CLINICAL AND EXPERIMENTAL
OPTOMETRY

CASE REPORT

Solar retinopathy and associated optical coherence tomography findings

Clin Exp Optom 2004; 87: 6: 390–393

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Submitted: 11 February 2004
Revised: 16 June 2004
Accepted for publication: 22 June 2004

Solar retinopathy is a rare but well-recognised clinical entity of macular damage, caused by viewing a solar eclipse or direct sun-gazing. Visual deterioration from solar retinopathy typically ranges from 6/9 to 6/60 and in most cases the visual loss is reversible. We present a case of solar retinopathy following direct sun-gazing and illustrate the damage within the retinal structure with optical coherence tomographic (OCT) findings. The visual prognosis of solar retinopathy is usually favourable but prevention remains the mainstay of treatment. The optometrist may play an important role in patient education and reassurance, as well as differentiating solar retinopathy from other likely macular abnormalities.

Key words: eclipse retinopathy, fovea, foveomacular retinitis, optical coherence tomography, solar retinopathy

Solar retinopathy, also known as eclipse burn, eclipse retinopathy or foveomacular retinitis, refers to maculopathy that occurs following prolonged exposure to solar radiation.[1] Adverse visual effects of solar radiation have long been recognised and cases of solar retinopathy have been reported since the 18th century. The famous astronomer Galileo Galilei was said to have injured his eyes while trying to observe the sun. The majority of reports of solar retinopathy have been due to viewing of a solar eclipse but direct sun-gazing due to religious rituals,[2-4] mental illness[5] and use of hallucinogenic drugs such as LSD[6,7] may also increase the risk of developing this form of retinopathy. When there is no confirmed history of prior solar exposure, the term foveomacular retinitis is used to describe the clinical entity that closely resembles solar retinopathy with characteristic visual symptoms and foveal lesions.[8]

Following solar exposure, patients typically present with symptoms of blurred vision, a central or paracentral scotoma, chromatopsia, metamorphopsia, photophobia and headache.[8] Ophthalmoscopic examination may reveal a small yellowish-white spot in the centre of the foveal region surrounded by an area of faint grey, granular pigmentation. The defect usually lies beneath or adjacent to the foveal reflex, which in most cases is still present. This spot may fade gradually with time (that is, two weeks) and assumes a reddish, sharply circumscribed appearance at the fovea, which Gass[1] described as pathognomonic of solar retinopathy. In patients with repeated episodes of direct sun-gazing, a larger lesion may be observed, giving the fovea a mottled, honeycombed appearance.[1,8] Most cases of solar retinopathy are bilateral but the extent of the macular lesions may be more pronounced in the patient's dominant eye.[9]

The extent of retinal damage in solar retinopathy depends on the intensity and

duration of solar exposure and such retinal damage can occur by three mechanisms: photochemical, mechanical and thermal damage.[1] Condenotti, Patelli and Brancato[9] suggested that retinal tissue compromise in solar retinopathy is more likely to be a combination of thermal damage with a rise in temperature and the photochemical changes in the retina mediated by the absorption of the short-wavelength light of the visible spectrum. If the photochemical process is thermally enhanced, the retinal pigment epithelial (RPE) layer may be the most susceptible to solar radiation due to the absorption of light energy by the melanin in the RPE.[8]

Histopathological studies have demonstrated that the RPE and outer segments of the photoreceptor layer are most susceptible to solar damage.[10] To investigate the structural damage of the retina incurred from solar retinopathy, optical coherence tomography (OCT) may be used to provide high-resolution, cross-sectional tomographic *in situ* images of the retinal tissues.[11,12] Here, we present a case report of solar retinopathy following direct sun-gazing and evaluate the OCT findings associated with solar retinopathy.

CASE REPORT

A 25-year-old Caucasian male presented with a one-month history of seeing spots before both eyes. On presentation, his visual acuity was 6/7.5 in both eyes with minimal refractive errors. A central scotoma and metamorphopsia were noted bilaterally on Amsler grid testing. The anterior segments appeared normal in each eye. Pupils were equal and round with no relative afferent pupil defects. The patient had a history of poorly controlled schizophrenia and was currently taking an antipsychotic drug Olazepine (Zyprexa) for treatment. There was no history of head or ocular injury, allergy or any recollection of ocular disease in his family.

