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Atypical corneal manifestation of sterile inflammatory infiltrates after alkali ocular burn

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Abstract

Sterile corneal infiltrates result from the inflammatory response to specific antigens, mobilizing leukocytes (infiltrates) from the limbal vessels and tear films. They are often observed in contact lens wearers or are secondary to the immune response to bacterial exotoxins and endotoxins. Here, we present an atypical case of sterile infiltrates following alkali ocular burns and a differential diagnosis of infectious infiltrates.

Keywords: Ocular burn, alkali, chemical burns, eye

Introduction

Chemical burns are common causes of ocular health damage and should be considered an ophthalmologic emergency due to their high morbidity ^[1].

In addition to injuring the ocular surface, alkalis can penetrate the eye, damaging the corneal stroma and endothelium as well as other structures within the anterior segment, such as the iris, lens, and ciliary body ^[1, 2].

Sterile corneal infiltrates are commonly observed in contact lens users and rarely reported in cases of chemical ocular burns, which can be mistaken for infectious infiltrates ^[3].

The objective of this study was to report an atypical manifestation of sterile infiltrates following a case of chemical ocular burns.

Case Report

Male patient, 31 years old, Caucasian, occupation: mason. He reported having received lime in his right eye while working, two hours prior to presentation, experiencing pain, reduced visual acuity, tearing, photophobia, and redness in the affected eye, despite abundant rinsing with water. The patient denied any personal, familial, or ophthalmological history.

Examination findings

Visual acuity: Right eye, hand movement of 30 cm; left eye, 20/20. The intraocular pressure was 14 mmHg in both eyes. Right eye biomicroscopy revealed pale tarsal conjunctiva, conjunctival chemosis, 3+/4+ bulbar conjunctival hyperemia, presence of inflammatory membranes on the superior and inferior tarsal conjunctiva, cornea with severe edema 4+/4+, complete epithelial loss, limbal ischemia at approximately 270°, and a negative Seidel test (Figure 1). Fundoscopy: Media opacity preventing examination of the right eye. No alterations were observed in the left eye. Treatment: copious rinsing with physiological saline; oral medications: doxycycline 100 mg every 12 h, Vitamin C 2 g/day, and prednisone 60 mg/day; ocular treatment of the right eye: moxifloxacin 0.5% every 6 h, prednisolone acetate 1% every 6 h, tropicamide 1% every 8 h, and preservative-free lubricant every 1 hour.

After five days of follow-up, the patient showed improvement in corneal edema and opacity. However, numerous peripheral inflammatory infiltrates were observed (Figure 2). Treatment was continued according to the chemical burn protocol, resulting in the complete resolution of the infiltrates. Partial limbal failure and corneal conjunctivalization were initially observed in ischemic areas. The condition stabilized and the patient achieved a visual acuity of 20/40 after 40 days of progression (Figure 3).



Fig 1: Anterior biomicroscopy at the initial consultation following ocular burn

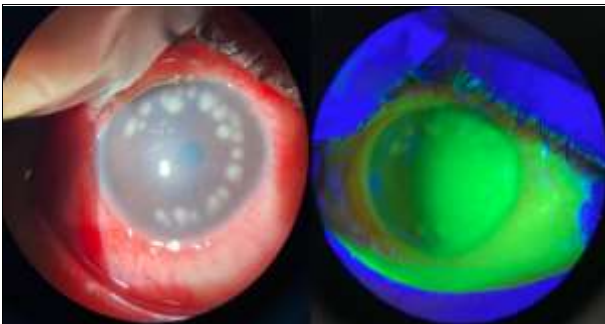


Fig 2: Anterior biomicroscopy on the 5th day after ocular burn, revealing peripheral perilimbal nummular inflammatory infiltrates.

