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EMERGENCIES IN OPHTHALMOLOGY

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5.1.5. Ocular Burns

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Key strengths

- 85% of ocular burns are caused by chemicals.
- Chemical ocular burns account for 10% of eye injuries.
- Most ocular burns affect active, young men and are sustained as a result of industrial, occupational or domestic accidents.
- Assaults account for 2.5% of chemical burns.
- Bases quickly and deeply penetrate ocular medium combining them.
- Acids penetrate more slowly than bases and denaturate the tissues.
- An ocular burn is always an emergency.
- Evaluation requires an ophthalmologist.
- Ocular rinsing shall be done as soon as possible, at the site of the accident, without waiting; repeated multiple times to remove all residues and foreign bodies.
- Routine pharmacological treatment is based on a topical anti-inflammatory corticosteroid, cyclopegic eye drops, preservative-free wound-healing ointment and eye drops containing a broad-spectrum antibiotic; oral tetracycline and Vitamin C; and painkillers administered orally or parenterally.

Introduction

Despite huge therapeutic progress in the last decade, burns remain one of the most challenging ocular emergencies that ophthalmologists encounter. Whether chemical, thermal or due to radiation, they account for 3-4% of all domestic injuries and 7-18% of eye injuries [1, 2]. Ocular involvement complicates some 15-20% of facial burns. Often affecting both eyes, most burns affect young males [3]. The main causes are industrial, occupational and domestic accidents together with assaults. With over 25,000 chemicals able to cause burns, chemical injuries to the eye are by far the most common and the most critical. Seriousness depends on the causal substance which is usually corrosive or an oxidising or reducing agent. The consequences can be terrible because even despite well-conducted treatment, loss of function is possible or even anatomical loss of the eyeball. Above and beyond any sight impairment, ocular burns can have huge psychological repercussions and compromise various daily activities as well as victims' capacity for work [3]. Burns due to heat or radiation are usually associated with more superficial injuries.

Clinical presentation

An ocular burn is an emergency. The initial physical examination should be quickly followed by the first therapeutic measures, in particular the ocular rinsing [2, 4]. It is worth making a distinction between findings of the initial physical examination, measurement and classification of the degree of seriousness, and description of problems arising during progression of the lesions.

■ FUNCTIONAL SIGNS

At presentation, symptoms vary but the following are common:

- Inflammatory red eye;
- Painful eye;
- permanently compromised sight;
- eye-watering, blepharospasm.

■ CONTEXT AND PREDISPOSITION

FREQUENCY AND CIRCUMSTANCES OF OCCURRENCE

Skin burns, on the decrease in industrialised countries in recent years, are almost all caused by flames or hot liquids. In the United States, the incidence of skin burns is 220 per 100,000 people per year [5]. The proportion of chemical skin burns ranges from 1.4% to 8.5%. In France, the incidence of skin burns is poorly characterised but it would be approximately 500,000 per year. Most occur as a result of domestic accidents or in the course of leisure activities. The head is involved in one-third of cases [6]. There are no data on isolated ocular involvement but the epidemiological characteristics of ocular burns are radically different because chemical burns predominate (85%) with thermal burns far rarer: it might be worth replacing the term "chemical ocular burn" with "chemical ocular injury". Chemical ocular burns are also responsible for the most severe lesions with the heaviest psychological and social repercussions (and sometimes legal repercussions). Most victims are young, working men. Chemical ocular burns account for about 10% of eye injuries. About 10% of bodily chemical burns also involve the eyes. In 2006 in the United States, of 2.4 million cases of accidental exposure to a chemical, 5.4% were accompanied by eye involvement [7]. In 1999-2000, chemical ocular burns accounted for 6.4% of all eye emergencies at a hospital in the Paris region with an ophthalmologic emergency unit [8]. More recently, in a study conducted for this book at three Parisian emergency units, they accounted for 1.08% of the emergency

attendances listed. Most ocular burns occur as a result of an industrial or domestic accident. Frequency and proportion depend on the local degree of industrialisation. In Australia, the most common cause is an accident at work (71 %) followed by domestic accidents (23 %) and assault (2.5 %) [10]. The percentage of domestic accidents associated with home repairs and gardening has been consistently rising, reaching over a third in some studies. The percentage due to assault varies between 2.5% and more than half, depending on country, social mores, socio-economic conditions and the degree of industrialisation [10]. In the United Kingdom, one-third of severe chemical ocular burns are due to physical assault. In London, the incidence is on the rise and many such assaults are perpetrated by teenage gangs [11]. In Martinique, Jamaica and Nigeria, eye injuries are seen in more than a third of assaults [12]. These burns are mainly confined to the face. Eyes and eyelids are affected in 19% of cases. In the Caribbean and Hong Kong, the aggressor's intention is to disfigure the victim and render him or her blind. Most such assaults are planned and arise in the context of an argument between lovers. In most cases, the substance that the assailant uses is ammonia which has dramatic repercussions. Most commonly, the assailant is female and the victim male [12, 13]. Tear gas burns can exceed a quarter of all ocular burns. Chemical peeling with trichloroacetic acid or some other chemical can also lead to ocular burns [14]. In children, chemical ocular injuries accounts for some 10% of eye injuries. In 2012, ocular burns accounted for 6.41% of eye injuries in children in the Greater Paris region [15].

CAUSAL AGENTS

Causal agents can be classified between three different categories, namely physical, chemical and biological agents. These are listed in 5-1-5. For physical agents, high temperature, a large volume and forceful impact will exacerbate the severity of the burn. As a rule, the volume of hot liquid that reaches the surface of the eyeball is small (<0.5 ml) and this is quantity reduced by blinking. However, solids and powders are not flushed out by blinking and remain in contact with the eye. Ocular burns account for between 13% and 62% of eye injuries associated with fireworks. In Alsace, because of the illegal importation and manufacture of fireworks that do not work properly, eye injuries from these materials are very common and account for more than a third of such burns in the area [16].

