patient’s visual acuity had improved to 20/160+1 OD and 20/125 OS. Funduscopic examination revealed considerable reduction in macular edema and retinal hard exudates in each eye and the proliferative retinopathy had responded well to the scatter photocoagulation (Figures 2A and 2B).

The serum lipid and thyroid panel values had improved, and the patient’s glucose control level was under much better control with a hemoglobin A1C of 7.1%. The serum lipid panel showed the following values: total serum cholesterol, 170 mg/dL (4.4 mmol/L); triglycerides, 527 mg/dL (6.0 mmol/L); LDL, 80 mg/dL (2.1 mol/L); and HDL, 34 mg/dL (0.9 mmol/L). A thyroid panel demonstrated improvement in TSH 0.36 µIU/mL and FT4 1.1 ng/dL (14.2 pmol/L).

Approximately 1 year after initiation of treatment, the patient’s lipid and thyroid panels continued to show a normal range of laboratory values. Funduscopic examination revealed further regression of retinal hard exudates in both eyes and subretinal fibrosis in the left eye (Figures 3A and 3B).

**DISCUSSION**

The importance of good glycemic control and management of dyslipidemia is demonstrated in this patient with severe diabetic retinopathy. However, because his macular function was poor (secondary to poor perfusion) and the degree of retinal hard exudate was severe, the patient’s visual acuity recovered to a level considered only as functional; he requires the aid of low vision devices. This limited recovery emphasizes the need for early detection and timely treatment of diabetic retinopathy. In a long-term follow-up study of patients treated in a clinical trial involving laser photocoagulation, more than 80% of the patients receiving follow-up care for 15 to 20 years maintained a vision of 20/40 or better. It is important for diabetics to know that they must have regular dilated eye examinations and that diabetic retinopathy can be effectively treated in at least 95% of cases.

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**REFERENCE**