Solar maculopathy

Solar burn, Phototoxic maculopathy, Eclipse blindness, Foveomacular retinitis

DESCRIPTION

Solar maculopathy is a degeneration of the macula associated with phototoxicity 'excessive light'. It is considered to particularly result from photochemical damage to the retina associated with ultraviolet (UV) radiation from the sun, although thermal effects associated with longer wavelengths may play an additive role. Damage to the retina and retinal pigment epithelium (RPE) from the sun is dependent upon the time of exposure, solar factors including the position in the sky and ocular factors including the crystalline lens spectral absorbance and the individual level of pigmentation. There is usually intense bleaching and shedding of photoreceptor outer segments and loss of RPE function.

Other phototoxic maculopathies have also been reported:

- Ophthalmic instruments or the operating room lamp, if ultraviolet radiation is not filtered out
- Welder's maculopathy can occur, usually accompanied by the more common photokeratitis. If severe, a macular hole may form
- Laser exposure. The type of retinal damage depends upon the wavelength of light, exposure time and power level. A laser (thermal) burn such as from krypton or argon laser is seen almost immediately, unlike a phototoxic burn.

SYMPTOMS

There is usually a history of solar exposure or viewing an eclipse. Typically, symptoms first occur several hours after exposure. There may be complaints of blurred vision, central or paracentral scotomata, metamorphopsia, chromatopsia or headache.

SIGNS

Vision may be decreased to 20/40 or even 20/100, often bilaterally. A solar maculopathy is usually less than 0.2mm in diameter, corresponding to the size of the retinal image of the sun. First signs of phototoxicity occur in the first one to two days, with a small yellowish foveal or parafoveal lesion and mild pigmentary changes and retinal oedema. The yellow spots are intraretinal, presumed to be xanthophyll pigment. Chronic RPE pigmentation effects are variable, ranging from de-pigmentation to hyper-pigmentation or RPE hyperplasia. Classically, the chronic lesion is described as reddish with sharply demarcated edges, somewhat like a stage 1A macular hole.

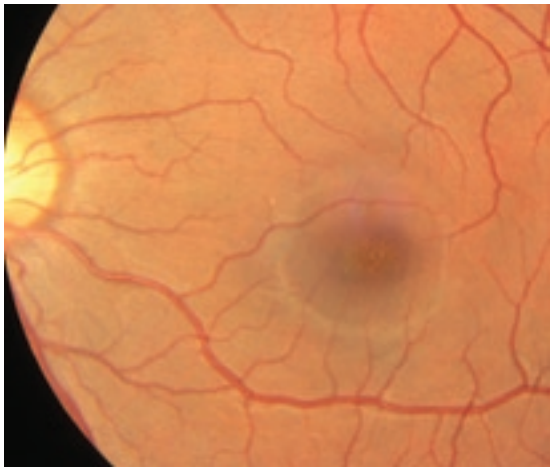

Figure 1 Central solar maculopathy, several weeks after exposure. The image shows loss of some central neural elements and retinal pigment epithelium and surrounding pigmentary changes

PREVALENCE

The condition is rare (approximately 1/10,000) and mainly reported in at risk groups: Military personnel, sun bathers, religious sun gazers, solar eclipse viewers and users of psychotropic drugs.

SIGNIFICANCE

Solar maculopathy is a preventable cause of visual loss.

DIFFERENTIAL DIAGNOSIS

Macular hole (Stage 1), central serous chorioretinopathy, macular oedema, age-related macular degeneration.

SEE ALSO

Stargardt's disease, Cone dystrophy.

MANAGEMENT

Additional investigations

Amsler grid will often demonstrate central or paracentral distortion (metamorphopsia). Fluorescein angiogram may show a small window defect or be normal. There may be chronic decompensation of the blood retinal barrier.

Ocular coherence tomography enables assessment of damage to the individual layers of the retina and RPE over time. Initially, the retinal layers show increased reflectivity, with changes to the RPE and choroid appearing one to two weeks later. In the chronic condition, months to years later, a lamellar retinal hole is usually evident corresponding to the damaged photoreceptors and RPE. The overlying neurosensory retina is attenuated and thinned. The vitreoretinal interface will be normal.

Advice

Prevention is the preferable treatment for solar maculopathy, since there is no specific treatment once damage has occurred. Patients and the public may benefit from health education programs regarding sun-gazing, particularly prior to a solar eclipse or in areas with a reduced ozone layer.

Prognosis

Generally, retinal tissue damage due to solar maculopathy is reversible to some extent, with improvement in acuity occurring within two to four weeks of exposure. Some patients experience a small persistent scotoma despite restoration of normal acuity. If there has been extensive damage to the photoreceptors and the RPE (initial acuity 20/200 or worse), there may be localised retinal atrophy and degeneration, with less favourable visual prognosis.

The full series of these articles is available in the book *Posterior Eye Disease and Glaucoma A-Z* by Bruce AS, O'Day J, McKay D and Swann P. £39.99. For further information click on the Bookstore at **opticianonline.net**

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