Ophthalmoscopic examination revealed a yellowish-white lesion in the fovea, surrounded by a circular rim of hyperpigmentation in both eyes (Figures 1 and 2). There was no foveal reflex present in either eye. On questioning, the patient recalled staring at the sun for approximately 10 minutes, one month prior to presentation and this led to the diagnosis of solar retinopathy.

The patient was referred for an OCT examination, three months after the initial presentation. His visual acuities remained at 6/7.5 in both eyes. OCT was performed using two cross scans centred on the foveal lesion. Cross-sectional retinal images produced by the OCT revealed localised loss of RPE cells at the centre of the fovea, with normal overlying retinal layers (Figures 3 and 4). No vitreo-retinal interface abnormalities were present in either eye.

COMMENT

Bechmann and colleagues[13] were the first to present the structural damage of the retina in solar retinopathy using optical coherence tomographic (OCT) investigation of the macula. They found a hyperreflective area at the fovea and all retinal layers were affected. In the case presented here, there was a localised loss at the RPE layer and all other retinal layers were normal. This is most likely due to photochemical damage to the pigmented RPE layer, preserving the overlying transparent retinal layers. Condenotti, Patelli and Brancato[9] have also described various OCT presentations in cases of solar retinopathy and alterations such as reduced intensity of the reflectiveness from the RPE layer, changes in the reflectiveness from the inner retinal layer were all observed. Our finding of localised RPE loss was similar to a recent report by Kaushik, Gupta and Gupta,[4] which described an excavation of the RPE-choroid complex following solar retinopathy. Thus, the retinal structural alterations demonstrated by the OCT may not be consistent in every case of solar retinopathy and the presentation may depend on the intensity, duration and light spectrum of solar exposure.

Condenotti, Patelli and Brancato[9] repeated the OCT scans in four patients, one month following the solar exposure and found that the retinal structural alterations that were previously observed on initial presentation had disappeared and visual acuities improved to 6/6 in all patients. Visual acuity of our patient remained at 6/7.5 in each eye and OCT scans showed focal loss at the RPE layer three months after the initial presentation. Generally, retinal tissue compromise due to solar retinopathy is reversible and most cases of solar retinopathy will improve significantly with time, with patients regaining good visual acuity (between 6/6 to 6/12) within a period of three to six months following exposure.[8] The improvement in visual acuity mostly takes place during the first two weeks to one month following exposure.[14] Individuals presenting with initial visual acuities of 6/60 or worse may be more likely to exhibit long-term reductions in their visual function. The return of good visual acuity may not translate to a full recovery of visual function and some patients may continue to experience a small persistent central or paracentral scotoma.[8] In cases that do not resolve spontaneously, final visual outcome is less favourable due to likely damage to the photoreceptors and the RPE, leading to retinal atrophy and degeneration.[15]

There appears to be considerable individual susceptibility to developing solar retinopathy, with a greater incidence observed in patients with clear media and minimal refractive errors. Aphakic patients, emmetropes and low hyperopes may be at greater risk of developing solar retinopathy due to an increased capacity for sharp focusing of the sun's rays on the retina. Despite lack of a standardised treatment protocol, visual deterioration resulting from solar retinopathy is usually reversible but prevention remains the best treatment option. Therefore, public health education concerning the hazardous consequences of direct sun-gazing is important, particularly in individuals who manifest inclinations towards such behaviour and in patients with untreated mental illness or those taking hallucinogenic agents. The risk of developing solar retinopathy may also be greater in regions with higher atmospheric transmission of UV-B radiation.[16] The optometrist is in a position to educate patients and reassure individuals who present with characteristic foveal lesions and a positive history of solar

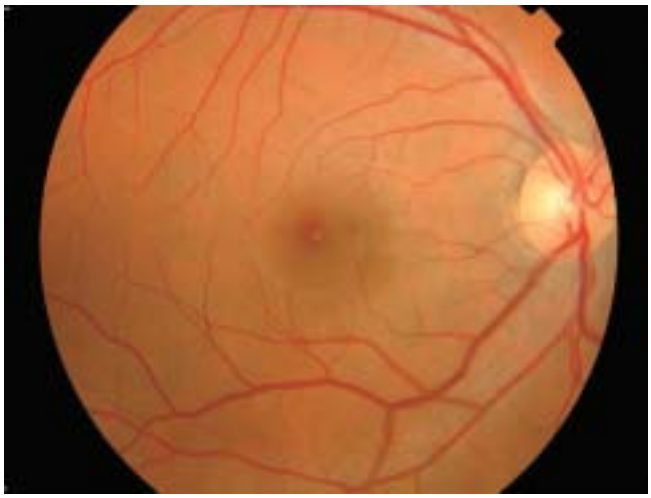

Figure 1. Fundus photograph of the right eye showing solar retinopathy that is typically characterised by a yellow lesion in the foveolar region surrounded by a cuff of hyperpigmentation