■ PHYSICAL EXAMINATION

ACUTE PHASE

An interview with the victim establishes the time and circumstances of occurrence of the burn, the nature of the product responsible and any procedures that have already been carried out. Coordination with a Poison Centre is sometimes indicated because they have information about the toxicity of all current industrial, natural and medicinal products. Functional symptoms (reduced visual acuity, photophobia, eye-watering, etc.) can be highly informative as can be the degree of pain. Pain and spasm of the eyelids can be managed by instilling anaesthetic eye drops. The eyelids are sometimes red, swollen, abraded-looking with burnt eyelashes and eyebrows. The eye is usually red due to diffuse hyperhaemia in the conjunctiva with point

5-1-5 box

Main agents involved in ocular burns

Physical agents
- Thermal: flames, hot liquids, fireworks
- Cold: very low outside temperature, cryotherapy
- Electrical: electrocution, cauterisation
- Microwave ovens: superheated water, eggs

Chemical agents
- Household products: detergents, nail varnish, solvents
- Industrial products: solvents and paints, bleach, petroleum derivatives, caustic soda, potash, ammonia
- Agricultural products: fertilisers
- Medicinal products: EMLA® Cream, sodium hypochlorite (dentistry)
- Tear gas and Chemical Mace
- Other: Lime Powder (ingestible calcium hydroxide), air bags

Biological agents
- Animals: centipedes, snake venom, vesicant insects (blister-inducing) insects
- Plants: Euphorbia (spurge, manchineel, pencil tree)

Oxidising agents
- Medicinal products: hydrogen peroxide, contact lens fluids

haemorrhages around the edge of the iris, sub-conjunctival haemorrhages, a perikeratotic circle or haemorrhagic chemosis. Mild burns are limited to superficial punctate keratitis (SPK) in the eyelid opening space or to more extensive ulceration of the corneal epithelium (Figs. 5-1-32 and 5-1-33). In addition to epithelial destruction, severe burns of the cornea lead to Descemet membrane folds and oedema which, in the worst cases, can look like porcelain and masks the iris and the lens (Figs. 5-1-34). More serious involvement is also characterised by ulceration and ischaemic patches or necrosis of the limbal region or bulbar conjunctiva. Ischaemic patches look white and swollen. They are due to interruption of the blood supply to the vessels of the episclera and conjunctiva. They are often concentrated in lower parts where the chemical has collected. An Amsler test (pricking to draw blood) can help with assessing the extent of conjunctival ischaemia. Serious forms are accompanied by inflammation in the anterior chamber, a tendency to synechia formation, severe endothelial injuries,

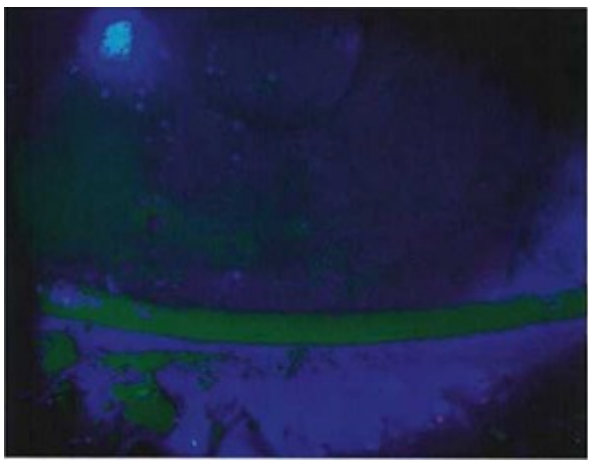

Fig. 5-1-32 ***Burn due to a weak acid***
Superficial punctate keratitis in the eyelid opening space combined with more extensive ulceration of the corneal epithelium in the lower part where the chemical product tends to collect.

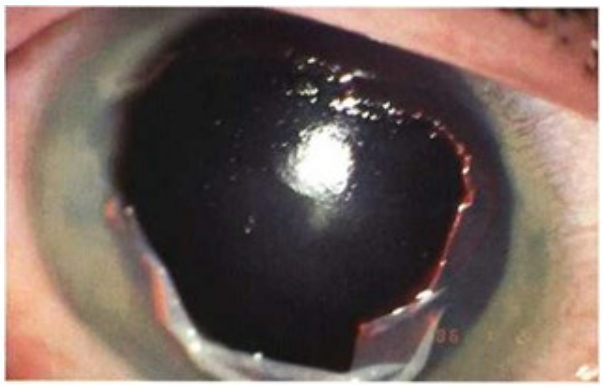

Fig. 5-1-33 ***Burn due to a strong acid.***
Complete destruction of the corneal epithelium which has folded in on itself at the bottom. The corneal stroma is transparent and the limbal region is not ischaemic.

hypertonia and corneal anaesthesia. Exceptionally, perforation may be observed at the outset [9]. It is also important to investigate injuries to the eyelids, especially the free edges and lacrimal points. In children, the initial examination would usually be carried out under general anaesthesia. All clinical observations (ulceration of the cornea and conjunctiva, ischaemia, etc.) are recorded on a daily basis (on a diagram or a photograph).

