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REVIEW

Chemical and thermal ocular burns: a review of causes, clinical features and management protocol

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Chemical and thermal ocular burns are among the most urgent ophthalmic emergencies, often resulting in permanent damage, and in some cases, blindness. These burns are the result of exposure to chemicals or radiant energy (thermal or ultraviolet). The most serious injuries are due to chemical burns by strong acid or bases. The purpose of managing these burns is to eliminate or limit the causative agent from penetrating the ocular structures by irrigation; and, promoting ocular surface healing through medical and surgical intervention. This review presents a current update on the causes of chemical and thermal ocular burns, their clinical features and the importance of appropriate and prompt treatment.

Keywords: acid burns, alkali burns, chemical burns, ocular burns, ocular irrigation

Introduction

Chemical and thermal ocular burns are among the most frequently reported causes of eye injuries, estimated to account for approximately 8-18% of ocular trauma.[1–3] These burns occur through accidents at work, home or during leisure activities;[4] tend to be bilateral,[5] and are seen more frequently in young males than females.[6] For example, Saini and Sharma[7] reported that young people working in laboratories and factories constituted two thirds of patients who experienced chemical injuries, and emphasised that the use of eyewear was mandatory when performing their duties. The injuries caused by chemical burns to the eye can range from mild unilateral conjunctival or corneal epithelial damage to sight-threatening damage to the conjunctiva and cornea.[8] The resulting vision impairment and blindness has important health, socio-economic and quality-of-life implications, which can lead to lost economic gain, and missed employment and educational opportunities, resulting in reduced quality of life generally.[9] The symptoms of chemical ocular burns include photophobia, tearing and pain, while conjunctival hyperaemia, subconjunctival haemorrhage and chemosis are some of the presenting ocular signs of the condition.[4]

Superficial punctuate keratitis is a sign of a mild ocular burn, while corneal opacification and oedema decrease the visibility of the iris and lens in severe burns. A mild anterior chamber reaction can occur.[4] Typical signs of a severe burn are more than 50% loss of the epithelium and perilimbal ischaemia.[4] These signs are usually coupled with "an inflammatory reaction of the anterior chamber, ocular hypertonia and corneal anaesthesia",[4] which results in the eye looking white, and indicates that there is no blood supply to transport the white blood cells needed to fight a possible infection. Timeous intervention is often key to preventing significant functional and anatomical damage to the ocular structures. The common causes, clinical features and management protocols of chemical thermal and ocular burns are discussed in this literature review.

Pathophysiology

While the course of an ocular burn depends upon the nature of the offending agent, chemical burns share common elements.[4] The initial phase of incineration is followed by a rush of inflammatory cells to produce various detergent enzymes (detersion), such as the matrix metalloproteinases (collagenases, gelatinases and stromelysin), which aggravate the destruction of the ocular structures.[10] This is followed by a scarring phase, which results from the regrowth of healthy tissue surrounding the burn.[11] The ischaemic lesions form as a result of the destruction of the vascular network, as well as to lesions of the corneal and conjunctival cells.[11] Corneal and the conjunctival scarring can occur because the surviving cells mutate into fibroblasts, and also as a result of division of the stem cells.[4]

Chemicals can be classified as either acidic or alkaline agents.[12] Many of these are used in homes, industries and agriculture, causing burns when they come into contact with the eye, resulting in a significant threat to vision, especially those that are alkaline.[4] The extent of the injury is influenced by various factors, such as the nature, quantity and concentration of the solution, the contact duration, solution penetrability and pH.[4] While most burns occur from direct contact with the outer eye surfaces, chemicals can also reach the ocular tissue through systemic absorption via the skin, lungs or digestive tract.[4] The intact cornea can resist a wide range of pH without injury, but a pH < 4 or > 10 results in an increase in permeability, which can cause severe ocular complications.[4]

Acid burns

Automobile battery explosions are a common cause of acid burns. The explosive nature of the injury can lead to significant damage of the globe, either by contusion or perforation.[4,11] Hydrofluoric, hydrochloric, chromic, acetic and sulphuric acid orvitriol are highly concentrated acids, with a pH of between 1.0 and 3.5, causing the worst accidents.[4,11] They cause rapid damage to the superficial tissue structures, but tend to be neutralised in a short period because the protons bind with the tissue protein, and precipitate and denature it.[4,11] Coagulation on the eye's surface establishes a barrier to further penetration, resulting in most acid burns being confined to the superficial tissue.[4] However, ocular lesions due to strong acids (a pH below 2.5) are deep and necrotising, affecting the conjunctival and limbal vessels.[4]

