SEVERE SOLAR MACULOPATHY ASSOCIATED WITH THE USE OF LYSERGIC ACID DIETHYLAMIDE (LSD)

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Patients with solar maculopathy typically have a mild to moderate decrease in vision and usually evidence minor changes ophthalmoscopically in the foveal area. Severe macular involvement may occur with sun gazing, particularly when the sun exposure is related to the use of lysergic acid diethylamide (LSD).

The association of solar maculopathy with hallucinogenic drugs is a recent observation. Ewald[1] in 1971 reported nine Army enlisted men with macular damage incurred by sun gazing during LSD "trips" or during "flashback periods." Schatz and Mendelblatt[2] reported two cases of LSD-related solar maculopathy in civilians in 1973. Schatz and Mendelblatt's patients had visual acuities of 20/30 or better in each eye when initially seen and had acuities of 20/20 bilaterally four months after the injury. Ewald did not report visual acuity. In 1975, von Domarus[3] described a patient with solar maculopathy associated with LSD ingestion. Visual acuity improved from 20/50 and 20/35 initially to 20/20 in each eye during seven months of observation.

CASE REPORT

On July 1, 1971, a 23-year-old white man was led into the eye clinic complaining of severe left ocular pain and blurred vision in both eyes. The patient said that he had taken a large oral dose of LSD the previous day at noon and had lain in the summer sun for four hours. He experienced visions of fiery volcanoes and waves of indescribably brilliant light. He recalled having had his arm over his right eye during part of the sun exposure. The patient then went to bed and slept until early morning when he awakened with severe pain in his left eye.

Sixteen hours after injury, first and second degree sunburns were present on his face and chest. He had severe actinic keratoconjunctivitis of the left eye. Mild epithelial edema was noted in the right eye. Best corrected visual acuity was R.E.: 20/200, and L.E.: counting fingers at 5 feet. The right fundus revealed absence of the foveal light reflex, a punctate, yellow, deep retinal foveal exudate, and moderate macular retinal edema (Fig. 1). The fundus of the left eye was difficult to see because of clouding of the cornea, but severe macular edema was detected. Amsler grid examination showed a 3-degree central scotoma in the right eye and a 12-degree central scotoma in the left eye. Prominent metamorphopsia was noted for both eyes. The patient stated that both eyes had previously been entirely normal.

Seven days after injury visual acuity was R.E.: 20/50, and L.E.: less than 20/400. The deep foveal exudate initially present in the right eye was much smaller and was surrounded by a red halo (Fig. 2). Mottling of the retinal pigment epithelium was present temporal to the main lesion. The left macula presented a striking picture of a "bull's eye" lesion with a hyperpigmented center surrounded by rings of alternating decreased and increased pigmentation (Fig. 3). Prominent retinal folds radiated from the lesion.

At six weeks, visual acuity was R.E.: 20/50, and L.E.: less than 20/400. A small focus of decreased

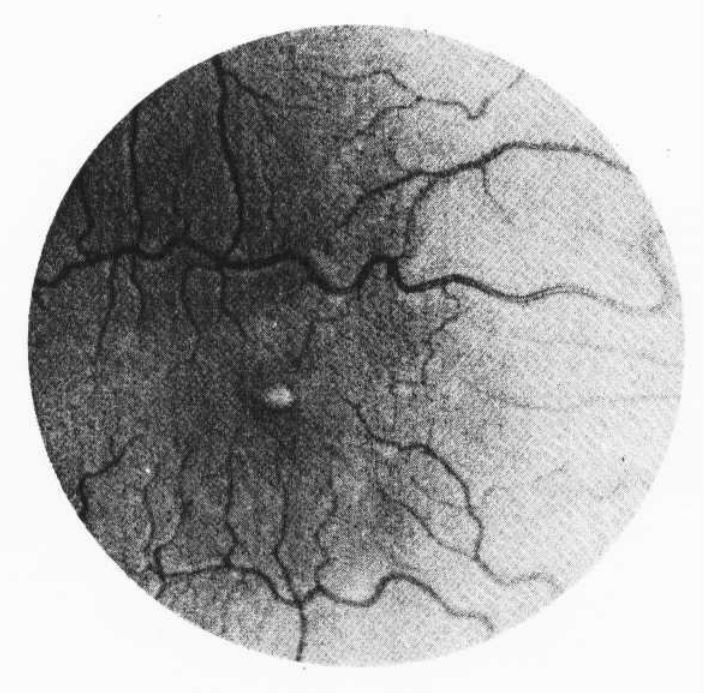

Fig. 1 (Fuller). The right macula 24 hours after solar injury. A punctate, deep retinal foveal exudate is clearly visible.

