

OCULAR INJURY CAUSED BY PREGLOX-L (A HERBICIDE CONTAINING PARAQUAT, DIQUAT, AND SURFACTANT). EFFECT OF TOPICAL REDUCED GLUTATHIONE.

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A 54-year-old man was splashed in the right eye with Preeglox-L (a herbicide containing paraquat, diquat, and surfactant). Three days after the topical exposure, conjunctival congestion, corneal edema, and Descemet's folds were observed. Instillation of 2% topical reduced glutathione decreased corneal edema temporarily. Six days after the exposure marked corneal epithelial defect occurred. Thereafter, the corneal epithelial defect gradually decreased. Topically applied reduced glutathione may be useful in the treatment of the early phase of ocular injury caused by Preeglox-L.

Key words: paraquat, ocular injury, herbicide, surfactant, glutathione

INTRODUCTION

Preeglox-L (ICI Japan, Tokyo) is a herbicide containing 5% paraquat, 7% diquat, and 3.2% polyoxyethylene as a surfactant (1). Topical exposure to the eyes of herbicides containing paraquat reportedly induce conjunctival congestion, persistent corneal epithelial defect, corneal edema, and iridocyclitis (1, 2). The corneal epithelial defect deteriorated one or two weeks after the exposure of Preeglox-L (1). We recently treated a patient with ocular injury caused by topical exposure of Preeglox-L.

CASE REPORT

A 54-year-old man was splashed in the right eye with Preeglox-L on August 20, 2001. He immedi-

ately washed his eye with a large amount of water, and visited Hori Eye Clinic. The patient complained of mild foreign body sensation. Superficial punctate keratitis was found in the right eye. His visual acuity was 1.0 OU. Topical instillation of 0.3% ofloxacin and 0.02% fluorometholone, four times daily, was prescribed. On August 23, the patient complained of severe ocular pain in the right eye, and was referred to Toyama Medical and Pharmaceutical University Hospital. His visual acuity was 0.2 OD and 1.0 OS. The intraocular pressures were 12 mmHg OU. The right conjunctiva appeared congested (Fig 1a). The right cornea stained with fluorescein (Fig 1b). The

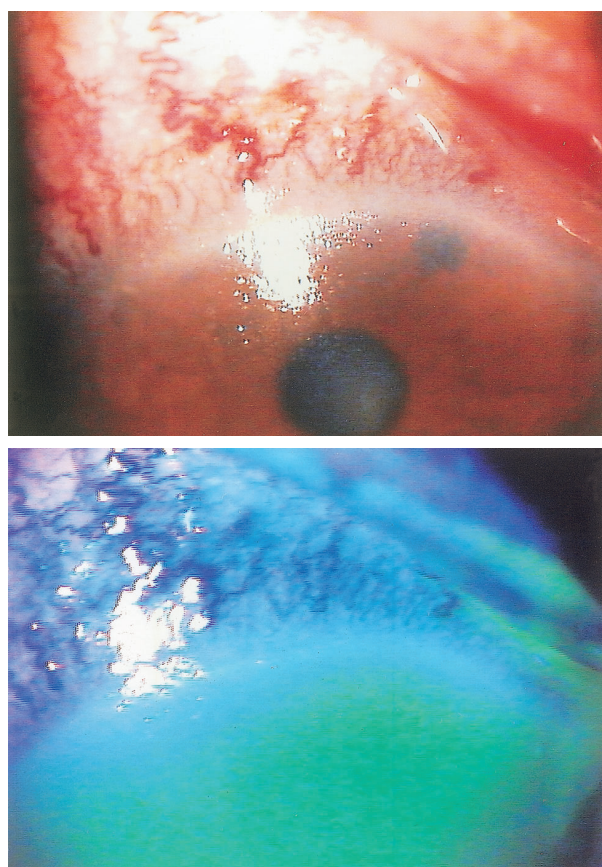


Fig. 1. On August 23, 2001 (three days after exposure), conjunctival congestion was seen in the right eye (a). The right cornea stained with fluorescein (b).

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pH of the tear fluid in the right cul-de-sac was 7.4. Culture of bacteria from the corneal surface showed negative growth. The right eye appeared otherwise normal. The left eye appeared normal. Topical instillation of 2% reduced glutathione, six times daily, was added to the treatment regimen. On August 24, a few areas of the right cornea stained with fluorescein (Fig. 2). The right visual acuity improved to 0.4. On August 26, the patient complained again of ocular pain. The right visual acuity decreased to 0.1. Marked corneal epithelial defect was found (Fig 3). Topical instillation of ofloxacin, fluorometholone, and reduced glutathione was continued. On August 30, fluorescein stained the central area of the cornea (Fig. 4). On September 5, superficial punctate keratitis was found. On September 12, the right visual acuity was 1.2. No corneal opacity was noted.

