

The Occurrence of Retinopathy in 11 Eyes After a Solar Eclipse, 1999

Received: March 25, 2002

Abstract: To present the visual outcome of patients with solar retinopathy and evaluate the effects of treatment on the visual prognosis.

One hundred and eighty-eight patients with visual disturbances applied for ophthalmic examination following the solar eclipse on August 11, 1999. All patients underwent routine ophthalmologic examination; those with fundus changes also underwent fundus fluorescein angiography, Amsler grid and computerised perimetry. Among them, nine patients (11 eyes) were evaluated as having solar retinopathy with decreased visual acuity and discernible fundus findings. Mean age was 21.5 (17-34) in this group. Five patients were given treatment, three were on oral methyl prednisolon and two were on ginkgo glycosides. Statistical analysis could not be performed because of the small number of patients in the treatment groups.

The duration of exposure was 1-30 min. The mean initial visual acuity was 20/32 (min 20/100, max 20/25). All eyes aside from one revealed positive Amsler grid tests. Computerised perimetry showed central scotoma in four eyes. The mean visual acuity at final examination (3 months later) was 20/24 (min 20/50, max 20/20). Metamorphopsia persisted in five eyes, and disability at near vision persisted in one eye after 3 months. Early and late fundoscopic findings did not correlate with either duration of exposure or visual acuity.

Reversible or persistent visual abnormalities may follow a solar retinal burn. Prevention seems to remain the best treatment. Corticosteroids may be beneficial in severe cases.

Key Words: Solar retinopathy, protection, and treatment.

Department of Ophthalmology, Faculty of Medicine, Firat University, Elazığ - Turkey

Introduction

Solar retinopathy is a well-recognised clinical entity of retinal damage caused by direct or indirect viewing of the sun. Synonymous terms include foveomacular retinitis, eclipse retinopathy, solar retinitis, eclipse blindness, eclipse burn and solar chorioretinal burn (1). While the majority of cases of solar retinopathy have involved eclipse viewing, sailors, photographers, religious sun-gazers, schizophrenics, people under the influence of hallucinogenic drugs and even sunbathers are at risk of developing this form of retinopathy (2-4).

The number of reported cases of solar retinopathy after the solar eclipse of the August, 1999 was extremely small because of extensive information in the media about appropriate protection and the presence of cloudy weather over most parts of Europe (5-8).

Various factors may determine the severity of the retinal lesions and the loss of visual acuity (VA) in solar retinopathy. Increased duration of exposure and the

protection used are the major risk factors for retinopathy and generally correlate with the severity of retinal lesions (1,3). However, retinal lesions with very short exposure to the sun were reported (9). Viewing an eclipse through binoculars, sunglasses, exposed photographic or radiographic film or audio CDs is never safe (10). Commercial solar filters certified as being safe have been shown to be the only safe method for eclipse observation (11). An increased risk of retinal damage has been associated with the use of photosensitising drugs like tetracycline and psoralen (1). Variability in the degree of susceptibility to photic damage suggests that host factors are also important (12).

Most cases of solar retinopathy improve over time without treatment (13). Despite the lack of a standardised protocol, corticosteroids and antioxidants are believed to be beneficial in the treatment of solar retinopathy (14,15). In this article we present the visual outcomes in a series of young adult patients who

developed solar retinopathy following an eclipse of the sun and try to analyse the effects of treatment with oral methyl prednisolon and ginkgo glycosides.

Materials and Methods

Ocular histories and examinations were obtained from 188 patients who reported visual disturbances between 2 and 6 days following a solar eclipse on August 11, 1999. All patients were living in the Elazığ region, where the total eclipse could be viewed, and all of them reported their symptoms following the viewing of the eclipse. In addition to a complete ophthalmologic examination including VA testing, slit-lamp examination, intraocular pressure determination and ophthalmoscopy, Amsler-grid, visual field testing and fluorescein angiography were performed in patients who had positive fundus findings with decreased VA. One hundred and sixty-nine of the 188 patients had either a history of after-image for a few minutes or displayed ocular surface problems due to extended ultraviolet exposure; so they were excluded. Ten patients with decreased VA due to refractive errors, amblyopia, and lens opacities were also excluded. Retinal changes along with VA loss were observed in 11 eyes of the remaining nine patients following the solar eclipse and diagnosed as solar retinopathy. During exposure, four patients were absolutely unprotected and five were protected by inappropriate methods like processed X-ray film, sooty glasses or ordinary sunglasses. Exposure intervals were between 1 and 30 min. None of these patients was predisposed to solar retinopathy because of any known risk factor like systemic or topical medications, or ocular and systemic diseases.