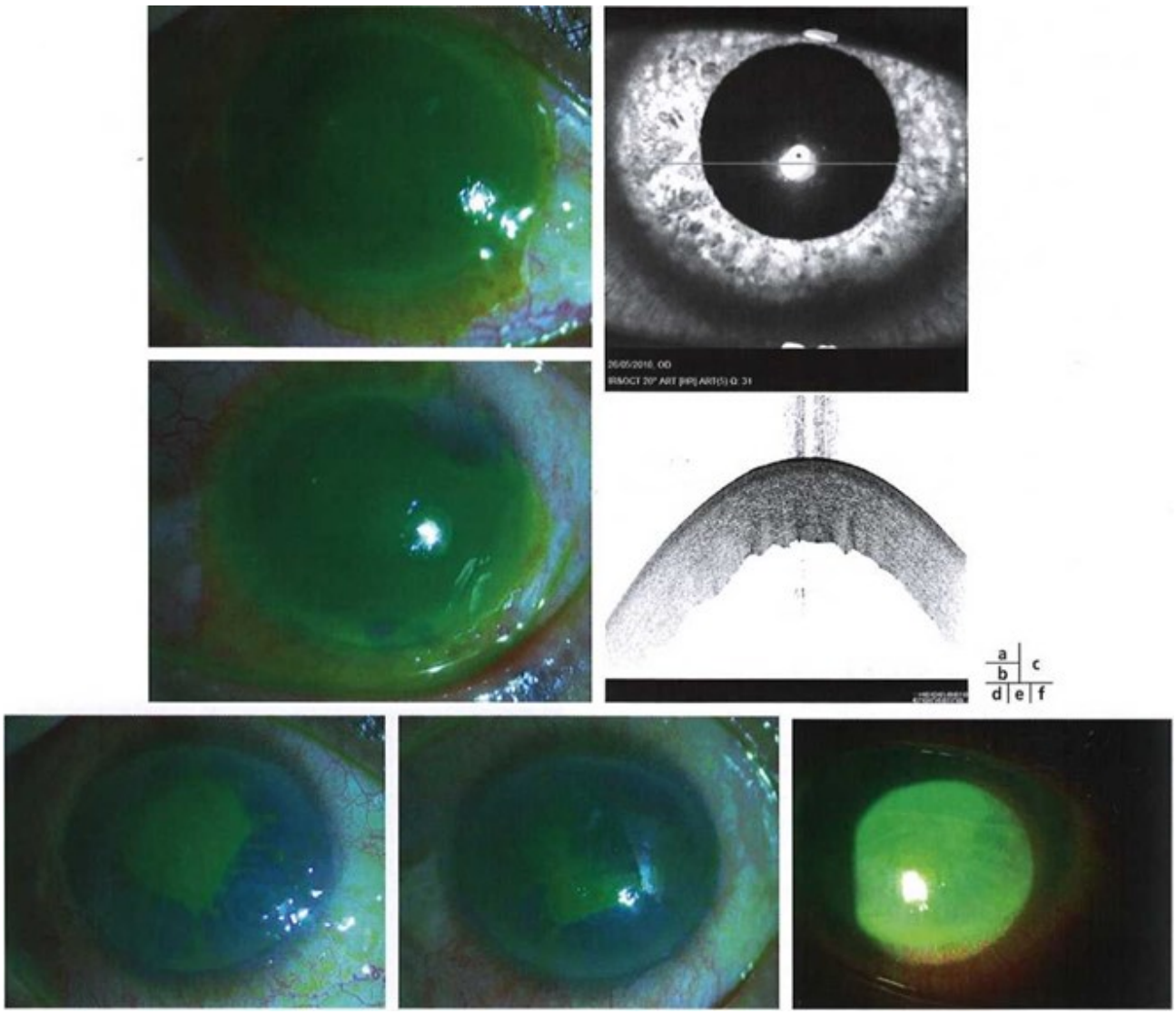

Fig. 5-1-34 ***Burn due to a strong acid (hydrochloric acid for pickling metals).***
a. Right eye, d2 Total destruction of the corneal epithelium. Corneal oedema : neither iris nor pupil visible. Punctate keratitis. Limbal involvement between 9 and 12 clock hours. Stage 4 in the Roper-Hall classification system, Grade V in the Dua classification system. **b.** Left eye, d2. Partial destruction of the corneal epithelium. Corneal oedema : neither iris nor pupil visible. Punctate keratitis. Limbal involvement between 6 and 9 clock hours. Stage 4 in the Roper-Hall classification system, Grade IV in the Dua classification system. **c.** Right eye, d2. OCT of the anterior segment. No epithelium. Hyper-reflective anterior stroma. Corneal oedema. Thickened cornea (720 pm) accompanied by irregularity of the endothelial surface. Descemet membrane folds seen in OCT under a wave-shaped, domed-looking endothelium in the anterior chamber. **d.** Right eye, d10. Centripetal healing of the corneal surface. Persistent ulceration in the middle of the cornea covering half of its surface area. Corneal oedema and punctate keratitis. **e.** Left eye, d10. Centripetal scarring of the corneal surface. Persistent healing failure in a triangular shape. Corneal oedema and punctate keratitis. **f.** Right eye, d10. Persistent folding of Descemet's membrane over apparent punctate keratitis.

CLASSIFICATION OF LESIONS

The purpose of classification is to establish prognosis and guide treatment strategy on the basis of the clinical findings of the initial examination. The most widely used classification system is that of Hughes as modified by Roper-Hall (Table 5-1-8) [17, 18]. This has four stages of severity based on the degree of stromal opacity and the extent of any limbal ischaemia. For Stage 1 and 2 burns, prognosis is good but for Stages 3 and 4 it is poor. However, the Roper-Hall system in not precise enough for Stage 4 with respect to limbal involvement [10]. In practice, a Roper-Hall Stage 4 burn can have a good outcome if the limbal involvement is less than 75%; in contrast, prognosis is very poor if the whole limbus is affected [19]. The heterogeneity of lesions within Stage 4 may also explain the inconsistency of outcome attained when the same surgical technique—limbal stem cell (LSC) transplantation—is used. Moreover, conjunctival involvement is omitted from the Roper-Hall classification system even though it is an important parameter. If the limbus is entirely destroyed, healthy conjunctival tissue can grow back over the cornea but if no healthy conjunctival tissue is left, there is a major risk of corneal perforation. The Roper-Hall classification system could be advantageously replaced with that of Wagoner (Table 5-1-9) or Dua *et al.* (Table 5-1-10) based on limbal and conjunctival involvement as detected by the uptake of fluorescein [9, 19]. Wagoner classification depends on the extent of limbal ischaemia proportional to the loss of LSCs. In Dua *et al.* classification, limbal involvement (not just ischaemia) is expressed in terms of the number of clock hours and involvement of the bulbar conjunctiva in terms of percentage of surface area. Using an analogue scale in which the first number gives the number of clock hours and the second the percentage of conjunctival surface area affected, gives an initial classification and subsequently makes it possible to follow changes by comparing scores at successive examinations. Prognosis is good for Grades I, II and III. For Grade IV which includes limbal involvement of between 6 and 9 clock hours and 50-75% corneal injuries, the prognosis may be either good or guarded. For Grade V, prognosis is poor and for Grade VI, very poor. Grades IV, V and VI correspond to Stage 4 of the Roper-Hall system which predicts a poor prognosis. In contrast to the Roper-Hall classification system, that of Dua *et al.* does not take the appearance of the cornea into account. Changes in this tissue's transparency are highly variable and often delayed. Nor does it take stock of the condition of the tarsal conjunctiva because examination of this is made very difficult if the eyelid is swollen, tight, indurated or thin and impossible to evert. The system of Pfister *et al.*, developed from that of Hughes, has six grades (normal, mild, moderate, moderate-to-severe, severe and very severe). This focuses on corneal and conjunctival involvement. It is rarely used but has the advantage of reporting the ultimate visual acuity (Table 5-1-11) [20].