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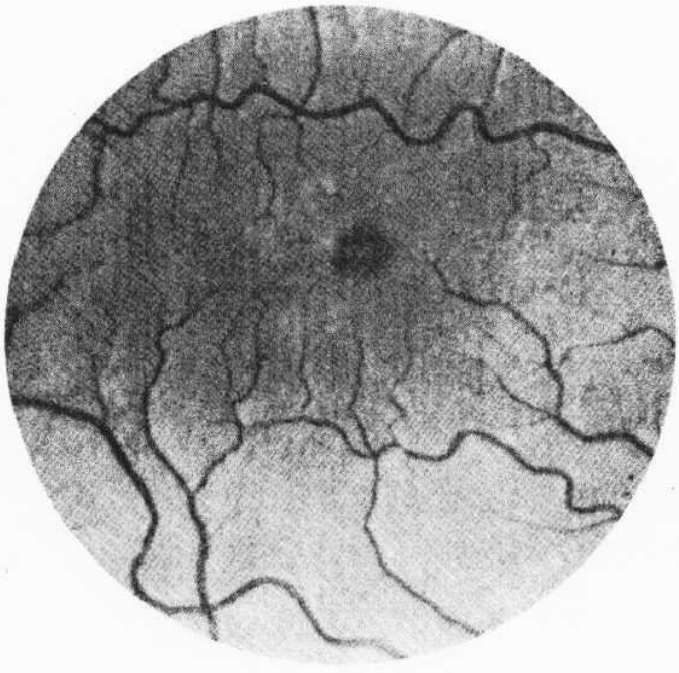

Fig. 2 (Fuller). The right macula seven days after injury. A halo surrounds the original foveal exudate which is now much smaller. Mottling of the retinal pigment epithelium is seen temporally to the main lesion.

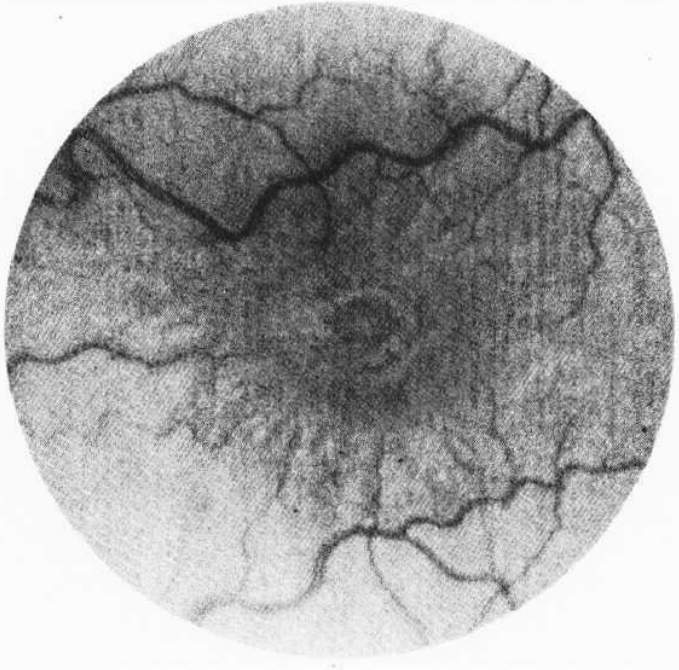

Fig. 3 (Fuller). The left macula seven days after injury. Striking picture of bull's eye lesion with hyperpigmented center surrounded by rings of alternating decreased and increased pigmentation. Note prominent retinal folds.

pigmentation was evident near the right fovea (Fig. 4). The left macula had a large central area of hyperpigmentation dotted with coarse pigment clumps in a zonular pattern (Fig. 5). A halo surrounded the main lesion. Radiating retinal folds were now barely discernible.