After completing the initial tests, three severely affected patients were given methyl prednisolon (60 mg/day) for 10 days following initial examination. Two randomly selected patients (cases 3 and 6) were put on ginkgo glycoside 9.6 mg b.i.d. for 4 weeks. The remaining patients were followed without medication. Because of the small number of patients and the lack of randomised distribution between groups, no statistical analysis could be performed.

Results

Blurred vision, central scotoma, metamorphopsia, headache and after-image were the main symptoms of

patients. The demographic features and clinical findings at first examination and treatment modalities are summarised in Table 1. The VA of the patients was between 20/100 and 20/25 (mean 20/32) at initial examination. All patients had discernible fundus changes; most of them being foveal grey-yellow cysts with surrounding oedema (seven eyes). Figure 1 shows left foveal lesion in case 2. The other fundus changes were macular oedema and discoloration without a cyst in two eyes (cases 4 and 7) and reddish, well-circumscribed depression again in two eyes (both eyes of case 5). Figure 2 shows right foveal depression with reflecting borders in case 5. Fluorescein angiographic findings were normal in all cases except case 4, in which a window defect was seen. Amsler grid revealed central/paracentral scotoma and metamorphopsia in all patients except case 7. Four of these eyes also showed central scotoma in a 10 degree area with computerised perimetry. Final VA in nine eyes was better than 20/25. Mean VA of our patients at the end of 3 months was 20/24 (20/50 - 20/20). The mean increase in VA of the treated eyes was 1.86 Snellen lines (2.2 line in corticosteroid group and 1 line in ginkgo glycoside groups). Mean increase in VA of the no treatment group was 1.25 lines. At the end of 3 months, three of the 11 eyes were free from symptoms. Metamorphopsia persisted in seven eyes; one with disability at near vision. The results of the follow-up examinations are summarised in Table 2.

Discussion

The extent of retinal damage and associated visual impairment is reported to be dependent upon the intensity and duration of solar exposure (3,13). In spite of the difficulties in stating the correlation of clinical findings with exposure time and protection in such a small group, these factors did not seem to be correlated with retinal lesions and the VA in our patients. It should be noted that reported periods were estimated and not measured exactly. None of our patients had appropriate protection; however, we estimated the total number of people who viewed the eclipse unprotected in the Elazığ region as being quite numerous. This supports the theory of individual susceptibility (1).

Reported symptoms of solar retinopathy include decreased VA, metamorphopsia, micropsia, central or paracentral scotoma, chromatopsia, photophobia, after-

Table 1. Table shows the demographics, exposure times, results of the first ophthalmologic examination and medications initiated. (BCVA: Best corrected visual acuity, MP: methyl prednisolone, GG: ginkgo glycoside).

Case	Age/sex exp time	BCVA Right-Left	Fundus examination	Fluorescein angiogram	Other tests	Treatment
1	34/F 10 min	20/20-20/30	Left foveal cyst and perifoveal granular appearance	Normal	Relative central scotoma with Amsler grid in left eye	None
2	18/F 30 min	20/40-20/30	Yellow-white, elevated irregular cyst & greyish halo in both foveae	Normal	Relative central scotoma & metamorphopsia in both eye (in visual field Amsler grid)	MP
3	17/F 15 min	20/25-20/20	Yellowish cystic lesion on right fovea	Normal	A faint scotoma on Amsler grid in right eye	GG
4	19/F 10 min	20/25-20/20	Greyish discoloration and oedema in right macula	Right faded window defect	Metamorphopsia (Amsler grid)/normal visual field in right eye	None
5	20/F 5 min	20/30-20/60	Bilateral small, reddish, well circumscribed depression; prominent in left eye	Normal	Bilateral metamorphopsia and central scotoma on Amsler grid	MP
6	24/F 1 min	20/20-20/25	Retinal irregularity in both foveae tiny cyst in left fovea	Normal	A faint scotoma on Amsler grid in left eye	GG
7	23/F 20 sec	20/25-20/20	Macular oedema and pigmentary changes in right eye	Normal	No abnormality	None
8	18/M 10 min	20/100-20/20	Juxtafoveal cyst in right eye	Normal	Metamorphopsia and central scotoma on Amsler grid in right eye	MP
9	21/M 20 min	20/30-20/20	Right foveal cyst, perifoveal oedema & pigmentation	Normal	Small central scotoma in right visual field testing	None

