

- Armstrong B, Fecarotta C, Ho AC, Baskin DE. Evolution of severe lightning maculopathy visualized with spectral domain optical coherence tomography. *Ophthalmic Surg Lasers Imaging*. 2010;41(suppl):S70-S73.
- Rivas-Aguino PJ, Garcia RA, Arevalo JF. Bilateral macular cyst after lightning visualized with optical coherence tomography. *Clin Experiment Ophthalmol*. 2006;34(9):893-894.
- Gardner TW, Ai E, Chrobak M, Shoch DE. Photic maculopathy secondary to short-circuiting of a high-tension electric current. *Ophthalmology*. 1982;89(7):865-868.
- Vicuna-Kojchen J, Amer R, Chowers I. Reversible structural disruption of the outer retina in acute welding maculopathy. *Eye (Lond)*. 2007;21(1):127-129.
- dell'Omo R, Konstantopoulou K, Wong R, Pavesio C. Presumed idiopathic outer lamellar defects of the fovea and chronic solar retinopathy: an OCT and fundus autofluorescence study. *Br J Ophthalmol*. 2009;93(11):1483-1487.

Crystallization After Intravitreal Foscarnet Injections

Foscarnet sodium is a pyrophosphate analog that interferes with the binding of the diphosphate to the viral DNA polymerase of cytomegalovirus (CMV), herpes simplex virus, varicella-zoster virus, and human immunodeficiency virus. Given its nephrotoxicity, intravenous administration of foscarnet is generally limited to ganciclovir sodium-resistant viral strains or dose-limiting neutropenia. Intravitreal foscarnet has been successfully used for CMV retinitis,¹⁻³ avoiding the systemic effects.

The adverse effects of intravitreal foscarnet injection are mostly related to the procedure rather than the drug itself, including retinal detachment, vitreous hemorrhage, endophthalmitis, and cataract.

Herein, we report crystal formation as a rare condition after intravitreal injections of foscarnet.

Report of a Case. A 49-year-old woman, diagnosed as having aplastic anemia secondary to immunosuppressive therapy for liver transplantation, had floaters and progressive painless decrease of vision in both eyes for 1 week. She was not receiving any systemic medication and her renal function was normal.

Visual acuity was 20/400 OD and counting fingers OS. Anterior segment examination findings were unremarkable. Fundus examination showed a whitish necrotizing plaque with mild vitreous haze in the right eye as well as perivascular exudates and retinal hemorrhages with mild vitreous haze in the left eye. A diagnostic anterior chamber tap was performed, confirming high loads of CMV DNA by polymerase chain reaction. There was no evidence of systemic CMV infection.

Ganciclovir therapy was not administered owing to the aplastic anemia. Foscarnet sodium treatment was initiated with both systemic (9 g/d) and local (2.4 mg/0.1 mL twice per week) administration. Two weeks later, 1 intravitreal injection per week was administered.

Two months later, after 11 intravitreal foscarnet injections had been administered, visual acuity was 20/32 OU. Fundus examination showed crystal formation in the vitreous anterior to the retina in the right eye (**Figure**). Spectral-domain optical coherence tomography showed foscarnet crystals on both the posterior hyaloid and the internal limiting membrane (Figure). Because the formation of foscarnet crystals did not correspond to any damage to the retina, weekly intravitreal injections of foscarnet were continued.

Comment. The treatment of CMV retinitis with intravitreal injections of foscarnet arose from the need to achieve an elevated intraocular antiviral level while avoiding the frequent, serious adverse effects of systemic administration.⁴

Few noncontrolled studies of intravitreal foscarnet injections have been published.¹⁻³ In the largest series, 11 patients experienced successful induction therapy (6 injections of 2400 µg given at 72-hour intervals) followed by weekly maintenance injections. Reactivation of the retinitis occurred in 33.3% of patients within 20 weeks.