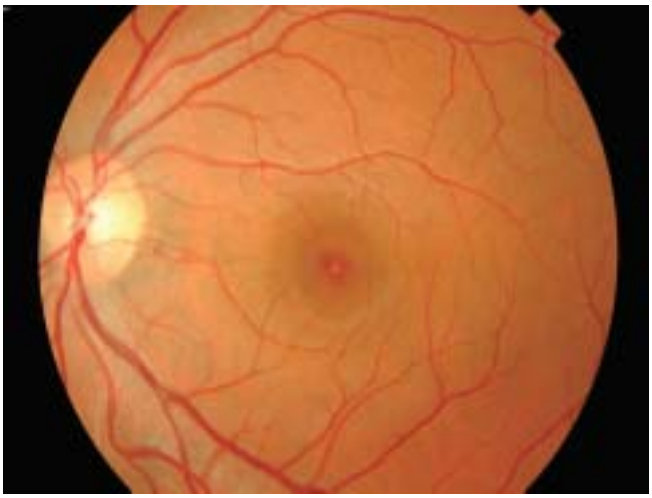

Figure 2. The patient's left fundus showing features identical to those of the right eye

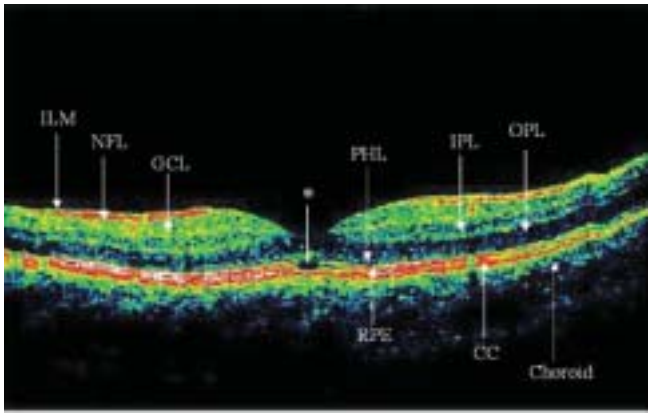

Figure 3. OCT scan of the right macula three months following the initial presentation. There was a focal loss of cells at the RPE layer (marked by the asterisk). All other retinal layers were unaffected. Abbreviations: ILM: internal limiting membrane, NFL: nerve fibre layer, GCL: ganglion cell layer, PHL: photoreceptor layer, IPL: inner plexiform layer, OPL: outer plexiform layer, RPE: retinal pigment epithelium, CC: choriocapillaris

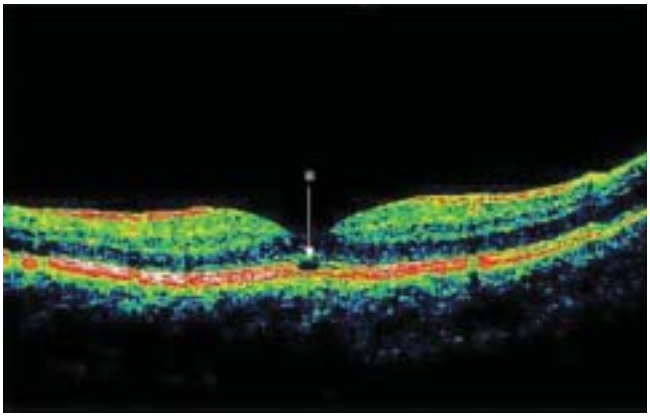

Figure 4. OCT scan of the patient's left macula showing the focal excavation at the RPE layer (marked by the asterisk), similar to that of the right eye

exposure, that visual acuity may improve over time. The optometrist should also establish a precise case history and in cases without a confirmed history of solar exposure, other macular diseases must be differentiated from solar retinopathy.

CONCLUSION

Retinal tissue compromise incurred as a result of solar retinopathy is effectively photochemical in nature, although the exact mechanisms responsible for producing solar retinopathy are unknown. Visual prognosis of solar retinopathy is usually favourable but patient education concerning the hazardous visual consequences of solar retinopathy is crucial.

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