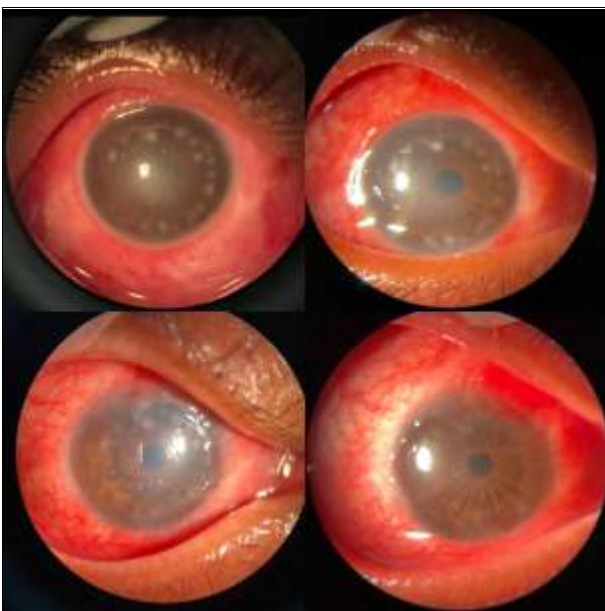


Fig 3: Evolution of peripheral nummular inflammatory infiltrates over 40 days following ocular burn.

Discussion

Chemical burns caused by acids or alkalis can result in extensive damage to the cornea, ocular surface, and the anterior segment of the eye, potentially leading to permanent visual impairment^[4].

Ahmed *et al.* demonstrated that chemical burns account for 2.2 to 13% of all ocular trauma emergencies, with a

higher incidence in children under 5 years of age in the context of household accidents involving household cleaning products and in adults within the context of both domestic and occupational accidents, particularly among young men with outdoor or industrial occupations. Chemical burns constitute 6-45% of all occupational eye injuries, ranking among the most frequent causes alongside foreign bodies^[5, 6].

Owing to their higher prevalence in everyday products, such as household cleaning agents and construction materials, accidental injuries caused by alkaline agents occur more frequently than those caused by acids^[4, 7, 8, 11, 12].

The severity of ocular injury depends on the duration of exposure of the eyeball to the aggressive agent, concentration of the agent, pH of the solution, and the rate of drug penetration^[1, 9].

Alkalies commonly associated with ocular burns include ammonia (NH₃), caustic soda (NaOH), lime [CaO or Ca(OH)₂], and potassium hydroxide (KOH).⁹ The hydroxide ion (OH⁻) saponifies fatty acids and leads to the hydrolysis of cell membranes, resulting in cellular rupture and death, facilitating the penetration of the caustic agent into tissues and causing deeper injuries. Cations react with the carboxylic groups (COOH⁻) of stromal collagen and glycosaminoglycans, and their hydration leads to loss of corneal stromal transparency^[1, 4].

When there is sufficient contact between alkaline compounds and the ocular surface, alkaline substances can be detected in the aqueous humor within 3-5 minutes. This leads to impairment of the ocular surface as well as the stroma, endothelium, and intraocular structures^[1, 7, 9].

Healthy tissues near the injury site trigger reactive inflammation and chemotaxis of polymorphonuclear leukocytes, leading to the release of lysosomal enzymes (collagenases) with collagenolytic activity. This process can result in severe corneal complications such as ulceration and corneal perforation^[9].

The described case presents a Grade III ocular burn according to the Roper Hall classification. This case exhibited atypical progression with the emergence of sterile inflammatory infiltrates even after intensive treatment for inflammation resulting from the burn^[10].

Sterile corneal infiltrates typically appear in the peripheral region of the cornea as small opaque spots ranging from 0.1 to 1.0 mm in size. They are usually located in the subepithelial or anterior stromal region, and can be singular or multiple. The epithelium may or may not be intact and patients often complain of ocular discomfort, photophobia, and tearing. In many cases, infiltrates have an immune origin, necessitating a differential diagnosis of bacterial keratitis^[3].

The patient in this study was administered broad-spectrum antibiotic eye drops initiated after the occurrence of ocular burns due to lime exposure. Owing to the nature of the lesions and the timing of their appearance, the hypothesis of sterile corneal infiltrates was proposed, which was confirmed by the favorable progression of the condition.

The present case contributes to the discussion of the progression of chemical ocular burns, emphasizing the differential diagnoses of corneal infiltrates, which can be either sterile or infectious. Clinical observation of these manifestations is of paramount importance for determining the most appropriate course of action.

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