Case Report

Solar Retinopathy

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Pak J Ophthalmol 2007, Vol. 23 No. 3

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Received for publication
March' 2007

Solar retinopathy is a well recognized clinical entity of retinal damage caused by direct or indirect viewing of the sun. Synonymous terms includes foveomacular retinitis, eclipse retinopathy, and solar retinitis.

We are presenting this interesting case report of a middle aged male patient presented in the eye OPD with the complaints of gradual dimness of vision, metamorphopsia and scotomas.

On examination best-corrected visual acuity was OD 6/18 OS 6/24. Anterior segment examination was unremarkable in both eyes; however, metamorphopsia and paracentral scotomas were documented on Amsler's grid in both eyes. On fundoscopy, bilateral round foveal lesions were noted.

Probing questions were asked to help determine the cause of these remarkable macular findings. The man turned out to be a ritual sun gazer and a witch doctor! The patient was advised strongly against sun gazing and was put on placebos. At the end of 6 months, Snellen's visual acuity improved to OD 6/6 (p) OS 6/12. Metamorphopsia and scotomas persisted and no physical change in the macular lesions was detected.

Solar retinopathy is a well recognized clinical entity of retinal damage caused by direct or indirect viewing of the sun. Synonymous terms include foveomacular retinitis, eclipse retinopathy, and solar retinitis[1].

CASE REPORT

A 56 year old man came to the eye OPD, Baqai Medical University Hospital, with the complaints of gradual dimness of vision over a period of 2-3 weeks associated with metamorphopsia and paracentral scotomas. Past ocular and systemic histories were unremarkable.

On examination visual acuity was OD 6/18 OS 6/24; no further improvement was noted with pinhole test or with refraction. Intra ocular pressure was 16mm Hg in both eyes and extraocular movements were full in range. There was no RAPD in either eye and rest of the anterior segment examination was unremarkable in both eyes; however, metamorphopsia and paracentral scotomas were documented in both eyes on Amsler's grid (black lines on white background).

On fundoscopy, bilateral round foveal lesions were noted. OD showed a lamellar macular hole at fovea 1/3rd disc diameter (photographs 1 & 2) and OS revealed a 1/3rd disc diameter round lesion marked in the center by a red apex and surrounded by a gray halo (photographs 3 & 4). Optic discs were normal in both eyes and no cells were detected in the vitreous in either eye. The peripheral fundi were also unremarkable in both eyes.

After these remarkable macular findings, probing questions were asked as to the use of any ocular or systemic drugs, family history of any significant eye disease, viewing of solar eclipse, blunt ocular trauma, automobile accident (whiplash injury) etc. Finally history of sun gazing for almost 1 hour/day, regularly at dawn stretching back to almost a month was elicited.