Table 2. Table shows the results of the first (at the end of first month) and second (at the end of the third month) follow-up examinations. (BCVA: Best corrected visual acuity).

Case	BCVA at 1 month Right-Left	Fundus examination 1 month	BCVA at 3 months Right-Left	Fundus examination/visual field 3 months	Persisting symptoms 3 months
1	20/20-20/20	Cystic lesion & mild pigmentary changes	20/20-20/20	Cystic macular lesion/ normal visual field	mild metamorphopsia
2	20/30-20/25	Elevated yellow cysts in both foveae	20/30-20/25	Tiny yellow cyst & pigmentary changes/right scotoma in visual field	Central scotomas & metamorphopsia
3	20/20-20/20	Yellowish cyst in right fovea	20/20-20/20	Yellowish discoloration/ normal visual field	No symptom
4	20/20-20/20	Greyish discoloration in left fovea	20/20-20/20	Greyish discoloration/ normal visual field	No symptom
5	20/25-20/30	Sharply circumscribed reddish depression in left fovea	20/25-20/25	Small depression in both foveae/left central scotoma	Bilateral metamorphopsia
6	20/20-20/20	Yellowish discoloration	20/20-20/20	Yellow dots in left fovea, normal visual field	Mild disturbance in Amsler grid (left)
7	20/20-20/20	No oedema-persisting pigmentary changes in right eye	20/20-20/20	Pigmentary changes/ normal visual field	No symptom
8	20/80-20/20	Juxtafoveal cyst-like lesion in right eye	20/50-20/20	Juxtafoveal cyst in right eye /persisting scotoma and metamorphopsia in visual fields	Central scotoma
9	20/25-20/20	Persisting cystic lesion in right eye	20/25-20/20	Disturbed foveal reflex in right eye due to cyst/tiny central scotoma	Moderate disability at near vision

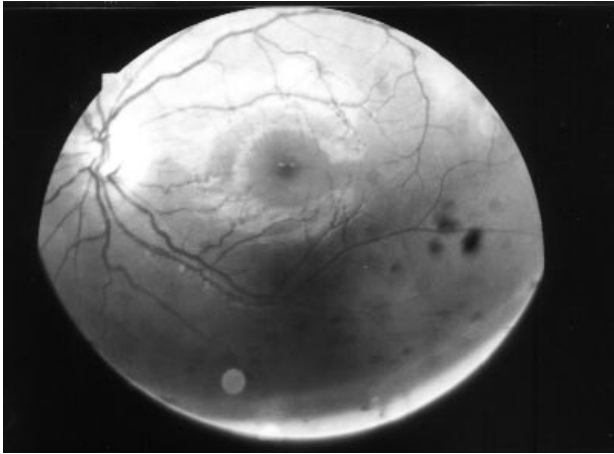


Figure 1. Left fundus photograph of case 2 shows elevated yellow cyst and surrounding greyish halo.

