

Ocular damage caused by Paraquat

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Paraquat is a well-known ingredient of many domestic and agricultural weed-killers (1,1'-dimethyl-4,4'-dipyridilium). It is now in widespread use and several fatal cases have been reported after accidental ingestion, proving its highly toxic properties (Bullivant, 1966). *Post mortem* findings in these cases show its effects, particularly on the mucosa of the alimentary tract (oesophagus and stomach), lungs, liver, and kidneys.

Little has yet been reported of its effects on ocular tissues; two cases are presented here showing the effect of direct contact with the eye.

Case 1

A gardener aged 57 years attended the out-patients department, having noticed a rapid reduction in the visual acuity of the left eye over the previous 4 weeks. He gave a history of having been splashed in the eye with a liberal quantity of Paraquat solution 4 weeks before. This was treated initially by the works nurse, who referred the case to his general practitioner when, after 7 days, he noticed a reduction in visual acuity. Immediately after the injury, however, he had had little discomfort.

Examination

The eye was white, visual acuity was perception of light, and a large lower corneal opacity was evident. This was circular and approximately 5 mm. in diameter (Fig. 1). Some peripheral infiltration and fluorescein staining were evident on examination.

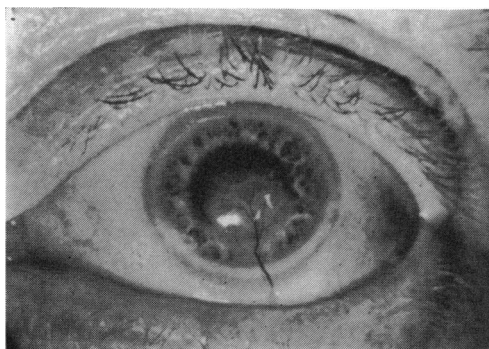


FIG. 1 Case 1 *Clearly-defined area of keratitis after Paraquat splash*

The area of keratitis involved the epithelium and the superficial part of the stroma. The anterior chamber showed no signs of inflammation.

Treatment

The eye was treated initially with gutt. atropine and gutt. chloramphenicol and after 2 weeks the peripheral infiltration and staining had disappeared. The cornea remained quiet and 5 months after the first visit a 6 mm. penetrating keratoplasty was carried out.

Histology

The corneal disc (Fig. 2) showed that the corneal epithelium had reformed, but was thickened in the centre, where Bowman's layer was missing. Elsewhere this layer was fragmented. A pannus degenerativus composed of recent collagen was present under the epithelium and included a few zones in which necrotic material persisted. In the deeper part of this pannus, there was a band of plasma cells and small vessels, parallel to the surface. Descemet's membrane was normal, as was the endothelium.

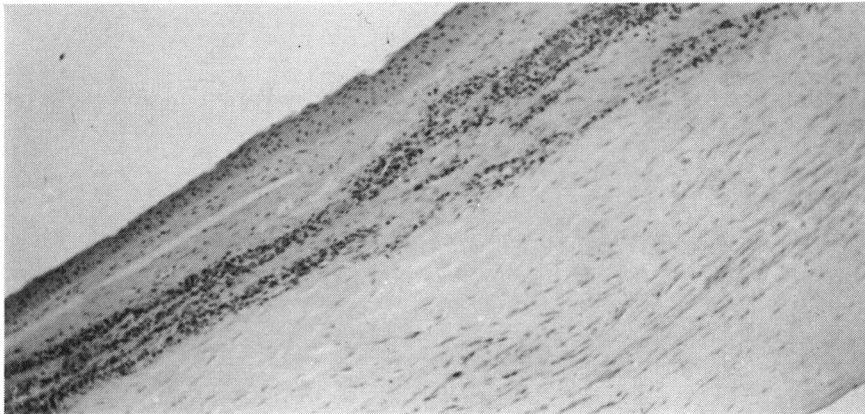


FIG. 2 Case 1 Photomicrograph of corneal disc section, showing loss of Bowman's layer. $\times 105$

Case 2

A 51-year-old housewife was accidentally splashed in the left eye during fruit spraying with a Paraquat solution, and 2 hours later the eye became sore and red. She was treated with oculentum chloramphenicol in the first instance by her general practitioner and 10 days after the incident she was first seen at the out-patients department.

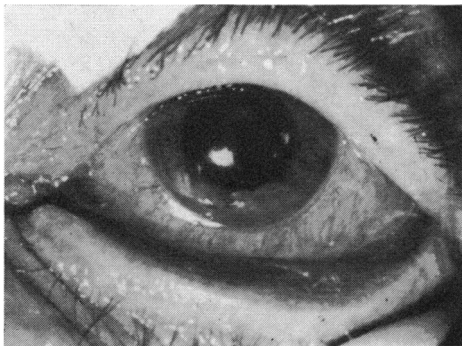


FIG. 3 Case 2 Conjunctival necrosis in lower fornix after Paraquat splash

Examination

The eye was injected and an area of conjunctival necrosis was evident in the lower fornix (Fig. 3). The cornea was healthy.

Treatment

She was given gutt. Betnesol and gutt. homatropine. The necrotic area remained localized, but was slow to heal, and 10 weeks after the incident some local congestion was still apparent.

Discussion

From the two cases presented, it is apparent that serious ocular injury and sequelae can occur as a result of splashing Paraquat into the eye.

In two previous papers (Clark, McEllegott, and Hurst, 1966; Cant and Lewis, 1968) it was noted that permanent corneal damage was not a feature of Paraquat burns; the first case reported here, however, clearly demonstrates the severe corneal damage that may occur in such cases. This, perhaps, is to be expected in view of the serious systemic disturbance that Paraquat can cause.

Summary

Two cases are presented of ocular damage caused by Paraquat, and the severity of lesions due to this substance is stressed.

References

- BULLIVANT, C. M. (1966) *Brit. med. J.*, **1**, 1272
CANT, J. S., and LEWIS, D. R. H. (1968) *Ibid.*, **2**, 224
CLARK, D. G., MCELLIGOTT, T. F., and HURST, E. W. (1966) *Brit. J. industr. Med.*, **23**, 126