Ocular exposure to paraquat resulting in keratopathy, pseudomembranous conjunctivitis and symblepharon

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Paraquat is a powerful herbicide still used in rural areas of New Zealand. We report the case of an 82-year-old man who had accidental exposure of 50:1 diluted paraquat to his right eye. He immediately irrigated the eye and presented two days later to his general practitioner with worsening vision. He was then referred to the hospital ophthalmology department.

He was seen the same day with right visual acuity (VA) of 6/60 improving with pinhole (PH) to 6/30. Examination revealed severe papillary conjunctivitis with superior pseudomembrane and a devitalised corneal epithelium. There was no frank corneal epithelial defect or apparent limbal ischaemia. The pseudomembrane was debrided and he was started on hourly prednisolone (1.0%) drop (Bausch & Lomb, New Zealand) and chloramphenicol (0.5%) drop (Bausch & Lomb, New Zealand) four times daily (QID).

On day three he had worsening discomfort and erythema. Examination showed 200 degrees of superior limbal ischaemia with a shallow epithelial defect. Oral doxycycline 100mg once daily, oral vitamin C 1g (QID), topical sodium ascorbate 10% every two hours (Q2H) and topical sodium citrate 10% (Q2H) were added to the existing treatment.

By day five, comfort was improved, with vision stable at 6/48 PH 6/24. There was 270 degrees of limbal ischaemia, with a large (80%) non-healing epithelial defect. Symblepharon was divided with a glass rod. A bandage lens was placed in case mechanical irritation from conjunctival papillae was contributing to the non-healing epithelial defect.

On day nine an amniotic membrane graft was sutured to the limbus with interrupted 10–0 nylon and continuous 8–0 vicryl to the fornices. Complete epithelial healing occurred by day 14 post-operatively. Vision had improved to 6/15 PH 6/9. Chloramphenicol (0.5%) QID was continued for 14 days post-operatively and prednisolone drops (1.0%) tapered over two weeks. At two months post-operatively, vision was 6/9–1 PH 6/7.5 with mild inferior symblepharon.

Discussion

Paraquat is a non-selective herbicide. It is extremely toxic to humans and as treatment outcomes are extremely poor,1 it is banned in many countries, though not in New Zealand. Fortunately, paraquat is rapidly rendered biologically inactive on contact with soil, somewhat limiting its toxicity to the surrounding environment.2 It is a commonly used agent for self-poisoning and has life-threatening effects when ingested at 20mg/kg, primarily via toxicity of the pulmonary tract, kidneys, liver and heart.3 Its mechanism of toxicity is thought to be via generation of free radicals, resulting in oxidative damage due to depletion of NADPH. This occurs as paraquat recycles in the redox reaction interrupting cellular metabolism. Reduced paraquat then re-oxidises, using oxygen to generate a superoxide radical, which binds macromolecules and damages membrane lipids.1

The immediate effects of ocular exposure include irritation, lacrimation and conjunctivitis. Short-term effects occur one to four weeks later, including conjunctival defects,
corneal epithelium loss (limbal stem cells are especially vulnerable) and anterior uveitis. Long-term effects include chronic conjunctivitis, symblepharon and epiphora due to punctual stenosis. Corneal oedema, superficial scarring and recurrent ulceration are also common.2

Histologically, the conjunctiva may show sub-epithelial fibrosis. Impression cytology of the conjunctiva in the case presented by Vlahos2 showed variable grades of squamous metaplasia and keratinisation with inflammatory infiltrate. The cornea may show loss of Bowman’s layer, with epithelial thickening and pannus formation under these areas.

Periocular tissues may also be affected by paraquat exposure. Fortunately, absorption across the skin is slow and limited, though significant local toxicity may be evident with contact dermatitis, blistering and ulceration.2

Previous case reports of ocular paraquat injury have been uncommon but share generally long recoveries and poor visual outcomes. Both Vlahos2 and Cant4 described young adults accidentally exposed to paraquat who developed pseudomembranous conjunctivitis, punctual occlusion, corneal pannus as well as anterior uveitis and symblepharon, respectively. Mckeag2 described a bilateral exposure resulting in progressively deteriorating corneal epithelial defects which took one month to fully heal. Joyce6 presented a case of paraquat toxicity with delayed presentation four weeks post-injury requiring a penetrating keratoplasty due to persisting corneal opacity.

Treatment of paraquat injury is difficult and limited options are available.9 Recommended first aid for ocular exposure includes copious irrigation with water for 30 minutes.7 Initial treatment should include preservative free steroid, antibiotic and lubricants.1 The use of topical ascorbic and citric acid and oral ascorbate is less clear though may be useful. Autologous serum should be considered for non-healing epithelial defects.4 The use in this case of amniotic membrane graft to aid healing of the persistent and worsening corneal epithelial defect was successful and has been shown to be effective with similar ocular surface defects.9 More recently the modification of amniotic membrane transplantation with coverage of the entire ocular surface has shown promising outcomes both for vision and symblepharon prevention.9

Competing interests:
Nil.

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