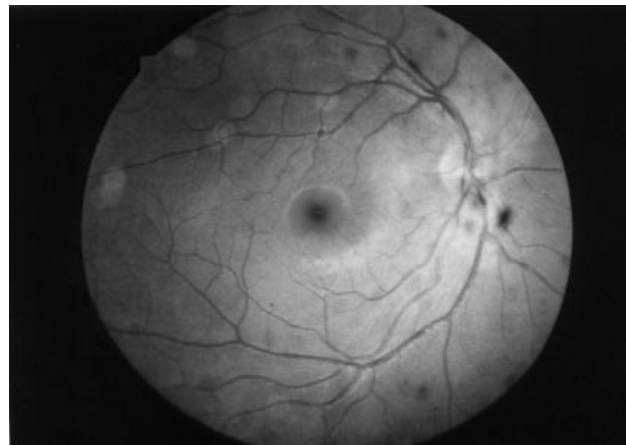


Figure 2. Right fundus photograph of case 5 shows reddish depression with sharp and reflecting borders in right fovea.

image and headache (1,6,12,16). Most of these symptoms were also seen in our group. Shortly after exposure to the sun, the VA was reported to decrease to a range between 20/40 and 20/100, but may be worse (12). Visual acuities were decreased in involved eyes (20/25 - 20/100) of our patients. In their group of 33 eyes, Kawa et al. reported that all of the patients revealed positive Amsler tests with decreased VA (17). Ten of the 11 eyes in our group had positive Amsler tests. Seven of our patients had unilateral involvement (five right and two left eyes). This was possibly due to the tendency of the patients to squint the non-dominant eye – mostly the left eye - to reduce their photophobia (1).

The extent of the fundus changes are variable in solar retinopathy. Michaelides et al. reported an abnormal macular appearance at presentation in 84% of their patients (6). This ratio was 47% in the case group of Verma et al. (3). In our group, all eyes had macular changes, since abnormal macular appearance was an inclusion criterion for this study. The typical fundus lesion is a small yellow spot with a surrounding grey zone in the foveolar or parafoveolar area within the first few days of exposure (18). Afterwards, pigmentary changes surround the yellow spot, which is usually replaced by a depression. A perifoveal greyish thickening, punctate atrophic disturbance of RPE or a pseudolamellar macular hole may persist (12,18,19). Along with this type of lesion which was seen in seven eyes, two different clinical findings were determined in our group: macular oedema with discoloration in two eyes and reddish depression in two eyes. The correlation between the severity of the fundus

lesions and VA is controversial (2,19). The most severe lesions in our group were seen with moderate VA loss and excellent visual prognosis (cases 1 and 4). Fluorescein angiography (FA) is often normal in the early and late stages of the disease, though may rarely show a small window defect corresponding to the lamellar hole (12,18). Atmaca et al. reported normal fluorescein angiographic findings in all of 86 eyes of their patients and evaluated it as a non-conclusive test in solar retinopathy (19). Dhir et al. reported microleaks and masking of choroidal fluorescein in early FA studies of severe cases (2). Only one eye in our group showed window defect, but there was no hole formation in this eye at the end of the third month.

The final VA was reported as 20/20 to 20/40 in most patients with solar retinopathy, which was reached within 3-9 months (12). Atmaca et al. reported a more prominent and earlier improvement in VA in eyes with an initial VA of 20/100 or better (19). In our group, eight eyes with 20/30 or better VA reached their final visual acuities of 20/25 or better at the end of the first month. Severe cases with VAs less than 20/40 (cases 2, 5 and 8) were still healing in the third month. Our results seem to support the theory of early improvement in mild cases (19). In spite of a generally good prognosis, return of VA to 20/20 does not always imply complete recovery because of persistent central scotoma, image distortion and macular changes which may dramatically reduce reading performance in some patients (17). Case 9 in this report displayed such a difficulty in reading even after the disappearance of his visual field defect. His findings were

probably due to a small central scotoma. Those minute scotomas are too small to be detected by standard perimeters, but may be found with scanning laser ophthalmoscopy (16).

Despite the controversial treatment of solar retinopathy, and lack of a standardised protocol, corticosteroids are believed to be beneficial in the treatment of solar retinopathy because of their ability to suppress inflammatory tissue reactions after injury (13,14,20). We used methyl prednisolone in three of our patients and, regarding the mean increase in VA, the outcome of these patients seemed better than the others. Antioxidants may also ameliorate retinal photic injury (15). We used the free radical scavenger ginkgo glycosides in two patients with mild involvement. Our results must be evaluated with suspicion since the groups are very small and the selection of patients for groups was non-randomised.

