SOLAR RETINOPATHY: A CASE REPORT

The human eye is constantly exposed to sunlight and artificial lighting. Therefore the eye is exposed to UVB (280-315nm), UVA (315-380 nm), and visible light (380-780 nm). Light is transmitted through the eye and then signals the brain directing both sight and circadian rhythm. Therefore light absorbed by the eye must be benign. Damage to the young and adult eye by intense ambient light is avoided because the eye is protected by a very efficient antioxidant system. There are protective pigments such as the kynurenines, located in the human lens, and melanin, in the uvea and retina, which absorb ambient radiation and dissipate its energy without causing damage. In addition, photo-protective eyewear can help in reducing lifetime exposure to harmful UV and blue-violet light.

Dr. Tomas Jaeschke
Ophthalmologist.
Universidad Del Salvador,
Buenos Aires, Argentina.

Introduction

Solar retinopathy is the retinal damage that results from exposure to solar radiation.1 Patients with solar retinopathy classically have a history of sun exposure through religious ritual participation,[2] solar eclipse viewing without proper precautions,[3,4] or sunbathing,[5] or from mental disturbances via drug intoxication or schizophrenia.[6]

Pathogenesis

Solar retinopathy occurs primarily through a photo-oxidative pathway rather than by direct thermal injury.[7]

The incident thermal damage resulting from looking at the sun through an adaptive pupil is far less than the threshold for detectable damage through ophthalmoscopy.[8]

The ozone layer filters the shortest wavelengths of ultraviolet light (UVC <280 nm), while the anterior segment of adult eye absorbs light in the UVB spectrum (280-315 nm) and the UVA spectrum (315-380 nm).[9-11]

At the other end of the light spectrum, the aqueous anterior chamber absorbs the longer wavelength infrared (IR) B and C light (1,400-10,000 nm).[7]

Although these structures absorb most of the harmful light spectrum, visible (380-780 nm) including highly energetic blue-violet light (415-455 nm) and near IR (IRA, 700-

KEYWORDS

solar radiation, solar retinopathy, UV, UVA, UVB, UVC, blue-violet light, chronic light exposure, photoxicity, sun protection, prevention, solar eclipse, retina, RPE, photo-oxidative stress, Crizal® Prevencia™

