it is already diluted to the appropriate concentration for intraocular injection (2.4 mg/0.1 mL) and is sterilized by filtration. This preparation has a pH of 7.4, which confers physiological biocompatibility.

We hypothesize that such high drug concentrations in the globe lead to sudden change of the vitreous pH, thereby causing crystallization of the foscarnet. Nevertheless, the exact reason for crystal formation needs further investigations. Previous studies have shown no clinical signs of retinal or optic nerve toxic effects.

The crystallization of intravitreous ganciclovir in a case of CMV retinitis, causing damage to the retina and optic nerve, has also been reported. However, we found no sign of retinal or optic nerve toxic effects in the presence of foscarnet crystals.

The formation of foscarnet crystals may be a complication of intravitreal treatment in cases of CMV retinitis. Further studies are warranted to determine the exact mechanism of the formation of foscarnet crystals and their role in the management of CMV retinitis.

Sebastián Martínez-Castillo, MD
Cristina Martín-Lambíes, MD
Roberto Gallego-Pinazo, MD
J. Fernando Arévalo, MD, PhD
Manuel Díaz-Llopis, MD, PhD

Author Affiliations: University and Polytechnic Hospital La Fe (Drs Martínez-Castillo, Marín-Lambíes, Gallego-Pinazo, and Díaz-Llopis) and Faculty of Medicine, University of Valencia (Dr Díaz-Llopis), Valencia, Spain; Retina Division, Wilmer Eye Institute, Johns Hopkins University School of Medicine, Baltimore, Maryland (Dr Arévalo); and King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia (Dr Arévalo).

Correspondence: Dr Martínez-Castillo, University and Polytechnic Hospital La Fe, Bulevar Sur s/n, 46023, Valencia, Spain (s.martinez@comv.es).

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Scleritis Associated With Toxoplasmic Retinochoroiditis

Scleritis in conjunction with toxoplasmic retinochoroiditis has been reported rarely in the literature. We describe a case of toxoplasmic scleritis in a pregnant woman.

Report of a Case. A 24-year-old woman who was 7 weeks pregnant visited the eye clinic with a 4-day history of pain, redness, blurry vision, and floaters in the right eye. Her medical history was unremarkable; in particular, she had no history of immunocompromise or immunosuppressive therapy. Her ocular history was significant for myopia. She also reported being told she had a “scar” in her right eye several years prior by an ophthalmologist. Best-corrected visual acuities were 20/30 OD and 20/20 OS. On examination, there was pain in the right eye with right gaze. Ishihara color plates were 9/10 OU. Anterior segment examination findings for the right eye were remarkable for 2+ sectoral scleral injection temporally (Figure 1), faint scattered keratic precipitates, and 1+ cell in the anterior chamber. Intraocular pressures were 14 mm Hg OD and 15 mm Hg OS. Dilated funduscopy revealed an inferotemporal chorioretinal lesion with central retinitis and hyperpigmented edges as well as overlying vitreous debris. Adjacent was an area of active, elevated, white retinochoroiditis with overlying vitreous debris (Figure 2). Findings from anterior segment and funduscopic examinations of the left eye were unremarkable.

Treatment was started with prednisolone acetate, 1%, 4 times a day in the right eye. Owing to the contraindication of sulfa-based medications as well as pyrimethamine in pregnancy, oral azithromycin therapy with a 1-g loading dose followed by 500 mg/d was initiated in conjunction with her obstetrician. Findings from serologic studies for human immunodeficiency virus, rapid plasma reagin, Lyme antibody, and QuantiFERON for tuberculosis were all negative. Serum was negative for toxoplasmosis IgM antibody, and the IgG level was elevated at 2.81 IU/mL (reference range, 0.0-0.9 IU/mL).

Four days after antibiotic therapy was initiated, treatment with oral prednisone was started for the active vitritis. The anterior chamber reaction quickly subsided and the prednisolone acetate treatment was discontinued. During the following month, the area of active retinochoroiditis decreased in size and developed pig-
Comment. Toxoplasmosis is believed to be the most common cause of posterior uveitis. Scleritis in association with toxoplasmic retinochoroiditis is an uncommon entity. Accordingly, in a review of 243 patients with scleritis, no patient was reported to have toxoplasmic infection as a cause; furthermore, 37% had a systemic rheumatologic disorder, only 7% had an infection, and 44% had an associated medical disorder. Herpes zoster virus was the most commonly reported infectious cause and rheumatoid arthritis was the most common rheumatic disease.

In cases of toxoplasmic scleritis, the inflammatory response is believed to extend from the active retinochoroiditis to involve the overlying sclera. Accordingly, pathologic specimens from eyes that were enucleated secondary to severe toxoplasmic scleritis displayed granulomatous inflammation of the retina, uvea, and episclera with associated retinal thickening. In many cases, the entire sclera extending outward from the retinitis was inflamed; however, in some cases, a region of uninfamed sclera separated the active scleritis from the underlying retinitis.

Isolated toxoplasmic retinochoroiditis can rapidly spread and lead to severe permanent vision loss when treated with steroids alone. In our patient with scleritis, a dilated fundus examination revealed an area of typical toxoplasmic retinochoroiditis, allowing for prompt diagnosis and treatment with appropriate antibiotic therapy. The patient improved and maintained excellent visual acuity. This patient's course underscores the importance of a complete examination in cases of scleritis, including a dilated fundus examination, to rule out an infectious retinochoroiditis in association with the scleritis.

Danielle S. Rudich, MD  
Pawan Bhatnagar, MD

Author Affiliations: Department of Ophthalmology, Mount Sinai School of Medicine, New York (Dr Rudich), and Department of Ophthalmology, Albany Medical College and Retina Consultants PLLC, Slingerlands (Dr Bhatnagar), New York.

Correspondence: Dr Bhatnagar, Retina Consultants PLLC, 1220 New Scotland Rd, Ste 201, Slingerlands, NY 12159 (bhat3@hotmail.com).

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Unusual Paraneoplastic Cause of Vision Loss: Combined Paraneoplastic Cone Dystrophy and Optic Neuropathy

Paraneoplastic cone dystrophy is a very rare condition with only a few cases reported in the literature. Paraneoplastic optic neuropathy is also a rare cause of cancer-associated visual disturbance. We describe a patient with subacute bilateral vision loss resulting from combined optic neuropathy and cone dystrophy of paraneoplastic origin (occult lung small cell carcinoma). The patient's serum contained antibodies reactive with a novel 42-kDa retinal antigen.

Report of a Case. A 55-year-old man had slight photophobia, photopsias, progressive loss of color perception, and slightly diminished visual acuity in both eyes over a month. Seven days before his initial visit, he developed massive painless vision loss in both eyes over a few hours. His medical history revealed active smoking and alcohol abuse as well as the removal of in situ epidermoid oropharyngeal carcinoma 1 month earlier.

On examination, best-corrected visual acuity was counting fingers at 2 ft OU. Color vision was abolished in both eyes. Slitlamp biomicroscopy showed 2+ cells in the vitreous of both eyes. Fundus examination revealed slightly pallid, swollen optic discs and narrowed arteries in both eyes (Figure 1A). Lumbar puncture revealed an elevated level of cells (9/µL) and proteins (855 mg/L) in cerebrospinal fluid with normal opening pressure. Brain magnetic resonance imaging...