Morphological Changes from Acute to Chronic Photic Retinopathy

Neha Arora¹, Aniket Patel¹, Ashish Kumar Ahuja¹
¹Sant Parmanand Hospital, Civil Lines, New Delhi, India
²Northern Railway Central Hospital, New Delhi, India

Summary

Photic Retinopathy is a photochemical retinal damage which may or may not produce permanent visual acuity loss. We are reporting 5 eyes of 3 patients, 1 of which is acute solar retinopathy, 2 eyes are chronic solar retinopathy and the other 2 eyes are chronic welder’s retinopathy showing morphological changes in photic retinopathy from the acute to chronic stage. The patient with acute solar retinopathy was successfully treated with oral prednisolone with complete regain of vision.

Keywords: Photic retinopathy, Acute solar retinopathy, Welder’s retinopathy, Photochemical damage

Introduction

Photic retinopathy, also known as foveomacular retinitis or solar retinopathy, is damage to the eye’s retina, particularly the macula, from prolonged exposure to solar radiation or other bright light, e.g., lasers or arc welders. Normal anatomical structures such as the cornea absorbs and filters the shortest wavelengths of UV light (UV-C<280nm), while the adult lens predominantly absorbs light in the UV-B spectrum (280-320) and part of the UV-A spectrum (315-440) less than 365nm. The aqueous in anterior chamber absorbs the longer wavelength IR B and C light (1400-10000). Although these structures absorb most of the light spectrum, the longer wavelength end of UV- A (365-440nm), visible (400-700nm) and near IR (IR A 700-1400nm) light can still pass through the ocular media and converge on retina and undergo absorption by photoreceptor and lipofuscin containing retinal pigment epithelium. This phototoxicity, mainly from the higher energy UV A and shorter wavelength of visible light, leads to generation of reactive oxygen species and subsequent oxidative damage to these epithelial cells and the surrounding photoreceptors.

CASE 1

A 22 year old female, an archer, came with the complaints of diminution of vision in the right eye, persistent dark spot near the centre of vision and persistence of after image since 1 week. There was history of targeting the sun during archery for 2-3 minutes, one week back. On examination, visual acuity was reduced in the right eye to 6/18 and preserved in the left eye as 6/6. Color vision was normal in both the eyes. Ophthalmoscopic examination of the fundus revealed a single hypopigmented patch in the centre of the fovea in the right eye with absent foveal reflex whereas fundoscopic examination was normal in the left eye. The SD-OCT revealed increased foveal hyper-reflectivity (rod shaped, full thickness) extending from outer segments of photoreceptor and the RPE to the inner layer of retina in the right eye (Figure 1). The patient was put on prednisolone in tapering dose along with ranitidine 150 mg.

After 2 weeks follow up, vision in the right eye improved from 6/18 to 6/9. The ophthalmoscopic examination of fundus revealed dull foveal reflex in the right eye. SD-OCT (after 2 weeks) became normal with normal macular thickness. The ophthalmoscopic examination of fundus after 3 weeks revealed normal macula with dull foveal reflex in the right eye. The SD-OCT (after 3 weeks) revealed hyperreflectivity at fovea with hypo-reflectivity between internal limiting membrane and retinal pigmentary epithelium (RPE) and irregularity of RPE with normal macular thickness (Figure 2).

After 6 weeks follow up, the ophthalmoscopic examination of fundus revealed hypopigmented patch with dull foveal reflex. The SD-OCT (after 6 weeks) revealed hypopigmented cystic spaces in the inner retinal layers, irregularities at the level of ILM and RPE with normal macular thickness (Figure 3).

As we know from the literature that over a period of time, it may resolve with no residual defect or the lesion will restrict to the outer retinal layers.
CASE 2
A 55 year old female patient (Tailor), came with the complaints of diminution of vision in both the eyes since 5 years. There was prior history of seeing solar eclipse in childhood using the mirror.

On examination, visual acuity was 6/60 in the right eye and 6/36 in the left eye. Best corrected visual acuity was 6/18 in both the eyes.