Alkali burns

The main bases of alkali include ammonia, sodium hypochlorite, and sodium, potassium and calcium hydroxide, which have a pH of between 12 and 14.[4,11] Alkali burns appear to be innocuous at first, but rapidly progress, and are more threatening to the deeper tissue.[11] They have poorer prognosis because the anion (hydroxyl) causes saponification of the fat and lipids, leading to a softening of the tissue, which enables increased penetration of the cation chemicals.[4,11] Further alterations to the ocular structures, such as the iris, iridocorneal angle, ciliary body and crystalline lens, can occur because of rapid penetration of the alkali.[4] Complete and irreversible ocular lesions occur at a pH above 11.5.[13]

The classification of chemical ocular burns

Tables 1 and 2 detail the classification of the severity of chemical burns by Hughes,[14] updated by Roper-Hall,[15] which are used clinically to guide treatment decisions and protocols.

The changes of limbus for grade IV burns are not accurately explained by the Roper-Hall classification. In 2001, Dua et al[12] proposed a new classification based on the importance of the deficit of limbal stem cells. The impact on vision can be categorised as follows:

- *Grade I-III:* Vision should recover.
- *Grade IV:* Usually vision is impaired to some degree, thus prognosis is guarded.
- *Grade V-VI:* The damage leads to severe visual impairment and vision loss.

Managing chemical ocular burns

The management of chemical burns normally takes one or any combination of three forms, namely ocular lavage, medical and surgery.

Ocular lavage

Irrigation with water or saline remains the most effective established intervention in terms of a positive prognosis and outcome with respect to ocular chemical burns.[8,16,17] Although these solutions are most commonly used because they are readily available, newer and more effective neutralising agents can be used.[8,17–19] These agents include a balanced salt solution, Ringer's lactate, buffers and Diphoterine®.[8] Urine dipsticks and universal indicator paper can be used to measure the conjunctival pH of patients with chemical burns. This process is important when identifying patients requiring irrigation, and should be continued until the pH in the conjunctival sac is neutral.[4] The affected eye must be rinsed with at least 1.0–1.5 litres of water or normal saline for no less than 15 minutes.[4] However, water and normal saline have low osmolarity, which can result in their increased uptake into the corneal stroma.[8]

Ringer's lactate and a balanced salt solution are more effective than normal saline because they have the same osmolarity as aqueous humour.[8] A balanced salt solution is routinely utilised in ocular surgery as it prevents corneal swelling, thus preserving the integrity of the corneal endothelium.[20] However, it is expensive and not routinely used outside of theatre.[20] While phosphate buffers are used in some emergency irrigating solutions, their use has been associated with stromal calcification, and is therefore not recommended as a first choice.[17] Diphoterine® is a hypertonic solution and has been shown to have better results than a normal saline solution as it creates a movement of water from the hypotonic anterior chamber to the surface of the hypertonic cornea.[19,21]

Irrigating the eye should be continued until the possibility of chemical action is eliminated. The pH of the tears can be periodically tested with litmus paper to determine if the condition still exists, and the visual acuities checked. Pain and lid oedema may make conducting an objective examination difficult, which can be overcome by instilling a local anaesthetic, such as proparacaine. The local anaesthetic may also serve as a prognostic indicator as the drops are irritating on instillation in the normal eye, while the absence of irritation indicates a problem in eyes with severe burns and damaged corneal nerves.

Medical treatment

Local corticoids reduce inflammation by decreasing invasion of the corneal stroma by polynuclear neutrophils.[22] Corticoids stabilise cell and lysosomol membranes against polynuclear neutrophils and antagonise the action of collagenase enzymes.[22] Although they limit conjunctival mucous cell destruction,[22] they also reduce keratocyte migration, inhibit collagen synthesis and

Table 1: Hughes' classification,[14] modified by Roper-Hall[15]

Grade	Corneal alteration	Limbal ischaemia	Prognosis
I	Epithelial damage. No corneal opacity	No ischaemia	Excellent
II	Oedematous cornea. The iris details are visible	Ischaemia less than half at limbus	Good
III	Total epithelial loss. Stromal oedema. The iris details are obscured	Ischaemia affects one third to half of all patients at limbus	Reserved
IV	Opaque. The iris or pupil is invisible	Ischaemia affects more than half of all patients at limbus	Poor

Table 2: Dua's classification[12]

