Optical coherence tomography and autofluorescence findings in chronic phototoxic maculopathy secondary to snow-reflected solar radiation

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A professional mountain trekker presented with gradual, moderate visual decline in one eye. The subnormal vision could not be explained by the examination of anterior and posterior segment of either eye, which was unremarkable. Optical coherence tomography and autofluorescence imaging revealed subtle defects in the outer retina, which correlated with the extent of visual disturbance. A novel presentation of retinal phototoxicity due to indirect solar radiation reflected from snow in inadequately protected eyes of a chronically exposed subject is reported.

Key words: Fundus autofluorescence imaging, optical coherence tomography, retinal phototoxicity, solar radiation

This study was performed at Retina-Vitreous Service, Aravind Eye Care System, Madurai, Tamil Nadu, India

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Manuscript received: 12.12.14; Revision accepted: 24.01.15
Repetitive long-term exposure to bright ambient light can result in subtle, cumulative retinal damage at the level of photoreceptors and retinal pigment epithelium (RPE). However, chronic retinal phototoxicity due to indirect exposure to bright ambient light has not been described. I report macular phototoxicity and supportive imaging findings in a mountain-trekker, secondary to prolonged exposure to solar radiation reflected from snow.

Case Report
A healthy 58-year-old man presented with a mild diminution of vision oculus sinister (OS) over several years. He had no history of any systemic illness, use of tobacco or prolonged systemic or ocular medication. There was no history of any sight-threatening early or late-onset eye disease in the family. He had been conducting trekking missions in the snow-covered Himalayan region for the past 30 years, occasionally without adequate protective goggles. On ocular examination, his best-corrected visual acuity was 20/20 oculus dexterus (OD) and 20/50 OS. Slitlamp biomicroscopy revealed a normal anterior segment oculus uterque (OU); intraocular pressures were 14 and 16 mmHg. Fundus examination was unremarkable OU [Fig. 1a and b]. Color vision, contrast sensitivity, and central visual fields were normal OU. Amsler’s grid chart revealed distortions temporal to fixation OS. Spectral-domain optical coherence tomography (OCT) (Cirrus HD-OCT, Carl Zeiss Meditec, Dublin, CA) revealed a mild irregularity of inner-segment outer-segment (IS-OS) OD with juxtafoveal interruption of the cone outer segment tips (COST) line; central macular thickness (CMT) was 203 µm [Fig. 1c]. OS showed relatively thinner fovea (CMT: 148 µm) and minimal subfoveal COST interruption [Fig. 1d].

Fundus camera-based autofluorescence imaging (fundus autofluorescence [FAF], Zeiss Visupac 450Plus IR, Jena, Germany) revealed increased macular autofluorescence and clusters of hypoautofluorescent spots OU, more predominant suprafoveally [Fig.1e and f]. The patient was advised to use ultraviolet UV-blocking sunglasses in bright outdoors.

Discussion
Late retinal findings on OCT and FAF have been reported after sun-gazing. Snow reflects 90% of the incident sunlight, and, therefore, is likely to cause indirect solar retinopathy. I am not aware of any previous imaging study of retinal phototoxicity secondary to long-term exposure to snow-reflected solar radiation. These OCT findings were similar to but more subtle than the reports of chronic solar and welding arc retinopathy. The IS-OS junction was intact, and the primary site of insult was localized to the COST line in both eyes. Photoreceptors and RPE are the primary targets for both chronic and acute phototoxic injury from visible-spectrum light by two discrete mechanisms (type 1 and type 2 damages). Due to their proximity to the RPE, the seat of light absorption, cone outer segments appear to be most vulnerable to degeneration from prolonged solar irradiance. RPE escapes chronic damage by mitotically regenerating itself. On FAF imaging, the patient also showed an increased macular autofluorescence, probably due to luteal pigment depletion. The hypofluorescent dot signals were not central, but paracentral and multiple, clustered at and above the macula. These FAF abnormalities have been described previously. These hypofluorescent dots were probably caused by focal deficits in lipofuscin due to photoreceptor damage. The abundance and clustering of these dots at and above the central macula was different from the previous reports. Potential explanations for this plurality and pattern could be the recurrent, prolonged exposures to snow-reflected sunlight, the angle of reflections from below, and the tendency of eyes to fix gaze level with the horizon. The FAF lesions were not clinically obvious, as previously observed. The visual symptoms also may not always correlate with physical findings. In this patient, the visual status correlated with OCT rather than FAF lesions: The symptomatic eye had thinner, atrophic fovea and subfoveal COST defects. Though the present patient failed to wear protective eyewear consistently, people in vulnerable vocations (e.g. skiers, welders) are likely to develop subclinical phototoxicity with prolonged exposure, even with protective eyewear. Periodic evaluation with FAF, OCT and functional investigations like microperimetry and multifocal electroretinography, in these vocational groups, may reveal subtle anatomic and functional defects, and possibly lead to modification of safety standards for acceptable ambient light.

References

Cite this article as: Shukla D. Optical coherence tomography and autofluorescence findings in chronic phototoxic maculopathy secondary to snow-reflected solar radiation. Indian J Ophthalmol 2015;63:455-7.

Source of Support: Nil. Conflict of Interest: None declared.