Table 5-1-8 – Hughes classification system as modified by Roper-Hall [17, 18].

Stage	Prognosis	Corneal involvement	Limbal ischaemia (% limbal circumference)
1	Excellent	Epithelial involvement, no corneal opacity	0 %
2	Good	Corneal opacity, iris details visible	< 33 %
3	Guarded	Total epithelial loss, stromal oedema obscuring iris details	33-50 %
4	Poor	Corneal opacity, neither iris nor pupil visible.	> 50 %

Table 5-1-9 – Wagoner classification system [9].

Grade	Extent of limbal ischaemia	Progress and prognosis
1	None or moderate	Rapid healing, no sequelae
2	<Half of the limbal circumference	Healing delayed, late superficial neovascularisation
3	>Half of the limbal circumference	Healing very slow, stromal neovascularisation, conjunctival pannus
4	Total, neighbouring conjunctival necrosis	Aseptic corneal necrosis, severe damage to the anterior segment

Table 5-1-10 – Dua classification system [19].

Grade	Prognosis	Limbal involvement in clock hours	Conjunctival involvement	Analogue scale
I	Very good	0	0 %	0/0 %
II	Good	3	< 30 %	0.1-3/1-29.9%
III	Good	3-6	30-50 %	3.1-6/31-50 %
IV	Good-to-guarded	6-9	50-75 %	6.1-9/51-75 %
V	Guarded-to-poor	9-12	75-100 %	9.1-11.9/75, 1-99.9%
IV	Very poor	12	100 %	12/100 %

Tableau 5-1-11 – Pfister *et al.* classification system [20].

Grade	Corneal involvement	Conjunctival involvement	Prognosis	Visual acuity
Normal	0	0	Very good	Normal
Mild	Erosion of the epithelium, mild oedema of the anterior stroma	0	No or minor corneal scarring	Loss of 1 or 2 lines
Moderate	Moderate opacity	Insignificant	Slow epithelial healing, moderate scarring	Loss of 2-7 lines
Moderate-to-severe	Opacity obscuring details of the iris	Conjunctival necrosis < 1/3 limbal conjunctiva	Corneal neovascularisation, scarring	< 1/10
Severe	Dense corneal opacity Pupil contour barely visible	Conjunctival necrosis > 1/3 and < 2/3 limbal conjunctiva	Severe neovascularisation , risk of ulceration and perforation	Limited to counting of the fingers
Very severe	White cornea Pupil not seen	Conjunctival necrosis > 2/3 limbal conjunctiva	ulceration and perforation common, risk of phthisis	Light perception

■ TIME-TO TREATMENT

Extreme ophthalmologic emergency, to be treated as soon as possible, absolute ophthalmologic priority:

- victim classification by A&E nurse (*classification infirmière des malades aux urgences,* CIMU) = score CIMU 2;
- Category 1 care triage

Interesting paraclinical signs for the emergency care

No paraclinical tests are required before treatment is started in priority.

Repeated measurement of local pH with pH stripes permits to adapt the methods of ocular rinsing.

The purpose of other paraclinical examinations is to:

- eliminate any associated lesion in a patient with multiple injuries, not specific to the burn itself;
- investigate burn damage to eye tissues although this is considered in the context of later care.

Aetiologic diagnosis

■ CHEMICAL BURNS

The chemicals most commonly involved are acids and bases. Acid burns account for 1.6% of eye injuries, and alkali burns 0.6%. Bases include the following: ammonia (NH_3) which is used in cleaning products, refrigerants and fertilisers, bleach (sodium hypochlorite), caustic soda (NaOH) used as a household cleaner, potash (KOH) used in fertilisers and lime ($Ca(OH)_2$) used in cement. Particles of caustic soda and lime stick very strongly to the conjunctiva and constitute a reservoir of the toxic product [9]. Magnesium hydroxide ($Mg(0H)_2$) found in fireworks causes severe injuries, both thermal and chemical. Among the acids, sulphuric acid or vitriol (H_2SO_4) is responsible for the worst accidents. It is used in the textile industry and is also found in car battery fluid. Hydrofluoric acid (HF), used to dissolve rust, is a strong oxidising agent and is highly toxic. Hydrofluoric acid quickly crosses cell membranes and, like basic substances, induces necrosis by dissolving membrane phospholipids there. Hydrofluoric acid is used in the glass industry, in particular for engraving. It is also used for cleaning (windows, printed circuits, car wheels). Chromic acid (Cr_2O_3) is used in metallurgy. Hydrochloric acid (HCl) is used in the production of organic compounds (vinyl chloride), for metal pickling and for household cleaning. Concentrated acetic acid (CH_3COOH) can cause serious injuries. Other substances used in the chemical industry can be irritant or corrosive, including compounds containing sulphur, chlorine (swimming pool disinfectant), solvents, detergents and pesticides. The most common tear gases and aerosols are based on orthochlorobenzylidene-malononitrile and chlorocetophenone. These are reserved for the law enforcement units. Pepper spray is intended for private citizens [21]. Eye-watering is almost immediate after exposure but only lasts a few minutes. Various irreversible lesions have been reported after their use: conjunctival and corneal necrosis, corneal opacity, etc. Burns due to vesicant gases (mustard gas, lewisite) are seen in armed conflict, terrorist acts and in people handling old munitions. The resultant ocular burn can be very severe and sometimes causes blindness. These are lethal compounds.

Biological agents derived from animals are far rarer than agents derived from plants. In particular, members of the spurge family (Euphorbiaceae) are often involved. Most species induce burns through contact with the latex in their branches and leaves. Euphorbiaceae are very common in hot climates and they are used as ornamental plants (cactiform species) in colder places. In the Caribbean (Martinique, Guadeloupe, etc.) and the southern United States, manchineel—considered the most dangerous tree in the world—is very common. Its latex can cause dramatic burns, possibly progressing to ocular perforation (Fig. 5-1-35) [22].

