



Figure 2. The left eye 27 years after corneal tattooing showing significant lightening of corneal pigment compared with original postoperative photographs as well as migration of tattoo pigment into the central 4-mm clear zone (A), and photographs taken after phototherapeutic keratectomy of the left eye in a 5-mm-diameter central zone and reapplication of dye to the peripheral cornea (B and C).

ment in that area, and dye was reapplied to the peripheral cornea to better match the hue of the contralateral iris (Figure 2B and C). The patient underwent the procedure without complication. This allowed her to continue wearing a tinted CL with an improved cosmetic appearance and functional results.

Comment. In this case, the excimer laser was used in a novel fashion to ablate central corneal tissue where dye had migrated from the previous tattooing procedure 27 years earlier. This procedure allowed for the creation of a precisely circular central clear corneal “pupil,” enhancing both cosmesis and light passage to maximize the patient’s residual vision. We believe that this method offers a simple and effective technique to clear the areas of aberrant dye that is known to migrate over time following corneal tattooing.

Kevin M. Cronin, BA
Jacob C. Meyer, MD
Keith A. Walter, MD

Author Affiliations: Wake Forest Eye Center, Wake Forest School of Medicine, Winston-Salem, North Carolina.

Correspondence: Mr Cronin, Wake Forest Eye Center, Wake Forest Baptist Medical Center, Medical Center Boulevard, Winston-Salem, NC 27157-1033 (kcronin44@gmail.com).

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Optical Coherence Tomography and Autofluorescence Findings in Photic Maculopathy Secondary to Distant Lightning Strike

Ophthalmic injuries due to lightning occur mainly from direct or indirect transmission of electric charge, resistance-induced heat, or heat-induced shock wave.¹ Most reports of lightning-induced maculopathy quote direct or indirect electric transmission as the cause and associate maculopathy with oculofacial injuries and sometimes loss of consciousness.¹⁻³ However, a high-voltage electric current can also induce photic retinopathy without contribution from the electric charge.⁴ We report a case of photic retinopathy caused solely by viewing a lightning strike.

Report of a Case. A 40-year-old healthy man visited our outpatient clinic with bilateral blurring of vision for 10 days, after watching a lightning strike about 2 m away through an open window. He was not using a computer or telephone at that moment and was not holding or leaning out of the window. He immediately noted a yellow after-image, but he experienced vision decline only after a day. There was no history of smoking, sun gazing, or exposure to a solar eclipse or welding arc. On examination, best-corrected visual acuity was 20/70 N10 OU. Eyelids, adnexa, and anterior segments—including the pupillary reactions—were unremarkable bilaterally. Both fundi showed a faint yellow spot at the central fovea (Figure 1 A and B). Color vision, contrast sensitivity, and visual fields (Humphrey 10-2, macular program) were unaffected. The Amsler grid test revealed bilateral metamorphopsia. Spectral-domain optical coherence tomography (OCT; Topcon 1000) showed central hyperreflective echoes and disruption of the inner segment–outer segment junction in each eye (Figure 1 C and D). Fundus camera–based autofluorescence (FAF; Zeiss Vi-