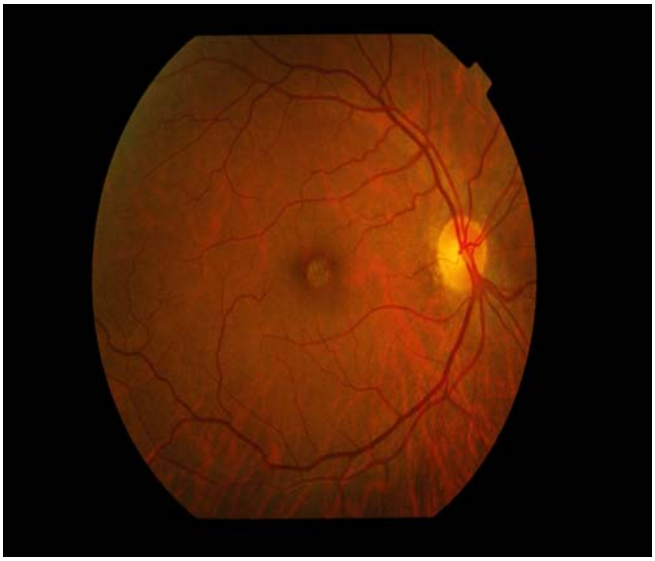

Fig. 1: Right eye: Lamellar macular hole

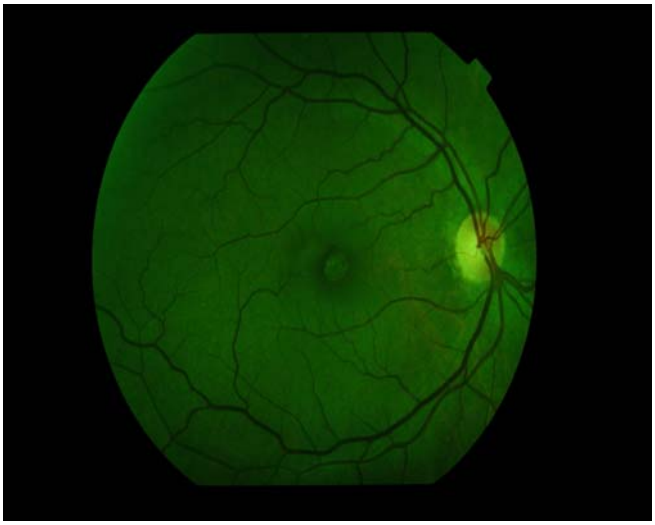

Fig. 2: Right eye: Lamellar macular hole (Red-free photo)

The patient turned out to be a ritual sun gazer and a witch doctor.

The patient was advised strongly against sun gazing. Placebos (Topical lubricants) were given and follow up was scheduled at 1-month interval. On repeated scheduled follow-ups visual acuity steadily improved in both eyes; however, no change was noted in the metamorphopsia or scotomas in either eye, also no change was detected in the size and appearance of the macular lesions.

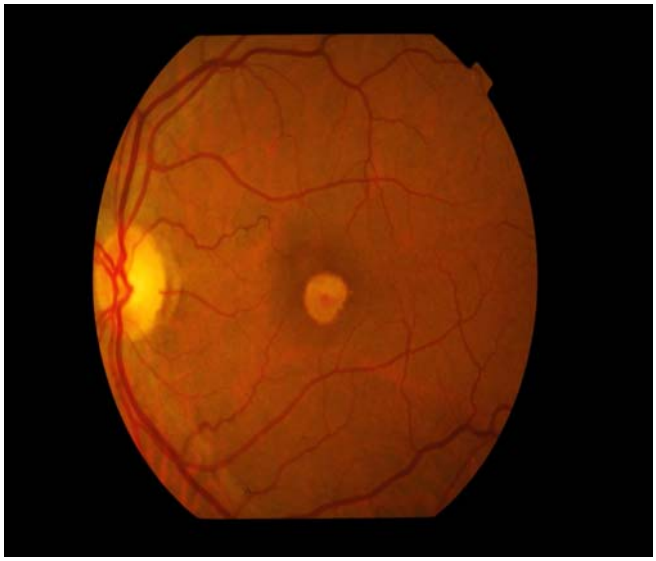

Fig. 3: Left eye: De-pigmented macular lesion surrounding a red apex

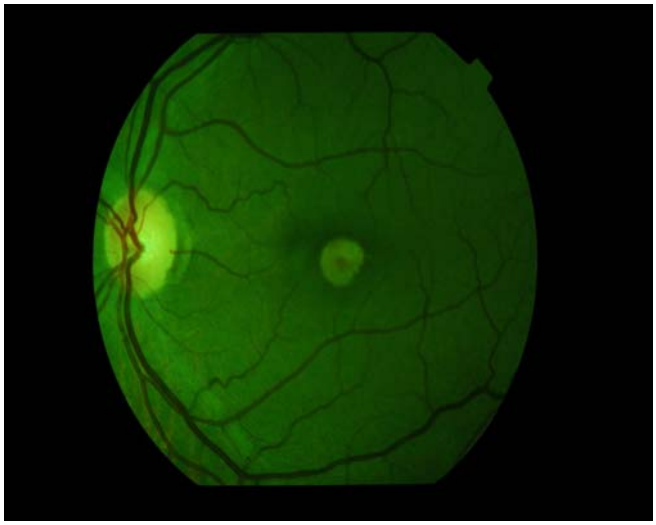

Fig. 4: Left eye: De-pigmented macular lesion surrounding a red apex (Red-free photo)

At the end of 6 months, Snellen's visual acuity improved to OD 6/6 (p) OS 6/12. Metamorphopsia and scotomas persisted and no physical change in the macular lesions was detected. Unfortunately the patient lost to follow up.