■ THERMAL BURNS DUE TO HEAT

Eye involvement in thermal burns is rare, between 1% and 5%. Cases usually involve burning with a flame or hot liquid in the home. Seriousness depends on temperature and duration of exposure. Because of rapid blinking and Bell's phenomenon, the eyeball is protected and burning is usually limited to eyelashes, eyebrows and eyelids (Fig. 5-1-36). However, ulceration of the cornea and conjunctiva can occur in the slit between the eyelids. Ulceration can be accompanied by stromal opacity or signs of LSC insufficiency. In the case of contact burns, solids that retain heat (cigarette ash, gunpowder from fireworks, etc.) and materials with a high melting point (iron: 1200°C; glass: 1500°C) can inflict deep injuries and even cause loss of the eyeball. The most serious eye injuries are seen in patients who have suffered third-degree skin burns or who have had an accident with a firework [16]. To treat superficial lesions, topical antibiotics are administered with instillation of artificial tears and sometimes cycloplegia. Severe lesions are treated in the same way as chemical burns. Scarring that prevents retraction of the eyelids is often complicated by trichiasis, entropion or ectropion that all expose the surface of the eyeball.

■ THERMAL BURNS DUE TO COLD

Cold burns occur as a result of exposure to very low temperature, the effect of which may be exacerbated by strong wind: high-altitude accidents, sporting activities in a cold climate (skiing, running, etc.), parachuting. The injuries affect the part of the eye behind the slit between the eyelids. Lesions may look like simple epithelial abrasion, an ulcer, oedema or frank gelling of the cornea.

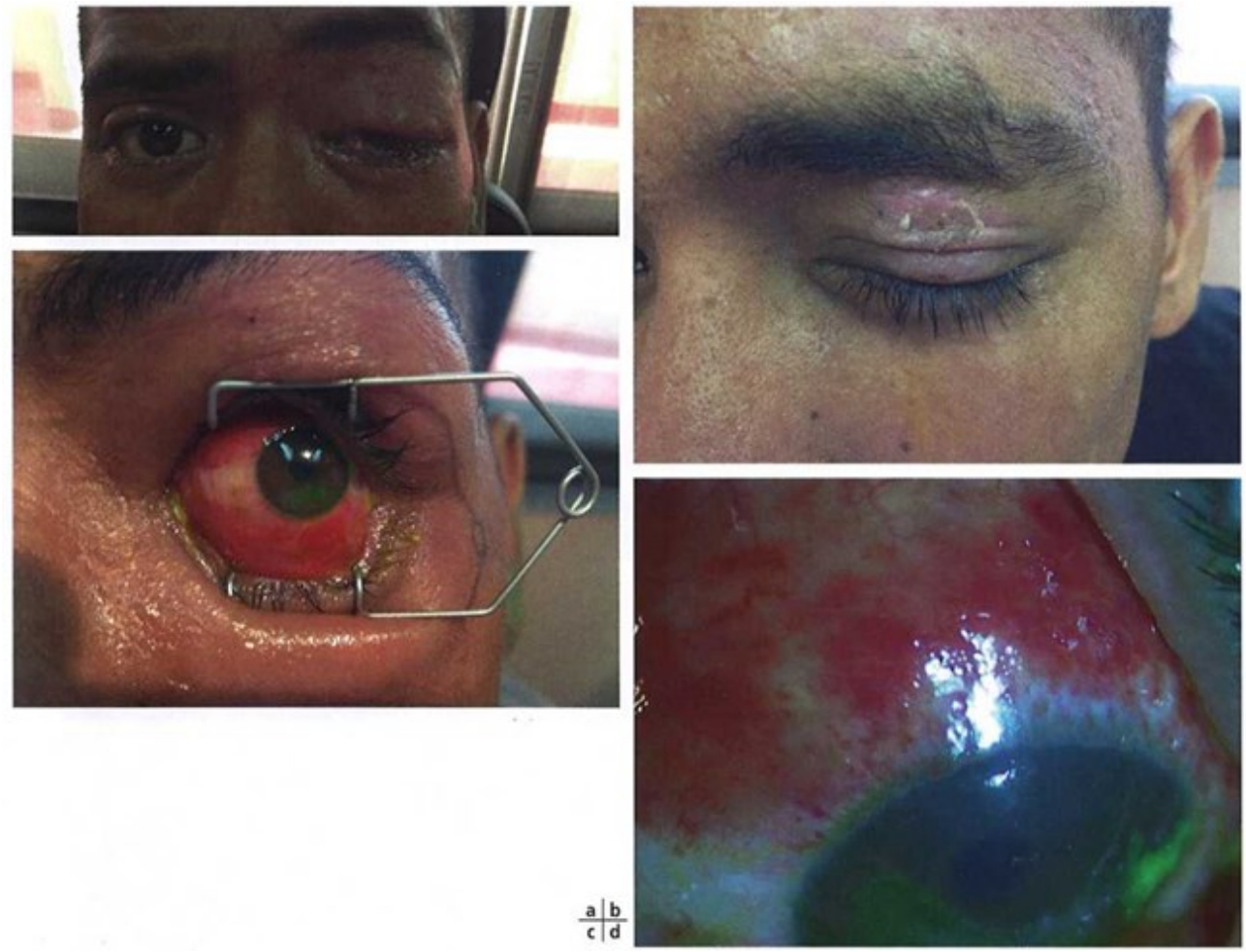

Fig. 5-1-35 ***Manchineel latex burn.***
a. First-degree burn of the left upper eyelid. **b.** Healing skin on the 8th day. **c.** Diffuse hyperhaemia of the conjunctiva, ulceration of the corneal epithelium extending over the entire lower half of the cornea. **d.** Punctate hemorrhages below the conjunctiva around the limbus. No corneal oedema or limbal ischaemia.

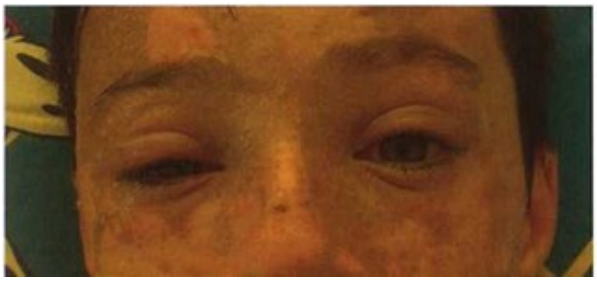

Fig. 5-1-36 ***Burn by a flame.***
Superficial, second-degree skin burn involving the whole face. Eyelids, eyelashes and eyebrows are all burned. Because of rapid blinking and Bell's phenomenon, the eyeballs were spared.