The pathogenesis of solar retinopathy is likely to be dependent on finely balanced components including an individual's age, ocular status, degree of exposure and intrinsic susceptibilities. Appropriate protective measures while viewing an eclipse and education about the hazards of direct staring are the mainstays in the prevention of this disease. This small population of solar retinopathy patients gave us a positive impression of the beneficial effects of corticosteroid therapy in visual acuities of severely involved eyes.

Corresponding author:

Nuray AKYOL

Firat Medical Center, Department of Ophthalmology

Elazığ - TURKEY

E-mail: NAKYOL@firat.edu.tr

References

1. Yannuzzi LA, Fisher YL, Slakter J, Krueger A. Thesis: Solar retinopathy. A photobiologic and geophysical analysis. *Retina* 9: 28-43,1989.
2. Dhir SP, Gupta A, Jain IS. Eclipse retinopathy. *Br J Ophthalmol* 65: 42-45, 1981.
3. Verma L, Sharma N, Tewari HK, Gupta S. Retinopathy after solar eclipse. 1995. *Natl Med J India* 9: 266-7, 1996.
4. Rai N, Thuladar L, Brandt F, Arden GB, Berninger TA. Solar Retinopathy. A study from Nepal and from Germany. *Doc Ophthalmol* 95: 99-108,1998.
5. Sutter FKP, Barmettler A, Helbig H. Sickie form macular whitening in a child after viewing a solar eclipse. *B J Ophthalmol* 84: 1433-4, 2000.
6. Michealides M, Rajendram R, Marshall J, Keightley S. Eclipse retinopathy. *Eye* 15: 148-51, 2001.
7. Wong SC, Eke T, Ziakas NG. Eclipse burns: A prospective study of solar retinopathy following the solar eclipse. *Lancet* 357: 199-200, 2001.
8. Bechmann M, Eht O, Thiel MJ, Kristin N, Ulbig MW, Kampik A. Optical coherence tomography findings in early solar retinopathy. *B J Ophthalmol* 84: 547-8, 2000.
9. Sadun AC, Sadun AA, Sadun LA. Solar retinopathy: A biophysical analysis. *Arch Ophthalmol* 102: 1510-12, 1984.
10. Mainster MA. Solar eclipse safety. *Ophthalmol* 105: 9-10,1998.
11. Stalmans P, Weckhuysen B, Schoonheydt R, Leys A, Missotten L, Spileers W, Dralands L. How to protect your eyes from solar retinopathy. *Bull Soc Belge Ophthalmol* 272: 93-100, 1999.
12. Lee BL, Sternberg P. Solar retinopathy In: Phototoxicity of the macula. *Ophthalmol Clin North America* 6: 231-237,1993.
13. Istock TH. Solar retinopathy: a review of the literature and case report. *J Am Optom Assoc* 56: 374-82,1985.
14. Kerr LM, Little HL. Foveomacular retinitis. *Arch Ophthalmol* 76: 498-504,1996.
15. Kozaki J, Takeuchi M, Takahashi K, Yamagishi K, Ohkuma H, Uyama M. Light induced retinal damage in pigmented rabbit 2. Effect of alpha-tocopherol. *Nippon Ganka Gakkai Zasshi* 98: 948-954,1994.
16. Eht O, Tavcar I, Eckl-Titz G. Microperimetry and reading saccades in retinopathia solaris. Follow-up with the scanning laser ophthalmoscope. *Ophthalmologie* 96: 325-31, 1999.
17. Kawa P, Mankowska A, Mackiewicz J. Solar Retinopathy. *Klin Oczna* 100: 235-237, 1998.
18. De La Paz MA, D'amico DJ. Photic retinopathy. Principles and Practice of Ophthalmology (Eds: Albert DM, Jacobiec FA, Robinson NL). Saunders Company Philadelphia-1994. pp 1032-7.
19. Atmaca LS, Idil A, Can D. Early and late visual prognosis in solar retinopathy. *Graefes-Arch-Clin-Exp-Ophthalmol* 233: 801-4, 1995.
20. Rosner M, Lam TT, Fu J, Tso MO. Methylprednisolone ameliorates retinal photic injury in rats. *Arch Ophthalmol* 110: 857-861,1992.