Slit lamp examination revealed nucleus sclerosis grade 2 with early posterior subcapsular cataract with peripheral cortical cataract in the right eye and nuclear sclerosis grade 2 with early peripheral cortical cataract in the left eye. Ophthalmoscopic examination of fundus revealed depigmented macular lesion with dull foveal reflex in both the eyes.

The SD-OCT revealed a rectangular shaped outer retinal hole restricted to the outer retina specifically extending from inner border of RPE line up to the ELM in both the eyes (Figure 4).

Discussion
Photic retinopathy is a generalised term used for retinal damage produced by light. The aetiology of photic retinopathy includes Sun Gazing (>90sec gazing exceeds threshold of retinal damage), Photosensitizing Drugs like tetracycline, psoralen, hydrochlorothiazide, furosemide, allopurinol, benzodiazepine, fluphenazine, use of Welding ARC without appropriate protection and may even be produced by the prolonged use of Operating Microscope. Photic retinopathy occurs primarily through a photo-oxidative pathway rather than direct thermal injury. Normal anatomical structures such as the cornea absorbs and filters the shortest wavelengths of the UV light (UV-C <280nm),
while the adult lens predominantly absorbs light in the UV-B spectrum (280-320) and part of the UV-A spectrum (315-440). At the other end of the light spectrum, the aqueous in anterior chamber absorbs the longer wavelength IR-B & C light (1400-10000). Although these structures absorb most of the light spectrum, the longer wavelength end of UV-A (365-440nm), visible (400-700nm) and near IR (IR A 700-1400nm) light can still pass through the ocular media and converge on retina and undergo absorption by photoreceptor and lipofuscin containing retinal pigment epithelium. This phototoxicity, mainly from the higher energy UV A and shorter wavelength of visible light, leads to generation of reactive oxygen species and subsequent oxidative damage to these epithelial cells and the surrounding photoreceptor. In Acute phototoxicity, damage is sustained by RPE depigmentation and a swelling of the outer retinal layers and secondly, the damage is transmitted to the inner layers of the retina. Histopathological studies have confirmed that both the RPE layers and the outer segments of the photoreceptor layer are the most susceptible to damage. Photic retinopathy more commonly occurs in younger patients, male>female (because of outdoor work). The symptoms are often bilateral but can also affect only one eye. They usually develop within 1 to 4 hours after exposure and include; decreased visual acuity, metamorphopsia/distorted vision, micropsia, central or paracentral scotoma of 1 to 7 degree, photophobia, chromatopsia, after image, frontal and temporal headache with orbital or retro-orbital pain. On fundus examination in Acute phase, photic retinopathy may initially appear normal or can show a small yellow spot at the fovea, encircled by faint grey granular pigmentation. The yellowish discoloration will usually become faint with time, leaving a pathognomonic reddish spot. Weeks following acute damage reveal pigmented foveal changes which appear like lamellar macular hole, lack of foveal reflex, and chorioretinal paramacular small pigmentary changes. The diagnosis can be confirmed using clinical history and fundoscopy. Amsler grid reveals paracentral or central scotoma and metamorphopsia. Fundus fluorescein angiography may appear normal or as early infiltration in fovea centralis in acute phase of disease and RPE window defect may appear in late phase. Fundus autofluorescence shows central hypo-autofluorescence encircled by faint hyper-autofluorescent ring which rules out pseudo vitelliform dystrophy. Optical coherence tomography helps to confirm the diagnosis, assess the extent of lesion and monitor its progression. It shows maintained foveal contour with increased foveal rod shaped full thickness hyperreflectivity that extends from outer segments of the photoreceptors and the RPE to the inner layer of the retina. It shows disrupted reflectivity in the outer retina or fragmentation of highly reflective outer retina, juxtapfoveal microcystic cavities in the outer retina, and interruption of the ERM and IS/OS junction. Multifocal Electro-Retinogram (mERG) shows decrease in amplitude with normal latency. Most cases of photic retinopathy improve over time without treatment. No guidelines exist for the treatment of photic retinopathy. Several case reports of photic retinopathy have reported the use of steroids in the treatment of macular oedema with equivocal results. Antioxidants and free radical scavenger gingko glycosides may also be used in mild cases to ameliorate retinal damage. The use of vitamin A and aspirin appears to reduce the risk of photo-toxic damage to the retina similar to the use of antioxidants. The regain in visual acuity in photic retinopathy is co-related with initial visual acuity, initial rate of recovery and degree of visual impairment. Worse long-term vision is significantly related to the presence of photoreceptor layer damage on OCT. Differential diagnosis includes solar retinopathy, welder’s maculopathy, post surgical photic retinopathy, accidental laser exposure, other causes of lamellar depression or holes like PVD or trauma, pseudo vitelliform dystrophy, tamoxifen retinopathy, early stage MacTel type 2. Acute retinopathy cases are rarely reported because they report late to the out-patient department if at all they report, or it might be because of its’ self-limiting nature. Here we are presenting an acute case of solar retinopathy with weekly OCT report up to 1 month follow up and chronic cases of solar and welders’ retinopathy (photic retinopathy), thereby presenting morphological changes in photic retinopathy from acute to chronic stage. There is a rare fundus picture reported earlier by Lee et al, they reported a case of acute central serous chorioretinopathy after direct solar viewing.