Injuries to the eyelids are common. Treatment consists of getting the patient out of the cold and wind and slowly warming him or her up by applying warm compresses [23]. Corneal injuries in the course of poorly conducted cryotherapy or iatrogenic lesions at the incision site for phacoemulsification are rare.

■ RADIATION BURNS

Burns due to ultraviolet radiation (400-280 nm) are the most common. Emission sources vary: prolonged exposure to the sun with rays being reflected (snow, ocean, desert), arc welding, disinfectant lamps, tanning apparatus. Ultraviolet rays are almost totally absorbed by the cornea and this can cause detachment of epithelial cells and stromal oedema [24]. Pain, blepharospasm, eye-watering and photophobia can onset about 12 hours after exposure. Examination shows SPK and conjunctival hyperhaemia. Problems regress within 48 hours and this can be accelerated by occlusion. A topical antibiotic is prescribed to prevent secondary infection. Cycloplegia or systemic painkiller treatment may sometimes be needed. Burns due to infra-red radiation (700-3000 nm) occur as the result of an explosion or during a solar eclipse. Injuries are limited to SPK on the cornea but this can lead to cataract or chorioretinitis. Exposure to ionising radiation (radiotherapy, radioactive isotopes) induces hyperhaemia in the conjunctiva and corneal injuries from simple SPK to full-blown perforation.

Immediate care

Therapeutic care for ocular burns has two aims:

- to eliminate or limit the aggressiveness and penetration of irritant or corrosive substances in eye tissues. This relies on the ocular rinsing which should be done as soon as possible;
- to control the inflammatory reaction and favour wound healing. This second objective relies on both medical and surgical measures which are usually coordinated.

■ Ocular rinsing

Although surgical techniques to restore destroyed limbal stem cells (LSCs) have considerably enhanced the prognosis of severe corneal burns, ocular rinsing remains a crucial step (Video 5-1-4). Outcome will depend on the earliness and effectiveness of the washing which plays a determinant role in progress and prognosis. Lesions are worse when no washing has been carried out. Washing is usually undertaken sooner after an accident at work than after an assault [10]. It should be started straight away at the place of the accident and continued during transportation of the patient to the ophthalmologist or hospital.

Video 5-1-4

Because of panic, pain or eyelid spasm, washing by the victim is not always effective and should always be repeated by professional care-providers (Fig. 5-1-37). It may be made easier by instilling anaesthetic eye drops beforehand. In children, a general anaesthetic may be required. Using Desmarres retractors or installing a blepharostat is sometimes indispensable. Setting up an infusion tube about 10 cm from the eyeball is better than using an automatic irrigation system. The disadvantage of these devices comprising a polyethylene tube or a Morgan lens (a polymethylmethacrylate scleral lens) is that they are difficult to set up, can cause iatrogenic injuries and do not ensure thorough rinsing of the whole ocular surface [25].

To expose the entire conjunctival surface area, patients should roll their eyes through all angles of view. Both eyelids need to be lifted and special attention should be paid to the conjunctival sacs. Any foreign bodies must be removed with the help of a surgical microscope. Conjunctival sacs must be scrupulously examined to look for solid particles sticking to the conjunctiva. Except the use of ethylene diamine tetra-acetic acid (EDTA) to remove particles of lime or cement, use of antidotes is not recommended. Puncturing the anterior chamber for washing is no longer recommended. Washing should last 15-30 with about 1.5 litres of solution. The pH of the ocular surface can be measured using an indicator strip and washing is continued until a normal pH (7.4) is attained [10].

Usually available on the premises of an accident, water is the rinsing solution that is universally used most frequently. Water is hypotonic with respect to the intraocular environment and can, thanks to epithelial lesions, penetrate into the corneal stroma, exacerbating the oedema and drag acidic or basic particles into the cornea. Using an isotonic or hypertonic solution is to be preferred because these create a flow going from inside the intraocular environment towards the outside. Kompa *et al.* showed that corneal oedema is inversely proportional to the osmolarity of the rinsing solution [26]. Running water and saline solutions have no buffering effect and act purely by dilution and mechanical flushing. Ringer's lactate and balanced salt solution (BSS) Plus® are tolerated better than normal saline solution. Ringer's lactate has very little buffering capacity [26]. BSS Plus® is special in that its osmolarity is close to that of the aqueous humour but it has no buffering activity. Solutions containing a phosphate buffer should be avoided because they increase the risk of irreversible corneal calcification. Moreover, the action of phosphate buffers is accompanied by an exothermic reaction. In France at the moment, only one hypertonic ocular rinsing solution is available (with an osmolarity of 820 mOsm/L). This is an amphoteric solution preservative-free, with strong buffering activity, sterile and has been used for years in the chemicals industry (Diphotérine® from Prevor) [27]. Diphotérine® is also effective against tear gas. - Amphoteric agents like EDTA act by sequestering ions and neutralising them through an amphophilic reaction. They can bind both acids and bases without either changing the pH of the medium or participating in any exothermic reaction. Washing an alkali burn with an amphoteric solution quickly restores a normal extraocular pH with inflection of the pH curve within seconds or minutes after the beginning of rinsing with Diphotérine®. Increase of the intraocular pH curve is not so fast and descent is faster [26-28]. In the light of their theoretical mechanism of action and results obtained in both laboratory and clinical context, amphoteric solutions are the most adapted for emergency rinsing of chemical burns [12, 29]. If prolonged irrigation is planned, normal saline supplemented with 0.01% lidocaine can be used. This anaesthetic solution may be tolerated better than pure normal saline. Washing with a solution containing seawater and vegetable oil (10% Calophyllum inophyllum oil and 90% Aleurites moluccana oil) may promote the regeneration of epithelial cells and attenuates inflammatory reactions [30]. Washing is also necessary for thermal burns because it helps decreasing temperature at the surface of the eyeball.