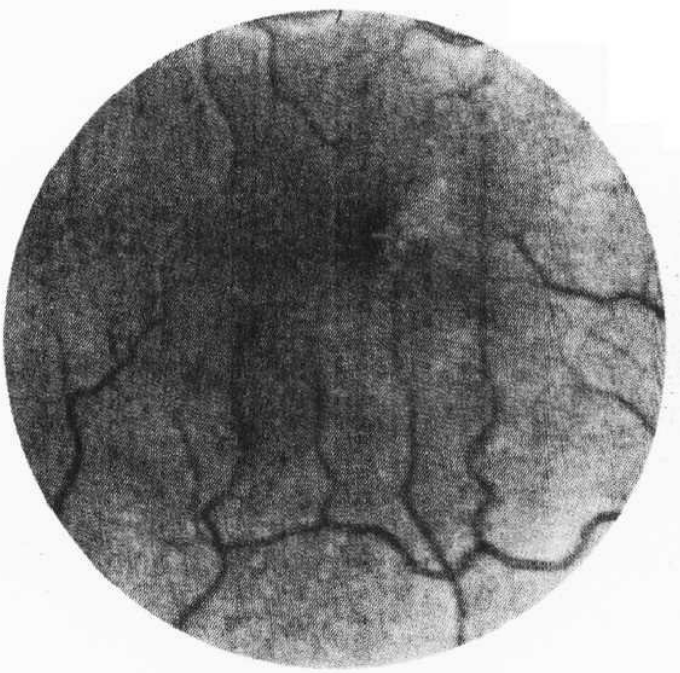

Fig. 4 (Fuller). A magnified view of the right macula six weeks after injury. A small area of decreased pigmentation is evident in the foveal region. Visual acuity is 20/50.

The patient was observed for four months before he was lost to follow-up. Final visual acuity was R.E.: 20/30— at distance (searching) and J2 at near, and L.E.: 5/400 with eccentric viewing at distance and J12 at near. A small central scotoma was demonstrated in the right eye; a dense 3-degree central scotoma remained in the left eye. Examination of the macula with a contact lens revealed a small irregular area of depigmentation of the retinal pigment epithelium near the right fovea. The overlying retina appeared thin but no definite retinal hole was present. The macula of the left eye remained grossly abnormal with large clumps of pigment scattered over a central oval area of hypopigmentation. The overlying retina appeared atrophic, but there was no true retinal hole. A distinct foveal light reflex was present.

The patient received 60 mg of orally administered prednisone daily within the first 24 hours of solar injury with gradual tapering over a six-week period. In addition, the more severely involved left eye had three subtenon's injections of corticosteroids during the first 12 days.

Fluorescein angiography was performed one, nine, and 19 days after solar damage occurred. Corneal edema of the left eye precluded angiography on day 1, but both eyes were studied on days 9 and 19. Angiography of the right eye gave normal results on day 1 (Fig. 6) and on the next two examinations. The initial fluorescein study of the left eye on day 9 showed a bull's eye pattern with central blockage of choroidal fluorescence surrounded by two concentric rings of mottled hyperfluorescence (Fig. 7). The angiographic studies of the left eye were unchanged on day 19. No fluores-