Grade	Analogue scale	Clinical findings	Conjunctival alteration (%)	Prognosis
I	0.0/0.0	0 clock hours of limbal involvement	0	Very good
II	0.1-3.0/1.0-29.9	≤ 3 clock hours of limbal involvement	≤ 30	Good
III	3.1-6.0/31.0-50.0	> 3–6 clock hours of limbal involvement	> 30–50	Good
IV	6.1-9.0/51.0-75.0	> 6–9 clock hours of limbal involvement	> 50–75	Good to reserved
V	9.1-11.9/75.1-99.9	> 9 < 12 clock hours of limbal involvement	> 75 < 100	Reserved to poor
VI	12.0/100.0	Total limbus (12 clock hours) involvement	Total conjunctiva (100%) involved	Very poor

delay cicatrisation.[23] The use of anti-inflammatory nonsteroidal treatment should be avoided as it lengthens the epithelial scarring process and modifies corneal sensitivity.[11] The parenteral administration of tetracycline reduces the incidence of corneal ulceration and facilitates cicatrisation.[24] Cycloplegics are given to minimise lens adhesion, as well as inflammation of the iris and ciliary body. The regular use of preservative-free artificial tears offers supportive treatment, and the local or parenteral administration of ascorbic acid has been reported to prevent corneal ulceration and retinal thinning.[25] Antibiotic eyedrops and parenteral tetracycline are given to minimise the risk of infections; while analgesics, taken orally or parenterally, are prescribed because corneal nerve lesions can be associated with intense pain.[11] Pressure patching is standard until re-epithelisation occurs, after which the person is referred for surgery.

Surgical intervention

Surgical treatment should be considered in severe burn cases, when the destroyed limbal stem cells need to be restored. Procedures such as excision, tenoplasty, preventing the formation of symblepharons, limbus transplantation, amniotic membrane transplantation, keratoplasties, cultivated epithelial limbus cell transportation, and conjunctival transplantation, using nasal or buccal mucous membrane samples, are used depending on the severity of the burn and the desired outcome.[4,11]

Thermal and radiation burns

Thermal burns

Damage due to thermal burns occurs at the time of injury. Most commonly, the causes are boiling liquid, molten metal, flames, gasoline explosions, steam and hot tar.[4] The extent of damage and impact on vision depend on the degree of the heat agent, area and duration of contact, as well as conductance of the tissue.[4] If the burn is caused by a flame, the eyelashes and lids are mainly affected because of the speed of the protective blink response.[4] When the eye is not protected, severe thermal ocular lesions are mainly associated with grade III cutaneous burns.[26] The mainstay treatment options for superficial lesions caused by thermal burns include a combination of local antibiotherapy, instillation of artificial tears, application of an occlusive dressing (until re-epitheliasation), and sometimes cycloplegia.[4] Common complications include retractile palpebral scars owing to conditions such as trichiasis, entropion or ectropion.[4]

Radiation burns

Sources of ultraviolet (UV) radiation are varied, and include those which are highly directional from the sun, diffuse and directional from the sun, and diffuse and specularly reflected from various surfaces on earth, e.g. snow, sand and water.[27] The amount of UV radiation varies with the time of day, angle of the sun, cloud cover and changes in the reflecting surfaces. Excessive exposure to UV radiation is associated with the development of pterygia, ocular neoplasms, photokeratitis, age-related cataracts and irreversible damage to the retina.[27] Preventive measures include wearing protective eyewear with UV-blocking tints.[27] Infrared (IR) radiation causes superficial punctate keratitis to the cornea, which has been reported to secondarily induce an increase in intraocular pressure.[28] When sufficient IR is absorbed by the iris, this can lead to pupillary miosis, aqueous flare (because of a breakdown in the blood aqueous barrier), hyperaemia and post synechiae.[28] IR on the lens produces anterior subcapsular opacities which first appear as discrete "whitish dots", and can lead to a network-like whitish opacity with sufficient exposure. This does not migrate towards the equator or post capsule, but fades and disappears within six weeks.[28] Laser IR can also cause chorioretinitis and retinal pigment epithelium thermal injury. To avoid the effects of IR on the ocular structures, protective wear, such as helmets and shields with filters, should be worn, particularly by glass blowers and steel workers.[28] Neutral grey to yellow tints, which provide protection against most optical radiation, are recommended.[28] Metallic oxide incorporated into glass absorbs 95% of UV and IR. Reflective filters vacuum-coated onto the front surface of the lens reflect unwanted IR.[28] Burns by UV and IR radiation, which cause cataracts or chorioretinitis, may lead to visual acuity deterioration and vision loss.

Conclusion

Akali injuries are more common than acid injuries as they are included more frequently in household cleaning agents, as well as industrial and building materials. Copious irrigation with water, saline solution or an agent, such as phosphate buffer, lactated Ringer's solution, a balanced salt solution, and Diphoterine® solution (to achieve therapeutic goals), is the most important and immediate intervention for chemical ocular burns. Medical and surgical management depends on the clinical findings. Cycloplegics are given to minimise lens adhesion, inflammation of the iris and ciliary body, while topical antibiotics are provided to minimise the risk of infection. Ocular burns in industries can be reduced through occupational health laws which educate workers and promote the use of safety eyewear.

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