Conclusion

Exposure to sunlight because of observing an eclipse (for religious reasons), drug use, microscope and ophthalmoscope lights are the main causes of photic retinopathy and all are easily preventable. Public awareness about the appropriate protective measure while viewing an eclipse and using welding arc and education related to hazards of direct sun gazing and training of welders are mandatory. Welders should be advised to avoid ingestion of photosensitizing drugs.

References

1. Istock TH. Solar retinopathy: a review of the literature and case report. J Am Optom Assoc 1985; 56:374-82.
2. Boettner EA, Wolter JR. Transmission of the ocular media. Invest Ophthalmol Vis Sci 1962; 1:776-783.
3. Dillon J, Zheng L, Merriam JC, Gaillard ER. Transmission spectra of light to the mammalian retina. Photochem Photobiol 2000; 71:225-229.
4. Slaney DH. How light reaches the eye and its components. Int J Toxicol 2002; 21:501-509.
5. Glickman RD. Ultraviolet phototoxicity to the retina. Eye Contact Lens 2011; 37:196-205.
6. Davies S, Elliott MH, Floor E, Truscott TG, Zareba M, Sarna T, et al. Photocytotoxicity of lipofuscin in human retinal pigment epithelial cells. Free Radic Biol Med 2001; 31:256-265.
7. Jain A, Desai RU, Charael RA, Quiram P, Yannuzzi L, Sarraf D. Solar retinopathy comparison of optical coherence tomography (OCT) and fluorescence angiography (FA). Retina 2009; 29:1340-1345.
8. Chen JC, Lee LR. Solar retinopathy and associated optical coherence tomography findings. Clin Exp Optom 2004; 87:390-393.
9. Kevin C. Chen, MD. Jesse J. Jung, MD. Alexander Aizman, MD. Solar retinopathy: etiology, diagnosis and treatment. Not always caused by sun exposure, solar retinopathy causes distinct injury to the retina. Retinal Physician 2013; 10:46-50.

10. J.D.M. Gass. “Photic maculopathy,” in Stereoscopic Atlas of Macular Diseases. St. Louis, Mosby, Mo, USA, 4th edition, 1997; 760-765.

Cite This Article as: Arora N, Patel A, Ahuja AK. Morphological Changes from Acute to Chronic Photic Retinopathy.

Acknowledgments: Nil

Conflict of interest: None declared

Source of Funding: None

Date of Submission: 26 May 2018
Date of Acceptance: 12 July 2018

Address for correspondence

Neha Arora MBBS
210/3, Bhargav Niwas, 3rd Floor,
Doongar Mohalla, Vishwas Nagar,
Shahdara, New Delhi - 110032
Email id: arora.nehambbs@gmail.com

Quick Response Code