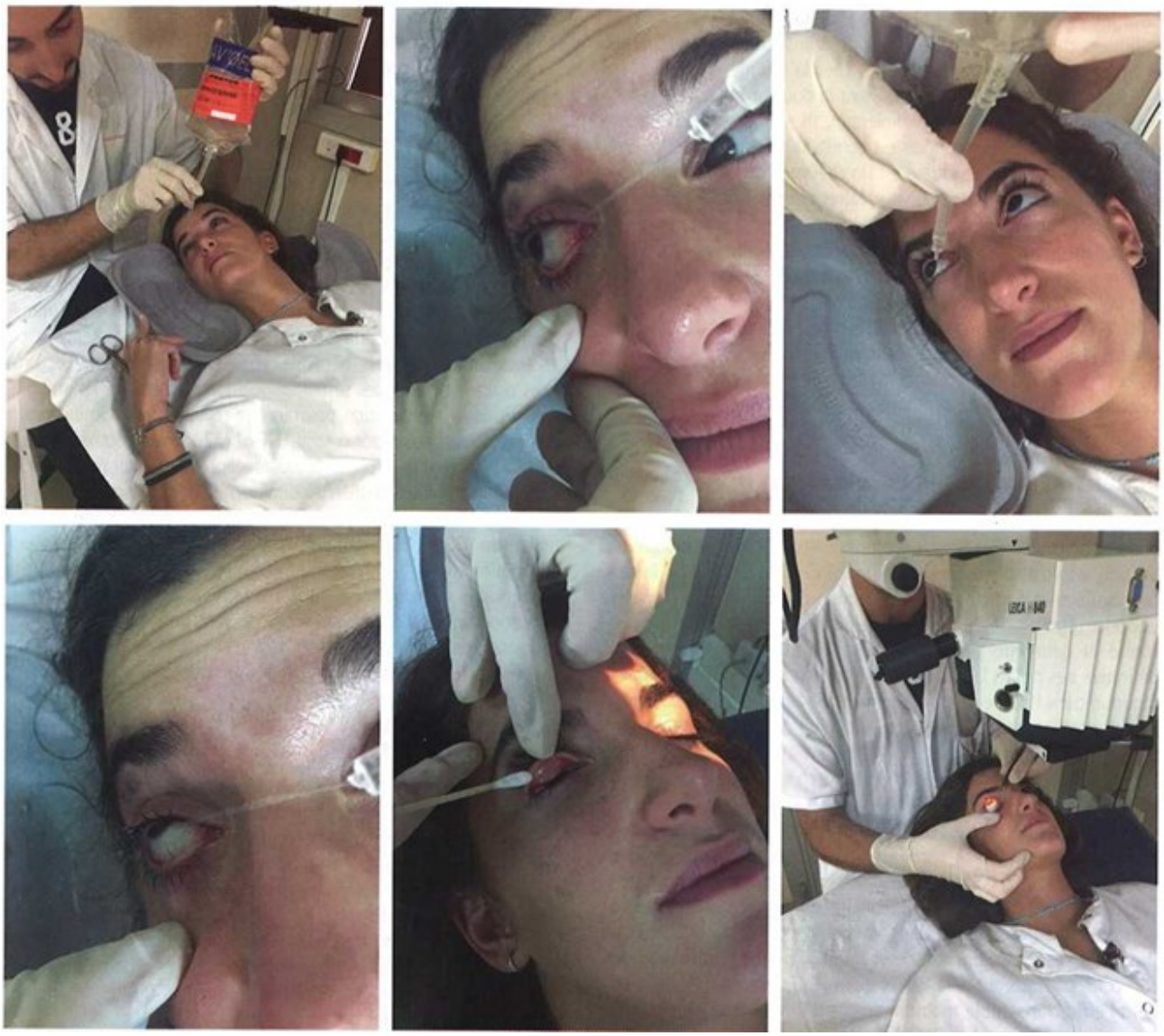

Fig. 5-1-37 ***Manchineel latex burn.***

a. Patent lying down. A disposable cardboard kidney dish is set up by the eye to be irrigated. One drop of anaesthetic solution is instilled. pH is measured using an indicator strip. **b.** an infusion tube is kept at about 10 cm from the eyeball. **c.** To expose the whole conjunctival surface, the patient should roll their eye through all angles of view. **d.** Thoroughly rinse out the conjunctival sacs. **e.** Turn both upper eyelids inside out to find any foreign bodies that should be removed using a swab. **f.** Removing foreign bodies with pliers is facilitated with the use of a surgical microscope.

■ MEDICAL TREATMENT

CONTROLLING THE INFLAMMATORY REACTION

Using topical corticosteroids now seems to be acceptable in the treatment of burns, especially chemical burns [10]. Although this has been controversial for a long time, it is justified by their ability to control inflammation. They cut down invasion of the stroma by polymorphornuclear neutrophil, stabilise cellular and lysosomal membranes in polymorphonuclear cells and inhibit collagenase activity. In chemical burns, corticosteroids cut down the destruction of conjunctival mucus cells. In animals, Donshik showed that intensive topical corticosteroid treatment for ten days after the burn does not exacerbate the risk of corneal perforation [31]. However, if corticosteroids are administered for more than eight days or for a longer course of treatment, they delay healing by inhibiting keratinocyte migration and collagen synthesis. Donshik *et al.* recommend discontinuing corticosteroids after the 10th days and restarting them after four weeks if there is still inflammation [31]. Brodovsky *et al.* and Davis *et al.* concluded that a combination of corticosteroids with topical or systemic ascorbic acid can be prescribed for more than eight days with beneficial effect and without any adverse reactions [10, 11]. The incidence of infectious complications does not seem to be affected by topical corticosteroid treatment [10, 12]. Corticosteroids can also be administered by sub-conjunctival injection: this allows a higher concentration, fewer instillation procedures and better compliance.

It has also been proposed to replace corticosteroids with non-steroid anti-inflammatory drugs or combine the two. However, these have a stabilising effect on neurological membranes, mimicking the impaired wound healing conditions seen in neuroparalytic keratopathy. Their use therefore remains controversial.

