INTRODUCTION: Alkalis possess both hydrophilic and lipophilic properties and have a higher pathological potential than acids to damage the eye. With the exception of hydrofluoric acid, acids penetrate much less readily into the corneal stroma than alkalis as corneal proteins bind acids thus preventing further penetration into the anterior chamber (1,2). Additionally, acids denature and precipitate tissue proteins and in doing so generate physical barriers to deeper penetration. In alkalic substances, the interaction of hydroxyl ions with cell membranes causes saponification, leading to cell disruption and death. This effectively destroys barriers to penetration, allowing the injurious agent to move rapidly into underlying tissues. The most significant injuries occur at a pH greater than 11. The ability of alkalis to easily penetrate the eye means that alkali injuries can easily cause the ocular surface, corneal stroma and other anterior segment structures.

Commonly encountered causes of alkali injury include ammonia (NH₃), lye (NaOH), potassium hydroxide (KOH), magnesium hydroxide (Mg(OH)₂), and lime (Ca(OH)₂) (3). Of these, the most serious injuries are caused by ammonia (found in fertilizers and cleaning agents) and lye (found in drain cleaners), while the most commonly reported injuries involve, a constituent of plaster. We perform a case series of six patients to determine common characteristics in presentation, management and outcome for alkali ocular burns.

METHOD: A retrospective review was undertaken of patients with alkali related ocular injuries seen at the Royal Brisbane Hospital, Australia between March 1978 and May 1999. Six patients were identified and their injuries classified as per the Roper-Hall system (see Table 1). An analysis of the cases was undertaken to determine common characteristics in management and prognosis.

RESULTS: Results are shown in Table 2. All patients suffered alkali burns with caustic soda and were treated initially with copious irrigation. Further treatment was individualised and comprised of antibiotics, cycloplegics, steroids and agents to promote corneal healing. Five of the six patients had vision return to pre-morbid levels (Roper Hall I – III). The one patient with severe burns (Roper Hall IV) was rendered legally blind.

CONCLUSION: Alkali burns can result in severe ocular injuries. Prognosis depends on severity of injury, timing to initiation of irrigation, and a high standard of ophthalmic care. Most essential is emergency eye irrigation and urgent ophthalmic review. Definitive management should be coordinated between specialized burns units and ophthalmologists. Prognosis varies according to the severity injury, specifically corneal epithelial damage and the extent of limbal ischaemia as per the Roper-Hall Classification.

While numerous medical and surgical interventions are available, their appropriateness must be assessed on a case-by-case basis. The great majority of alkali burns appear to be either workplace related or secondary to domestic accidents, with caustic soda being the most common causative agent. Prevention of these injuries with workplace health and safety promotion remains the ultimate aim.