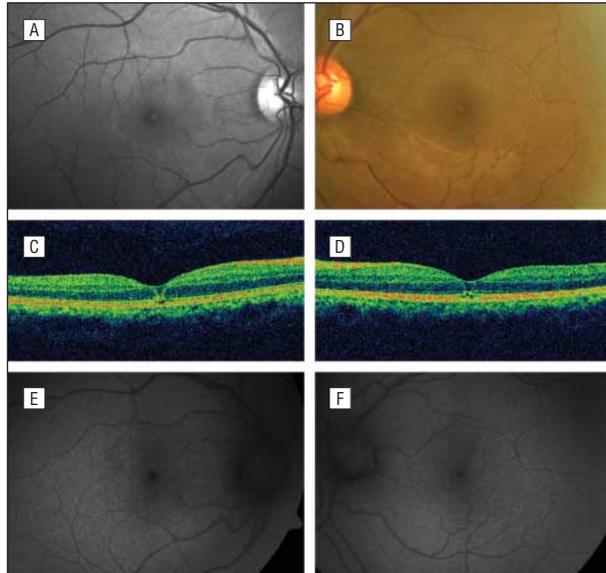


Figure 1. Lightning-induced photic maculopathy at the initial visit. A red-free photograph of the right eye (A) and a color photograph of the left eye (B) demonstrate a pale yellow spot with a dark halo at the foveola. Spectral-domain optical coherence tomography (horizontal 6-mm scan) shows a wedge-shaped pattern of hyperreflectivity in the photoreceptor layer with interruption of the external limiting membrane and inner segment-outer segment junction, more prominent in the right eye (C) than in the left eye (D). The central macular thickness is 130 μm OD (C) and 124 μm OS (D). Fundus camera-based autofluorescence shows central dark spots corresponding to the outer lamellar defects in the right (E) and left (F) eyes, with increased autofluorescence signal in the perifoveolar macula.

supac 450 Plus IR), with excitation and barrier filters set at bandwidths of 510 to 580 nm and 650 to 735 nm, respectively, revealed bilateral increased central hypoautofluorescence and decreased parafoveal hypoautofluorescence (Figure 1E and F). After 1 month, best-corrected visual acuity improved to 20/50 OD and 20/40 OS; the inner segment-outer segment disruption persisted in each eye. By 12 months, best-corrected visual acuity had improved to 20/25 N6 OD and 20/20 N6 OS. Fundi showed resolution of the yellow spot into a faint ring in each eye (Figure 2A and B). Optical coherence tomography revealed minimal inner segment-outer segment defects (Figure 2C and D); the macular thickness was essentially unchanged. The Amsler grid test showed minimal distortion in each eye. Imaging by FAF revealed normalization of the macular autofluorescence pattern in each eye (Figure 2E and F).

Comment. Photic maculopathy, essentially a photochemical reaction, differs from thermal foveolar burns typically attributed to lightning. Although both primarily affect macular retinal pigment epithelium, thermal energy affects the entire retinal thickness.⁴ Lightning maculopathy has been reported to consist of bilateral foveal cystic changes that degenerate into foveal atrophy and pigmentary disturbances over time.¹⁻³ Our patient had more subtle outer retinal involvement, documented by spectral-domain OCT and FAF imaging. The hyperreflectivity and interruption of outer retinal layers, as seen on OCT in our patient, parallel the acute and chronic changes observed in welding arc and solar retinopathy.^{5,6} dell'Omo et al⁶ have demonstrated similar OCT and

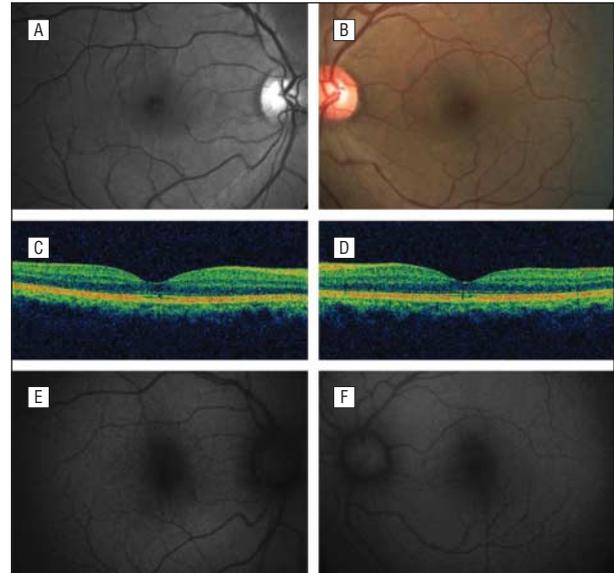


Figure 2. Lightning-induced photic maculopathy at the final follow-up. One year after the initial visit, the central yellow spot has attenuated into a mottled, broken ring in the right (A) and left (B) eyes. Spectral-domain optical coherence tomography shows resolution of central hyperreflectivity and restoration of the external limiting membrane, and the inner segment-outer segment junction shows a minimal residual central defect in the right (C) and left (D) eyes. Autofluorescence images of the right (E) and left (F) eyes reveal normalization of the signal attenuation patterns at the central macula.

