Brit. J. Ophthal. (1959) **43**, 366.

SOLAR RETINITIS *

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SOLAR retinitis has frequently been reported after a solar eclipse (Wadensten, 1947; Knudtzon, 1948; Flynn, 1952; Nirankari, 1954; Malik, 1957). Cases of solar burning can also arise at other times, when sun-gazing is practised as a misconceived therapeutic measure to strengthen the eyes (Tower, 1948). We have collected a series of 56 cases in this country, fifty eyes affected after a total or partial solar eclipse and six after sun-gazing.

Material and Methods

56 eyes of thirty patients with solar retinitis, who attended the S.N. Hospital, Agra, or the associated hospitals of the G.S.V.M. Medical College, Kanpur, or in private practice, were examined.

A careful history was taken of the time spent gazing at the sun or solar eclipse as well as of the method used in looking at it. The subjective symptoms, refraction, age, and ophthalmoscopic findings were recorded.

Most of the patients were treated by retrobulbar injections of 1 ml. Priscol every alternate day. A total of ten or twelve injections was usually necessary. If both eyes were involved each eye was given a separate injection at one sitting.

Observations

The lesions were classified into four grades according to the ophthalmoscopic changes seen at the time of the first examination at the clinic (Table I).

Grade I: The macula was apparently normal.

Grade II: The macula was congested and was surrounded by an oedematous area of retina.

Grade III: A greyish-white patch surrounded the fovea which was surrounded in turn by a black pigmentary ring.

Grade IV: Macular cyst, macular hole, and gross pigmentary changes.

TABLE I
INCIDENCE OF VARIOUS GRADES OF SOLAR RETINITIS

Macular Lesion After	Grade			
	I	II	III	IV
Solar Eclipse	2	23	16	9
Sun-gazing	1	2	2	1
Total	3	25	18	10

* Received for publication June 24, 1958.

The patients had observed the solar eclipse through crossed fingers, smoked glasses, dark plane lenses, shadow reflexion in a shallow metallic pot of water, and directly. There was hardly any difference in the lesion which could be attributed to the mode of observation.

The symptoms appeared at various intervals, usually within 1 to 4 hours of looking at the eclipse. The onset was insidious in cases of sun-gazing, and the duration of the development of the symptoms could not be ascertained.

The subjective symptoms varied and their severity bore little relation to the objective signs present. Some complained of a dazzling sensation, and clouding of vision was present in all cases, especially in the earlier stages. Metamorphopsia, micropsia, disturbance of colour vision, translucid scotoma, photophobia, and persistent after-images were commonly complained of, but none of these was universal. Most of the eyes in Grade III were those of patients who had seen the total eclipse of the sun, and those in Grades I and II were of patients who had observed a partial eclipse.

Three patients who refused treatment but remained under observation are rather interesting as examples of the evolution and final pattern of the lesions.

Case 1, a man aged 20 years, complained of photophobia, glare, and diminution of vision after looking at a partial eclipse. The macula was apparently normal, and he was advised to take the treatment for eclipse blindness to which he paid no heed; 4 days later the macular area was congested and red and was surrounded by an oedematous area of retina. A month later in the macular area there was a greyish-white patch surrounding the fovea which in turn was surrounded by a black pigmentary ring. He did not report for further observation.

Case 2, a woman aged 35 years, complained of a translucid scotoma when she first visited the hospital, and gave a history of having observed a total solar eclipse a month earlier. The macular area was congested and red and was surrounded by an oedematous area of the retina which was turning greyish-white. After about 2 months she had gross diminution of vision. The fovea was surrounded by a greyish-white patch, which in turn was surrounded by a black pigmentary ring. She at length agreed to have retrobulbar Priscol therapy but eventually progressed to gross pigmentary degeneration of the macular area with no visual improvement.

Case 3, a man aged 23 years, was in the habit of sun-gazing. One day he saw a black spot moving in front of the right eye, which he used in gazing at the sun. A greyish-white patch surrounded the fovea, and around it was an oedematous area of retina progressing towards pigmentary degeneration. It eventually progressed to an appearance of macular hole.

Results of Treatment

The response of cases treated with Priscol is summarized in Tables II and III (overleaf).

