Welding arc maculopathy and fluphenazine

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Abstract
A 45-year-old male patient presented with a bilateral maculopathy following unprotected exposure of less than two minutes’ duration to a manual metal arc welding unit. He had been receiving the drug fluphenazine for the previous 10 years for treatment of depression. We believe that the drug fluphenazine, which had accumulated in his retinal pigment epithelium, may have rendered him particularly susceptible to retinal photic damage.

The retina is susceptible to at least three different forms of light damage – thermal, mechanical, and photochemical.123 Thermal damage is produced when the retina is exposed to a temperature rise of 10° to 20°C. Photochemical damage most likely results from longer exposure to bright sources that produce a considerable amount of blue or near ultraviolet light.4 Light levels required to cause acute photoretinitis are encountered by exposure only to very bright light sources, such as the sun, welding arcs, arc lamps, and tungsten halogen lamp filaments. The type of retinal damage depends on wavelength, energy level, duration of exposure, and degree of pigmentation.

The radiation emitted from welding units covers a wide spectrum, from infrared to ultraviolet and beyond. Excessive exposure to ultraviolet radiation can result in photophthalmia (‘welder’s flash’) and may, under chronic exposure, result in UV cataract.56 Retinal injuries resulting from exposure to welding arc radiation have been reported but are not commonly seen.578 We report here a case of welding arc maculopathy in a patient who had been taking the phenothiazine drug fluphenazine.

Case report
A 45-year-old man noticed bilaterally decreased visual acuities soon after welding. There was no accurate record of the exact duration of exposure. However, the patient’s instructor, who was observing him from a distance, estimated it to be certainly no longer than two minutes. The patient had just begun a training course and had no previous welding experience. He had raised the protective visor which he was wearing in order to see more clearly. This was not noticed by the instructor as the patient was crouched and had his back towards him. He had been using a manual metal arc welding unit with a flux coated rod.

When seen the next day the patient complained of bilateral scotomata and metamorphopsia. At no time did he complain of ocular pain. His best corrected visual acuity was 6/18 in the right eye and 6/12 in the left. The anterior segments were normal. There was no evidence of a punctate keratopathy when the corneas were stained with fluorescein. There was a circular yellow oedematous lesion approximately 250 μm in diameter present at each fovea (Figs 1A, B). Fluorescein angiography carried out five days later was normal. In an effort to demonstrate the capillary-free zone better an enhanced fluorescein angiogram was prepared. The angiogram negative was printed in contact register with an unsharp mask to produce the finished result. This enhanced angiogram demonstrated mild diffuse fluorescence in the foveal avascular zone (Fig 2). The patient was followed up on a regular basis for the next six months. During this period his visual acuities returned to 6/5 in each eye. The results of Amsler grid testing and pattern electroretinograms were also normal in both eyes. Examination of his
Typically, patients with maculopathy complain of a central scotoma and metamorphopsia. Bilateral yellow oedematous lesions at the fovea are characteristic. The lesions measure approximately 200 \( \mu \text{m} \) in diameter, even though the size of the arc image is only 20 \( \mu \text{m} \). The 200 \( \mu \text{m} \) corresponds to the area about which a fixation point moves because of normal eye movements. The raised lesions, which represent acute retinal oedema, gradually subside over several weeks and are replaced by motting of the pigment epithelium or occasionally by a small lamellar hole. Most patients recover normal vision, though a few experience a permanent visual deficit.

One of the unusual features of this case is that though the patient was only welding for two minutes he developed a bilateral maculopathy without any evidence of a keratitis. The traditional welding rod, which was used by this patient, produces a relatively greater amount of radiation in the ultraviolet range than the newer metal-arc inert gas welding units. The fact that the patient did not develop a keratitis while exposed to this ultraviolet enriched radiation would seem to confirm the short duration of exposure as reported to us by both the patient and his instructor.

All phenothiazine drugs are deposited in the retinal pigment epithelium to some extent, and most are concentrated there at levels far higher than those found in other body tissues. Quite a number of the phenothiazines, including fluphenazine, have been associated with photosensitising rashes. These drugs may act as psoralens, and it has already been shown that photosensitisation cataractogenesis can be produced in rat lenses when they are incubated with the drug chlorpromazine and irradiated with ultraviolet light.

We postulate that this patient sustained macular burns from welding because the drug which had accumulated in his retinal pigment epithelium over the years may have acted as a photosensitising agent, thereby rendering his retina particularly susceptible to photic damage. The potential role of fluphenazine in this case

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**Discussion**

A wide variety of ocular changes are produced by welding arcs, the type of change depending on the wavelength of the arc emissions, the duration of the exposure time, and the power level. The commonest is a keratoconjunctivitis. This is caused by the ultraviolet radiation, which the welding arc produces in large amounts. Welding arc emissions, however, cover quite a wide spectrum, much of which falls within the short or blue wavelengths in the visible spectrum. Photochemical damage to the retinal pigment epithelium and photoreceptors, especially cones, is responsible for retinal damage below photocoagulation threshold. The damage produced is much more pronounced for the short or blue wavelengths in the visible spectrum and is enhanced by increased retinal temperatures. Welding arc maculopathy almost certainly represents an example of photochemical injury.

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**Figure 2** Enhanced fluorescein angiogram of the right eye, demonstrating fluorescence in the foveal avascular zone.

Fundus at the six-month review showed minimal stippling of the pigment epithelium surrounding each fovea (Figs 3A, B).

The patient was being treated with the drug fluphenazine for endogenous depression. He received 12.5 mg intramuscularly fortnightly for 10 years without interruption. The total estimated dose was calculated to be 3.25 g.

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**Figure 3A** Right eye.

**Figure 3B** Left eye.

**Figure 3A, B** Photographs taken at six months showing pigmentary disturbance at either fovea.
remains conjectural. However, we believe this is the reason why the patient developed the maculopathy after such a short period of welding.

All patients should take full precautions when welding to avoid inadvertent retinal damage. Patients taking phenothiazine drugs may be at special risk and should be advised accordingly.

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