FAF findings in presumed chronic solar retinopathy. They attributed the decreased foveolar signal in solar retinopathy to photoreceptor death, an event unlikely in our patient, who experienced resolution of both OCT and FAF abnormalities along with visual recovery. A plausible mechanism in our case could be increased absorption of FAF signal at the foveola due to accumulation of photoreceptor debris. The increased perifoveal FAF signal was reported to result from increased lipofuscin accumulation or decreased luteal pigment.⁶ The former mechanism was likely in our case as well, with lightning-induced acute metabolic stress being the cause for transient accumulation of the fluorophore. This article alerts physicians to the potential for phototoxic effects from viewing lightning at close range and adds another dimension to the spectrum of lightning maculopathy.

Dhananjay Shukla, MS, MAMS
Abhishek Sharan, MS
Ramesh Venkatesh, MS

Author Affiliations: Retina-Vitreous Service, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, Madurai, Tamil Nadu, India.

Correspondence: Mr Shukla, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, 1 Anna Nagar, Madurai 625 020, Tamil Nadu, India (daksh66@gmail.com).

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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

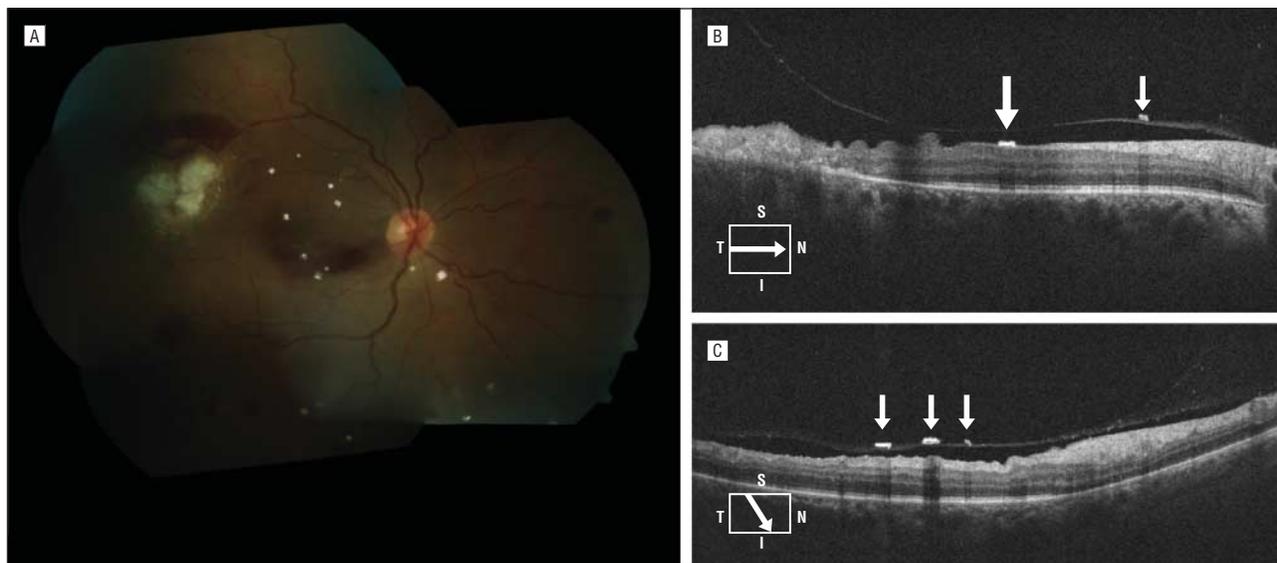


Figure. Retinography of the right eye shows white reflective foscarnet crystals anterior to the retina (A), and spectral-domain optical coherence tomographic images show hyperreflective crystals (arrows) on the internal limiting membrane and the detached posterior hyaloid (B and C). I indicates inferior;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