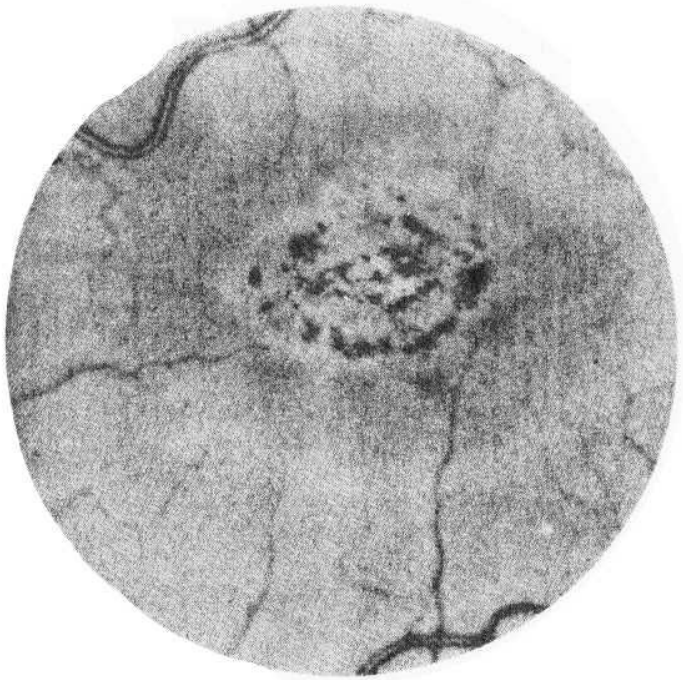

Fig. 5 (Fuller). A magnified view of the left macula six weeks after injury. Severe retinal damage is obvious. A large central oval of hypopigmentation is dotted with coarse pigment clumps in a zonular pattern. Visual acuity is less than 20/400.

cein leakage was demonstrated in either eye at any time.

Discussion

Sequential fundus photographs and fluorescein angiography give clear evidence of major damage to retinal pigment epithelium caused by protracted sun gazing. Typical cases of solar maculopathy show ophthalmoscopic changes confined to the foveal area, normal fluorescein patterns with no retinal vessel leakage, and no transmission of choroidal fluorescence through retinal pigment epithelial "window" defects (J. D. M. Gass, personal communication, March 11, 1975). Ewald and Ritchey[4] reported one instance of transmission of early choroidal fluorescence in a case of late stage solar maculopathy. They noted four other late cases of solar maculopathy with questionable foveal holes that appeared to be normal by angiography.

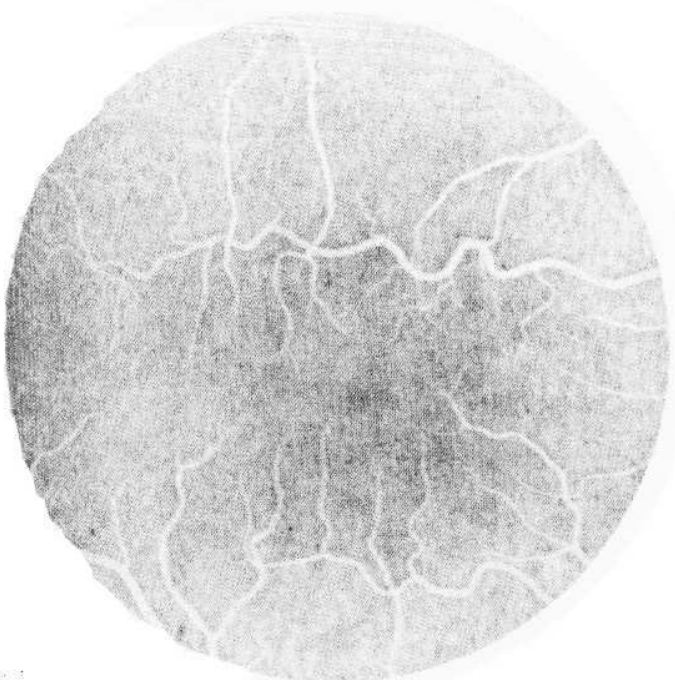

Fig. 6 (Fuller). Normal fluorescein angiogram of right macula 24 hours after injury.

There is evidence that sun gazing causes so-called foveomacular retinitis[4] that typically seems to be normal by fluorescein angiography at all stages of the disease.[5,6] Hunter Little, however, noted parafoveal fluorescein leakage by slit-lamp angioscopy in acute cases of foveomacular retinitis on rare occasions (personal communication, March 21, 1975).