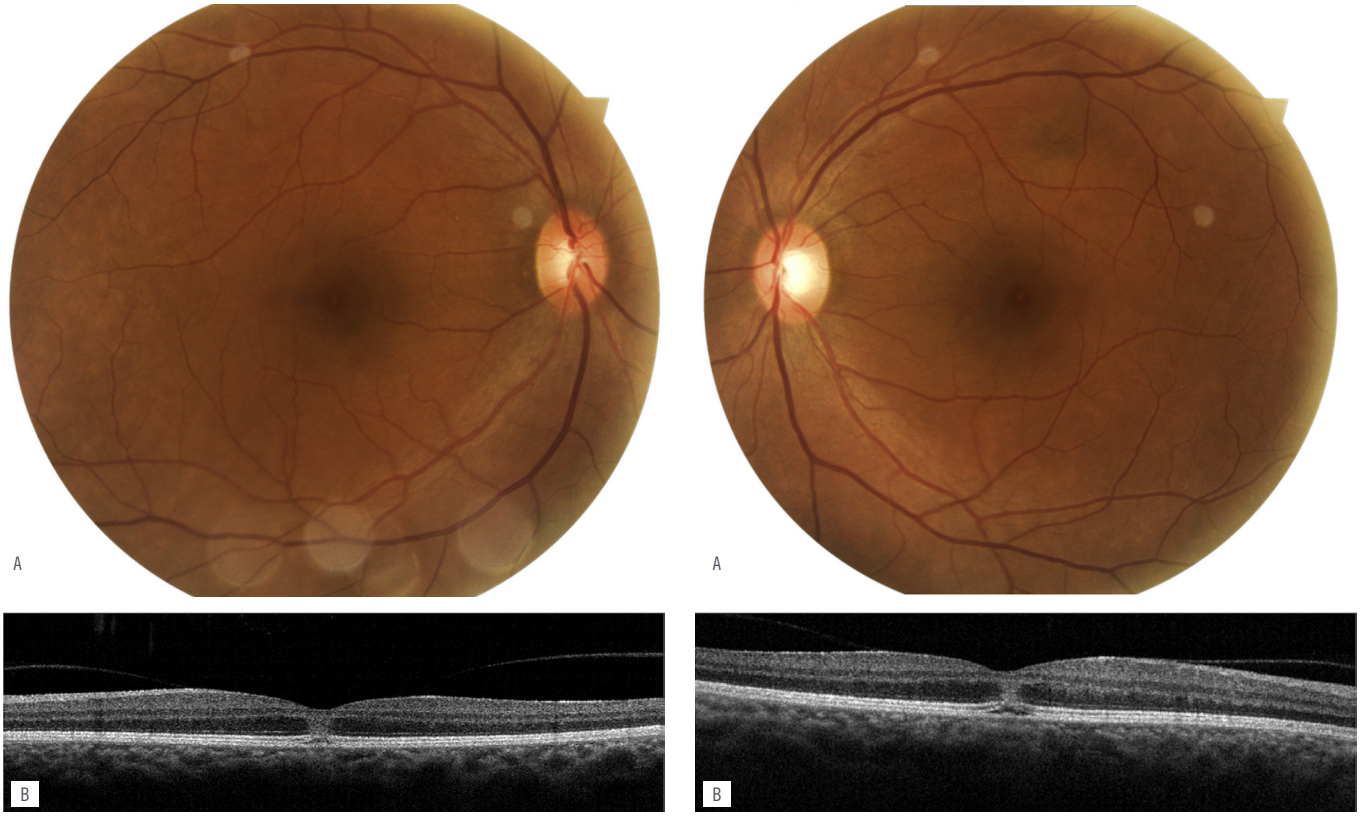

FIG. 1| Images showing a) retinography with yellow lesions at both foveas. b) oct image with disruption of the inner and outer segments of the photoreceptor layers.

1,400 nm), it can still pass through the ocular media and converge on the retina under absorption by the photoreceptors and lipofuscin-containing RPE-retinal pigment epithelial cells.[7]

Phototoxicity and Anatomic Damage

This phototoxicity, mainly from the higher-energy UVA and the shorter wavelengths of visible light (blue-violet light, 415-455 nm), leads to generation of reactive oxygen species and subsequent oxidative damage to these epithelial cells and the surrounding photoreceptors.[12-14]

Histopathological studies have confirmed that both the RPE layer and the outer segments of the photoreceptor layer are the most susceptible to damage.[15,16]

Specifically, the primary lesion seems to occur in the melanosome-containing RPE layer, followed by subsequent photoreceptor damage, likely secondary to disruption of the supportive RPE.[17]

We know that there are two mechanisms of light damage with slightly different absorption or action spectra but both peaking in the visible blue-violet. For relatively low intensity and very long exposures we see what is described as type I damage which appears to result from absorption within the light-sensitive cells and short wavelength or blue cones seem to be the most sensitive.[18,19,20]

By contrast for relatively high intensity shorter exposures we recognize type II damage whereby the primary damage seems to occur in the RPE-retinal pigment epithelium[21] and is thought to be associated with absorption by lipofuscin.

Case report

A woman presented with a 7-day history of bilateral central scotoma following [5–7] min of direct sun-gazing.

On examination, visual acuity was reduced to 20/100 in both eyes. Slit-lamp biomicroscopy showed discrete yellow lesions at both foveaa. Optical coherence tomographyb

> « Solar retinopathy is the retinal damage that results from exposure to solar radiation »

(OCT) scanning revealed corresponding retinal pigment epithelial (RPE) defects.

The patient was managed conservatively with regular follow-up and OCT scanning.

The patient's symptoms progressively improved over the following months without treatment. Almost a year later, her symptoms had decreased in both eyes, but a central scotoma persisted. Visual acuity returned to 20/60 in both eyes. OCT changes in both eyes improved over time; however, central RPE defects persisted.

She did not attend further follow-up after this point.
Discussion

Das et al. (1956) reported cases among Punjabis who look at the sun for religious reasons. Pittar (1943) reported one case in an anti-aircraft gunner firing at an enemy plane flying out of the sun, and fighter pilots performing the same maneuver have suffered similar consequences. Irvine (1945) has recalled the case of a seaman "steering into the sun's eye.» Bates (1920) advocated prolonged sun-gazing as the treatment of myopia, with disastrous results.
The warnings made by Knudtzon (1948) need repeating, especially as they bear on the false sense of protection afforded by dark glasses, red glass filters, smoked glass, and overexposed negative films. The only safe way of observing the sun, the method used by astronomers, is to project the sun's image on to a screen. For the amateur a pin-hole and sheet of matt paper held in the shade behind it will suffice (Gilkes et al., 1961).

How to protect your eyes
The commercially available solar shades however are highly suitable for solar viewing. First, a high light absorption and reflection is obtained, which allows a comfortable observation of the sun globe. Furthermore, a virtually linear absorption of all ultraviolet, visible and infrared wavelengths was found, which indicates that gazing at the sunlight is safe.