Discussion

Our cases are of interest as we have been able to observe most of them within a short time after exposure (not later than a week). Most of the patients had developed symptoms and perhaps signs within 4 hours of gazing

TABLE II
RESPONSE TO RETROBULBAR PRISCOL THERAPY

Total No. of Cases Observed	Number Refused Treatment	Number Treated	Response		
			Good	Fair	Failure
56	10	46	18	20	8

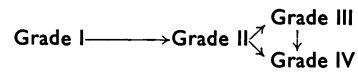
TABLE III
RESPONSE TO PRISCOL THERAPY ACCORDING TO GRADE

Grade	Number Treated	Response		
		Good	Fair	Failure
I	2	2	Nil	Nil
II	22	15	7	Nil
III	15	2	5	8
IV	7	Nil	Nil	7
Total	46	19	12	15

at the sun during the eclipse. Nearly all the patients showed micropsia and metamorphopsia followed by blurring and misting of the vision. Some showed disturbance of colour vision and a large number had translucid scotoma (blurring in a particular area of visual field as if one is seeing through mist). In some cases absolute and/or positive scotoma developed later.

The symptoms were closely related to the severity of the fundus lesion. In Grade I the usual symptoms were metamorphopsia, micropsia, photophobia, glare, and diminution of vision. In Grade II there was blurring of vision, fogging, and translucid scotoma. In Grades III and IV absolute and positive scotoma with gross diminution of vision occurred.

From the cases observed it seems that the severe grades of solar retinitis had initially passed through milder grades, through the following sequence:



The three patients who refused treatment bear testimony to this assumption. Even some of those who were under treatment in Grade I and in whom the lesions progressed in spite of treatment followed the same sequence. However transitory the stages may be, the lesions occur in this regular sequence.

Three patients who had observed the eclipse with both eyes and had one amblyopic eye developed the lesion only in the non-amblyopic eye. This is rather difficult to explain. Has the amblyopic eye some resistance to the absorption of the visual energy, is there a lack of concentration of visual

energy at the macula due to the defective focusing mechanism, or does some special substance present in normal retinae but absent from amblyopic eyes absorb visual energy?

Birch-Hirschfeld (1904) was of opinion that both the visible radiation without infra-red rays and pure infra-red rays are capable of producing this lesion. Duke-Elder (1954) considers that the amount of active energy is in itself sufficient to produce this lesion, and that the harmful effects of these concealed visible rays are hardly ever realized during an eclipse. Our cases not only support the view of Duke-Elder but show that the severity of the lesion depends on the amount of visual energy absorbed. The cases which occurred after a partial eclipse were milder because the patient had less time to gaze at the sun and less visual energy was absorbed. The severer cases occurred after a total eclipse which permitted them to gaze at the sun for a much longer time. In considering the nature of solar retinitis, we feel that the solar energy excites an angiospastic phenomenon leading to micropsia, metamorphopsia, and diminution of vision without observable macular signs. This leads to temporary ischaemia and exudation, and the constriction results in a dilatation of the peripheral capillaries creating a state of peristasis with the transudation of the fluid into the surrounding tissue.

The diminution of vision in Grade I is probably due to a temporary lessening or cessation of the macular circulation leading to partial or total ischaemia. In Grade II the visual disturbance is associated with macular oedema, but the visual change is probably the result of continual vascular spasm and disturbance of nutrition. In these two grades gross organic changes do not occur and the pathology is reversible. Priscol therapy in these two grades gave good or fairly good results leading to the full restoration of vision.

In Grade III the result of Priscol therapy was equivocal. More than half the cases showed no response, two cases resolved completely (13·3 per cent.), and five showed a fair degree of improvement (33·3 per cent.). The response to therapy could not be linked with apparent clinical changes at the macula, but the two cases which showed complete resolution were on the border line between Grades II and III. In some cases the organic changes in the retina are not so far advanced that reversal is impossible though clinically the accumulation of pigment may indicate otherwise.

The results of Priscol therapy in all Grade IV cases were poor, the macular area having undergone ischaemic necrosis.

Summary

(1) 56 cases of solar retinitis were observed, six after sun-gazing and fifty after looking at a solar eclipse.

(2) No unusual symptoms were recorded.

(3) Four grades of solar retinitis were seen.

(4) The severity of the lesion was dependent upon the amount of visual energy absorbed.

(5) It is suggested that the amblyopic eye resists the visual energy stimulus, or else there is a lack of concentration of visual energy at the macular area either through a defective focus mechanism or through lack of the normal visual chemical substance.

(6) The basic pathological defect in solar retinitis is probably identical to central angiospastic retinopathy, *i.e.* a vasospasm and diminution of retinal nutrition in that area cause ischaemia.

(7) In all cases of Grades I and II and some cases of Grade III, the retrobulbar injection of 1 ml. Priscol is the treatment of choice. Priscol probably acts by vasodilation and improves the nutrition of the retina.

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