LSD is the most potent psychotogen known. Small doses cause profound mood changes with dramatic distortion of visual, tactile, and auditory perception.[7] The vivid imagery evoked by sun gazing while drugged encourages prolonged exposure. Sun gazing with LSD is particularly dangerous because of the drug-induced mydriasis.[8] Also, LSD may cause partial cycloplegia in humans, removing yet another ocular defense against light damage.[9]

The psychologic and physiologic changes induced by LSD may promote major visual impairment if related to sun gazing. My patient experienced more severe retinal damage than is typically seen in cases of solar maculopathy.

Summary

A 23-year-old man sustained severe macular damage by sun gazing during a hallucinogenic drug-induced state. Sequential fundus photography and fluorescein angiography documented prominent focal injury to the retinal pigment epithelium.

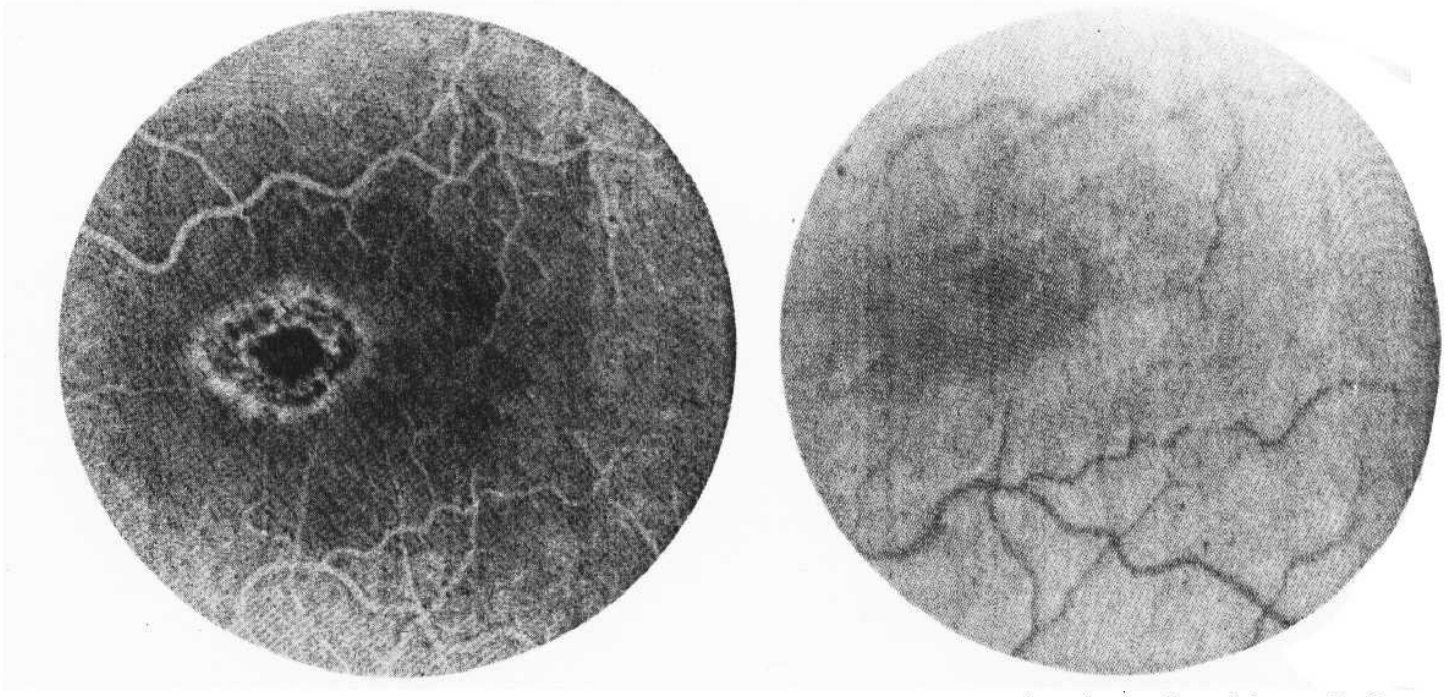

Fig. 7 (Fuller). Left, Early fluorescein angiogram of left macula nine days after injury. Bull's eye pattern with central blockage of choroidal fluorescence is surrounded by two concentric rings of transmission hyperfluorescence. Right, Late phase of angiogram of left macula nine days after injury. No fluorescein leakage has occurred.

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