With Crizal® Prevencia™ lenses, Essilor has turned photo-protective concept into a reality in a clear (non-tinted) everyday lens. These lenses reduce exposure to ultraviolet (UV) light — coming from in front or reflecting off the back surface of lenses and they attenuate the harmful wavelengths of blue-violet light. Because they reduce (but don't fully block) transmission of just a narrow band of blue-violet light (415-455 nm), excellent color transmission, as well as transparency, are maintained, providing superior clarity of vision. Because the damaging effects of blue-violet light are cumulative, wearing Crizal® Prevencia™ lenses may help protect the eye by reducing lifetime exposure to harmful UV and blue-violet light.

Conclusions
Observing a solar eclipse can cause permanent eye damage.

In the case of accidental solar retinopathy it is difficult to suggest any protective measure other than greater public awareness of the dangers involved.

More effective methods of prevention are advocated and it is urged that public awareness of the dangers be aroused. The only commendable filtering devices for this purpose are the commercially available solar shades or the aluminum-coated Mylar foil. •

KEY TAKEAWAYS

- The human eye is constantly exposed to sunlight and artificial lighting.
- Therefore the eye is exposed to UVB (280-315nm), UVA (315-380 nm), and visible light (380-780 nm).
- Chronic light exposure to certain wavelengths may lead to phototoxicity and anatomic damage
- As such, exposure to solar radiation was evidenced to causes retinal damage and solar retinopathy
- Protective pigments located in the human lens, uvea and retina, absorb ambient radiation and dissipate its energy without causing damage.
- Photo-protective eyewear can help in reducing lifetime exposure to harmful UV and blue-violet light.

REFERENCES

1. Gass JDM. Stereoscopic Atlas of Macular Diseases: Diagnosis and Treatment. Vol. 4. St. Louis, MO; Mosby; 1997:760-763.
2. Das T, Nirankari MS, Chaddah MR. Solar chorioretinal burn. Am J Ophthalmol. 1956;41:1048-1053.
3. Agarwal LP, Malik SR. Solar retinitis. Br J Ophthalmol. 1959;43:366-370.
4. MacFaul PA. Visual prognosis after solar retinopathy. Br J Ophthalmol. 1969;53:534-541.
5. Ridgway AE. Solar retinopathy. BMJ. 1967;3:212-214.
6. Anaclerio AM, Wicker HS. Self-induced solar retinopathy by patients in a psychiatric hospital. Am J Ophthalmol. 1970;69:731-736.
7. Glickman RD. Ultraviolet phototoxicity to the retina. Eye Contact Lens. 2011;37:196-205.
8. White TJ, Mainster MA, Wilson PW, et al. Chorioretinal temperature increases from solar observation. Bull Math Biophys. 1971;33:1-17.
9. Boettner EA, Wolter JR. Transmission of the ocular media. Invest Ophthalmol Vis Sci. 1962;1:776-783.
10. Dillon J, Zheng L, Merriam JC, et al. Transmission spectra of light to the mammalian retina. Photochem Photobiol. 2000;71:225-229.
11. Sliney DH. How light reaches the eye and its components. Int J Toxicol. 2002;21:501-509.
12. Davies S. Elliot MH, Floor E, et al. Photocytotoxicity of lipofuscin in human retinal pigment epithelial cells. Free Radic Biol Med. 2001;31:256-265.
13. Jain A, Desai RU, Charalel RA, et al. Solar retinopathy comparison of optical coherence tomography (OCT) and fluorescein angiography (FA). Retina. 2009;29:1340-1345.
14. Chen JC, Lee LR. Solar retinopathy and associated optical coherence tomography findings. Clin Exp Optom. 2004;87:390-393.
15. Tso MO, La Piana FG. The human fovea after sungazing. Trans Am Acad Ophthalmol Otolaryngol. 1975;79:788-795.
16. Hope-Ross MW, Mahon GJ, Gardiner TA, Archer DB. Ultrastructural findings in solar retinopathy. Eye. 1993;7:29-33.
17. Ham WT, Mueller HA, Ruffolo JJ Jr, Clarke AM. Sensitivity of the retina to radiation damage as a function of wavelength. Photochem Photobiol. 1979;29:735-743.
18. Noell WK, Walker W, Kang B & Berman S (1966): Retinal damage by visible light. Invest Ophthalmol 5: 450–473.
19. Wu J. et al. Photochemical Damage of the Retina. Surv Ophthalmol 2006. 51 (5): 461-481
20. Sperling HG & Johnson C (1980): Differential spectral photic damage to primate cones. Vision Res 20: 1117–1125.
21. Ham WT, Ruffolo JJ, Mueller HA, Clarke AM & Moon ME (1978): Histologic analysis of photochemical lesions produced in rhesus retina by short-wavelength light. IOVS 17: 1029–1035.