We question whether our patient’s baseline anticoagulant therapy contributed to the unusually slow progression of vision loss. A few reports propose beneficial effects of anticoagulation and antiplatelet therapy in GCA-related vision loss. Buono et al² reported improvement in acuity and posterior ciliary artery blood flow with the addition of heparin sodium therapy in a patient with progressive visual loss despite corticosteroid therapy for 5 days. A retrospective study of 175 patients with GCA reported that patients taking aspirin had fewer cranial ischemic complications at admission and that fewer such complications developed, compared with patients not taking aspirin.³ The protective effect of aspirin therapy⁴ and improvement of vision and blood flow with heparin therapy⁵ imply that thrombosis may contribute to vision loss in patients with GCA. However, more studies are needed to validate the beneficial effects of anticoagulant therapy.

Visual loss secondary to choroidal ischemia has been previously described in GCA. One report described visual loss secondary to isolated choroidal nonperfusion over 1 to 2 days. The patient’s vision improved with high-dose corticosteroid therapy.⁶ Another article described 3 patients with vision loss from choroidal ischemia that progressed over the course of seconds, several hours, and 3 days, respectively.⁷

The visual decline in patients with GCA typically occurs quickly, over the course of seconds to days. In our patient, vision loss from choroidal ischemia secondary to GCA progressed over 24 days. Giant cell arteritis must be considered in older patients with slowly progressive vision loss.

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Autoimmune Retinopathy After Chronic Renal Allograft Rejection

Autoimmune retinopathy, such as carcinoma-associated retinopathy¹ and melanoma-associated retinopathy,² is characterized by retinal dysfunction and the presence of autoantibodies that can occur in the absence of malignancy.³ Herein, we report a case of presumed autoimmune retinopathy occurring after chronic renal allograft rejection.

Report of a Case. A 51-year-old man was referred to us on February 2, 2000. In 1982, he had undergone renal transplantation for chronic renal failure caused by glomerulonephritis that he had had since his childhood. In 1993, he developed chronic renal rejection and underwent a second renal transplantation. However, he developed chronic rejection a third time and has been undergoing continuous ambulatory peritoneal dialysis since June 1998. He noticed “flashing light” in both his eyes in December 1999 and nyctalopia immediately thereafter. Within the following 1 to 2 weeks, he noticed constriction of his visual field.

At the initial visit, his best-corrected visual acuity was 20/25 OD and 20/15 OS. Slitlamp biomicroscopy revealed no abnormalities. Funduscopic examination, fluorescein angiography, and indocyanine green angiography revealed questionably narrowed retinal blood vessels and diffuse atrophy of the retinal pigment epithelium (Figure 1). Goldmann visual field perimetry revealed enlargement of the Mariotte blind spot and a mild general visual field constriction (Figure 2A and B). Electroretinogram was unrecordable. Western blot demonstrated that his serum was immunoreactive for 50-, 60-, and 70-kD bovine retinal protein (Figure 2E). Results of immunohistochemistry revealed that the patient’s serum reacted against glomerulus of rat kidney. Magnetic resonance imaging of the brain and computed tomography of the chest showed no abnormalities.

Since his electrolyte balance could not be properly managed with continuous ambulatory peritoneal dialysis (HD) was introduced on March 4, 2000. Thereafter, he reported the recovery of the narrowed visual field and nyctalopia. Goldmann visual field perimetry showed the reduction of relative scotomas (Figure 2C and D). The antiretinal antibodies were undetectable (Figure 2E) on March 15, 2004. The electroretinogram in May 2004 showed reduced a- and b-wave amplitudes of maximal electroretinographic responses of both eyes, suggesting an improvement of visual function.

Comment. Renal rejection involves dynamic change of humoral mechanisms, and autoantibodies can be found in the patient’s serum.⁴ We consider that these autoantibodies were induced by chronic renal rejection. He underwent thorough and detailed examination of systemic carcinoma in January 2002 and in September 2003, and the findings were unremarkable.

Improvement in the subjective symptom and visual function was noted after the introduction of HD and coincided with the disappearance of the antiretinal antibody, suggesting that the antiretinal antibody is a cause of this pathologic condition and that the introduction of HD is related to its disappearance. In support of our hypothesis, it is generally accepted that HD ameliorates immunological reaction. Investigators⁵ previously reported the remission of autoimmune diseases after the introduction of HD. Notably, some patients with carcinoma-associated retinopathy also responded to corticosteroid treatment, resulting in an improvement in the visual field.¹ Our case report raises the possibility that
toantibody may be involved in previously unrecognized conditions.

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Figure 2. Goldmann perimetry visual fields in the right (A) and left (B) eye at the initial visit and after the introduction of hemodialysis (HD) in the right (C) and left (D) eyes. E, Western blot analysis of the bovine retinal extract with serum from the patient (dilution 1:1000) before and after the introduction of HD. Note the patient's serum reacts with 50-, 60-, and 70-kDa retinal proteins before HD. F, Immunostaining of paraffin-fixed rat kidney specimen with serum from the patient (dilution 1:100), visualized by horseradish peroxidase–conjugated rabbit anti–human IgG as the secondary antibody. Note that staining is observed in response to glomerulus of the rat kidney.