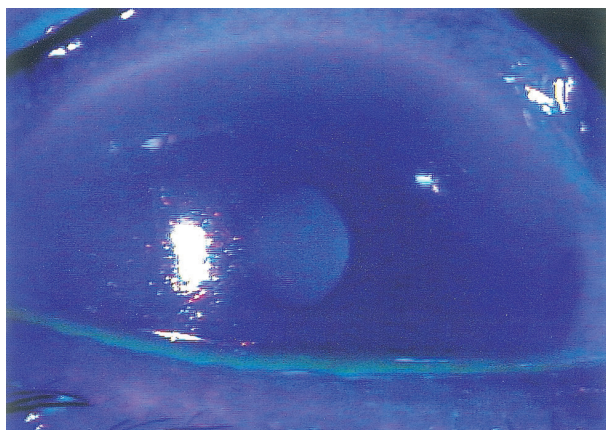


Fig. 2. After topical instillation of reduced glutathione, few areas of fluorescein staining were found on the right cornea on August 24, 2001.

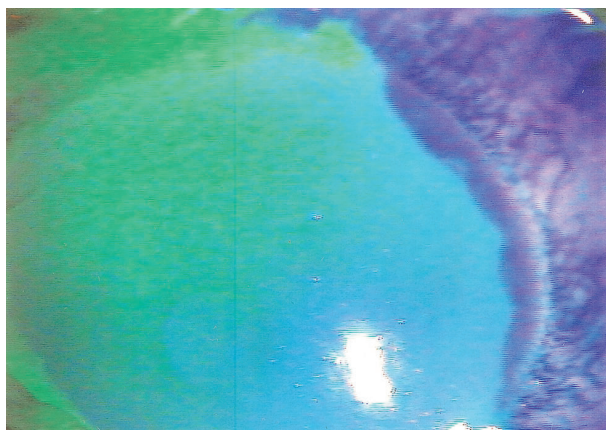


Fig. 3. Six days after exposure, fluorescein stained almost entire cornea.

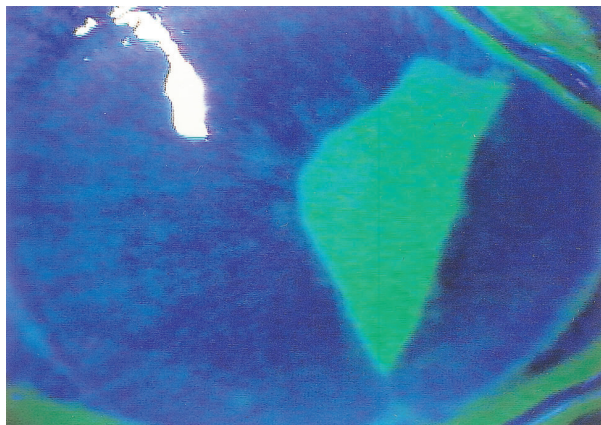


Fig. 4. Ten days after exposure, fluorescein stained the central area of the cornea.

DISCUSSION

Topical exposure of Preeglox-L induced mild superficial keratitis just after contact and severe corneal epithelial defect one or two weeks later; thereafter, the corneal lesions gradually healed (1). Our patient complained of mild foreign body sensation just after exposure. Superficial punctate keratitis was found at that time. Three days after the exposure, he complained of severe ocular pain. Corneal epithelial defect and conjunctival congestion were found. The described findings in our patient were similar to those of Preeglox-L-induced corneal lesions described by Nirei *et al.* (1) and were different from those observed in acid or alkali burns.

Nordquist *et al.* reported that paraquat generates oxygen-free radicals, and that the radicals induce corneal tissue injury (3). Hagen *et al.* reported that glutathione protects against paraquat-induced oxidative injury in pulmonary alveolar type cells (4). Nirei *et al.* also reported that Preeglox-L-induced corneal lesions deteriorated gradually, despite of topical instillation of steroid (1). Therefore, our patient was treated with topical instillation of reduced glutathione. In our patient, corneal edema decreased the next day after glutathione treatment, but recurred 6 days after the exposure. The turnover period of corneal epithelium is approximately 7 days(5). It is likely that the toxic mechanisms of Preeglox-L may be different before and after 7 days, and that the early phase (up to 7 days) of ocular lesions may be caused mainly by free radicals and that the delayed phase (from 7 days to one or two months) may be

induced by substances other than free radicals. Grant has reported that surfactant rather than paraquat or diquat is responsible for corneal injuries (6). It is likely that the surfactant contained in Preeglox-L may play a role in the pathogenesis of the delayed phase of the ocular lesion. Topical reduced glutathione may be effective in the treatment of the early phase of the ocular injury caused by topical exposure of Preeglox-L.

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