DISCUSSION

The first clinical description of retinal damage in association with the viewing of an eclipse was by Saint-Yves in 1722[2]. Foveomacular retinitis was

originally described as a distinct clinical syndrome of unknown cause consisting of bilateral decreased vision and foveal lesions in young military personnel[3]. In many instances, however, a history of sun gazing was subsequently elicited[4]. Many of these patients were diagnosed with psychiatric disorders[5]. Other cases occur in military personnel who have followed the flight of air planes near the sun[6]. Photic retinopathy has also been described in association with direct sun gazing by sunbathers, and patients with psychotic disorders, as part of religious rituals, and in association with the use of drugs such as lysergic acid diethylamide (LSD)[7]. The appearance and clinical course in each instance was identical.

Various factors have been implicated in determining the severity of the retinal lesions. Increasing length of exposure is a risk factor[8]. However severe lesions have been described in individuals with minimal exposure, and vice versa. It has been suggested but not proved that increasing fundus pigmentation protects against the photic damage[9]. Patients with uncorrected high refractive errors may be protected. In the presence of amblyopia and strabismus, the dominant eye is more susceptible to damage[9]. Younger patients may be at increased risk because of the transmissibility through clear media. LSD induce mydriasis and cycloplegia which make the drug abuser more susceptible to severe retinal burn[10].

Prior to 1970, the cause was believed to be due to thermal damage produced by the absorption of infrared rays by the RPE[11]. It is postulated that the principal mechanism of photochemical damage is from retinal irradiance by high energy wavelengths, including short wavelength-visible blue light and lower levels of UV-A or near-UV radiation (320-400nm).

Symptoms of solar retinopathy usually develop within 1 to 4 hour after exposure and include decreased vision, metamorphopsia, micropsia, and central or paracentral scotomata of 1 to 7 degrees. Patients may also present with chromatopsia, photophobia, after image, and frontal and temporal headache with orbital or retro orbital pain. Acutely, vision usually ranges from 6/12 to 6/36 but may be worse. There is no correlation between the severity of the fundus lesion and the visual acuity[12].

The fundus examination is variable. Although usually bilateral, unilateral cases are not uncommon. The typical lesion is a small yellow spot with a surrounding gray zone in the foveolar or parafoveolar area within the first few days after exposure. In mild cases however, little or no change can be noted. The foveal reflex may be lacking but becomes more distinct as the lesion resolves. After several days, the yellow spot becomes reddish with a halo of surrounding pigmentary change by 10 to 14 days, this lesion fades and is usually replaced by a red, well circumscribed, faceted lamellar hole or depression. The oval lamellar depression, which has a diameter of 100µ to 200µ is believed to be permanent and is highly suggestive of previous episode of sun gazing[13]. Several lesions may be present suggestive of multiple exposures. Similar lesions have been produced by blunt trauma and whiplash injury.

Fluorescein angiography is often normal in the early and late stages of the disease.

Visual acuity usually improves to 6/6 to 6/9 by 6 months.[14] Even with improvement in visual acuity, residual metamorphopsia and central or paracentral scotomas may persist.

Tso[15] has studied the histopathologic features of lesions in patients who gazed at the sun for 1 hour prior to enucleation for uveal melanoma. Approximately 2 days after sun gazing, most of the injury involved the RPE. Necrosis, pigment granule irregularity, and focal detachments of the retinal pigment epithelium were described. In one patient who demonstrated early leakage on Fluorescein Angiography, a focal detachment of the sensory retina was found at the site of the lesion. The photoreceptors were intact. The RPE adjacent to the lesion lost its apical pigment granules and extended along Bruch's membrane beneath the detached RPE. After 48 hours to 5 days, there is photoreceptor destruction. Much of this damage is reversible and may explain the ability of many patients to recover good visual function after sun gazing[16].

Oral corticosteroids have been used in the treatment of acute lesions associated with severe visual loss. However, no beneficial effect has been clearly demonstrated.

Appropriate protective measures when viewing an eclipse and education about the hazards of direct sun gazing are of utmost importance in the prevention of this disease.

CONCLUSION

Sun gazing, ritual or otherwise, is a well-recognized cause of photic damage to the retina. This case report

has been presented to highlight the hazards of direct or indirect sun gazing and to negate the generalized misconception among the general public as well as doctors that solar retinopathy occurs only after viewing solar eclipse.

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