For the injection, the commercial preparation of foscarnet for intravenous infusion is used directly because

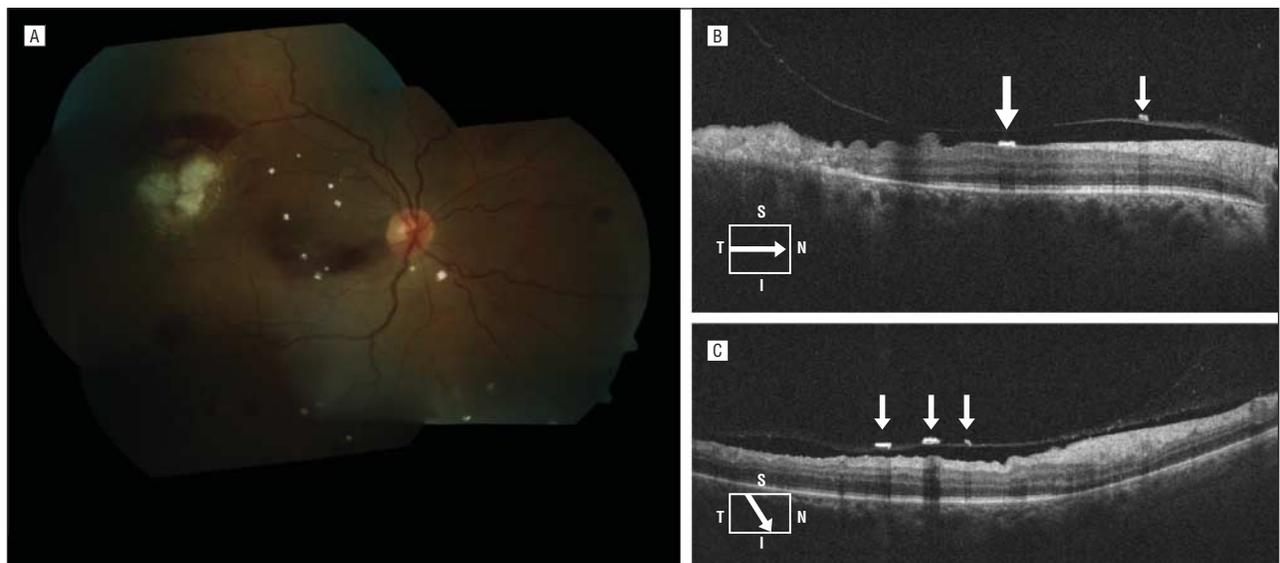


Figure. Retinography of the right eye shows white reflective foscarnet crystals anterior to the retina (A), and spectral-domain optical coherence tomographic images show hyperreflective crystals (arrows) on the internal limiting membrane and the detached posterior hyaloid (B and C). I indicates inferior; N, nasal; S, superior; and T, temporal.

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.⁵

The crystallization of intravitreal ganciclovir in a case of CMV retinitis, causing damage to the retina and optic nerve, has also been reported.⁶ 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.

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Financial Disclosure: None reported.

1. Diaz-Llopis M, Chipont E, Sanchez S, España E, Navea A, Menezo JL. Intravitreal foscarnet for cytomegalovirus retinitis in a patient with acquired immunodeficiency syndrome. *Am J Ophthalmol.* 1992;114(6):742-747.
2. Lieberman RM, Orellana J, Melton RC. Efficacy of intravitreal foscarnet in a patient with AIDS. *N Engl J Med.* 1994;330(12):868-869.
3. Diaz-Llopis M, España E, Muñoz G, et al. High dose intravitreal foscarnet in the treatment of cytomegalovirus retinitis in AIDS. *Br J Ophthalmol.* 1994;78(2):120-124.
4. Engstrom RE Jr, Holland GN. Local therapy for cytomegalovirus retinopathy. *Am J Ophthalmol.* 1995;120(3):376-385.
5. Wong R, Pavesio CE, Laidlaw DA, Williamson TH, Graham EM, Stanford MR. Acute retinal necrosis: the effects of intravitreal foscarnet and virus type on outcome. *Ophthalmology.* 2010;117(3):556-560.
6. Choopong P, Tesavibul N, Rodanant N. Crystallization after intravitreal ganciclovir injection. *Clin Ophthalmol.* 2010;4:709-711.

Scleritis Associated With Toxoplasmic Retinochoroiditis

Scleritis in conjunction with toxoplasmic retinochoroiditis has been reported rarely in the literature.¹ We describe a case of toxoplasmic scleritis in a pregnant woman.



Figure 1. Anterior scleritis of the temporal aspect of the right eye.

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 fundoscopic examination of the right eye 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 fundoscopic 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.^{2,3} 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-