Tetracyclines inhibit collagenase activity and corneal ulceration in experimental burns. This activity is independent of their antimicrobial activity and is due to the chelation of zinc. They also inhibit the activity of polymorphonuclear cells. Administered systemically, tetracyclines cut down the incidence of corneal ulceration and promote healing [32]. Doxycycline and minocycline are prescribed at a dosage of 200 mg a day. Oral

tetracyclines are usually well tolerated but may cause digestive irritation and induce photosensitivity. They are contraindicated in children of under 8 (staining of the teeth and hypoplasia of the dental enamel) and pregnant women.

Cycloplegic eye drops reduce pain and limit the formation of iridocrystalline synechiae: they are systematically prescribed. Phenylephrine is contraindicated because of its vasoconstrictive effects. Citrate chelates calcium and inhibits the chemotaxis and adhesion of neutrophils as well as the release of lysosomal enzymes. It inhibits collagenase activity and reduces the incidence of corneal ulceration in rabbits. It is more effective when administered topically than when it is administered systemically [20]. Its use at a concentration of 10% in eye drops together with 10% ascorbate might accelerate re-epithelialisation after a severe burn [10]. Citrate is not available in France.

Collagenase inhibitors like acetyl-cysteine and synthetic thiols have been shown to be effective in experimental burns but no clinical evidence is available.

HELPING WOUND HEALING

Burns of the conjunctiva are associated with a reduction in the number of mucus cells. Impairment of the mucous layer of the lacrimal film compromises its ability to stick to the ocular surface and contributes to weakening of the corneal epithelium. Regular instillation of preservative-free artificial tears is recommended and this can be reinforced by temporarily or definitively blocking the lacrimal points. Ascorbic acid is a cofactor in collagen synthesis and its concentration in the aqueous humour is reduced in the context of a burn. Vitamin C also possesses antioxidant activity that inhibits the damaging action of free radicals released as a result of a chemical ocular burn. Pfister showed that ascorbate administered either topically or systemically reduced the incidence of thinning and ulceration of the cornea, in both the laboratory and the clinical context. Administration of eye drops containing 10% ascorbate may be more effective given the likelihood of injuries to the ciliary bodies that limit the concentration of ascorbic acid in the anterior chamber [33]. According to Brodovski *et al.*, adding Vitamin C makes it possible to continue administering topical corticosteroids for longer than a week [10]. However, it seems to be less beneficial for installed ulcers. Vitamin C eye drops are no longer available in France but an oral form can be prescribed at a dosage of 2-3 g a day [10, 12, 34].

Autologous Serum Eyedrops (ASE) contain growth factors (epidermal growth factor [EGF], transforming growth factor [3 [TGF-|3], vascular endothelial growth factor A [VEGF-A], nerve growth factor [NGF], etc.) and trophic ingredients (anticollagenase, vitamins, cytokines, fibronectin, etc.) for the cornea. ASE are used at a concentration of 20-50% diluted in 0.9% saline solution. Administered as of Day 8 together with anti-inflammatory drugs, ASE improve functional symptoms and reduce corneal inflammation, neovascularisation and clouding. ASE promote epithelial healing. No allergic-type side effects, depositions or infections have been observed. Beneficial effects are due to the anticollagenase activity of macroglobulin and the promotion of wound healing by Vitamin A. According to Sharma *et al.*, serum extracted from umbilical cord material enhances corneal healing and transparency significantly better than ASE or artificial tears. It also reduces limbal ischaemia and neovascularisation. Higher concentrations of EGF, TGF-p and NGF may explain the superiority of umbilical cord serum [35]. Practical difficulties with the production of ASE and umbilical cord serum currently limit their use in France, in particular the risks of infection of the technicians and contamination of the product as well as the absence of harmonisation between blood transfusion centres.

The assessment of matrix therapy has been under way for a number of years. ReGeneraTing Agent (RGTA) OTR4120 has proven effective in corneal healing after an experimental chemical burn and also in the clinical context of severe LSC deficiency [36].

OTHER MEDICAL TREATMENTS

Infection is prevented by instilling eye drops containing a broad-spectrum antibiotic which is relatively non-toxic to epithelia (fluoroquinolone) as well as administering a systemic tetracycline. Oral or systemic painkillers can also be readily prescribed because corneal nerve injuries can cause intense pain. Ocular hypertonia of immediate or delayed onset can be treated with pressure-reducing eye drops and systemic acetazolamide, or even surgery. Chemical ocular burns can also be complicated by retinal problems which can be prevented by the administration of inhibitors of tumour necrosis factor-α (anti-TNF-α) [37]. Systemic oxygen therapy administered in the acute phase may attenuate limbal ischaemia, accelerate epithelialisation and reduce corneal neovascularisation. It is dispensed via a face mask at a tension of 100% and a flow rate of 10L/min for an hour, twice a day with the patient sitting down. This treatment modality is easy to organise, non-invasive and inexpensive [38]. Growth factors such as fibronectin and retinoic acid are not used in current clinical practice. Contact lenses are rarely used because they cause discomfort and might favour superinfection.

ALGORITHM FOR THE EMERGENCY TREATMENT OF OCULAR BURNS

Once normal pH has been restored by washing, there is less certainty or consensus on the treatment strategy to be adopted. With an objective of standardisation and effectiveness, we have reviewed what approaches to take according to the degree of injuries as evaluated by the classification systems of Roper-Hall and Dua *et al.* (Fig. 5-1-38) [10, 18, 19, 39]. Severe burns (especially if both eyes are affected) and children require full hospital admission because the patient will have to be monitored daily with the regular instillation of various products (every hour for corticosteroids in the first few days).

■ SURGERY

Prognosis for severe ocular burns has substantially improved in the last decade as a result of the development of surgical techniques to restore destroyed LSCs.

DEBRIDEMENT AND ABLATION OF NECROTIC TISSUES

The aim of ablation is to manage the inflammatory reaction elicited by breakdown products from necrotic conjunctival tissue. This cuts down the generation of cytotoxic reactive oxygen intermediates and removes any caustic material that has collected in these tissues. It should be carried out as soon as the eyeball has been rinsed and any foreign bodies have been removed. Necrotic conjunctiva and subconjunctival tissue should be ablated, if necessary as far as the superior and inferior fornices. Only necrotic, avascular tissue should be removed down to tissue layers in which the blood circulation has been spared [40].

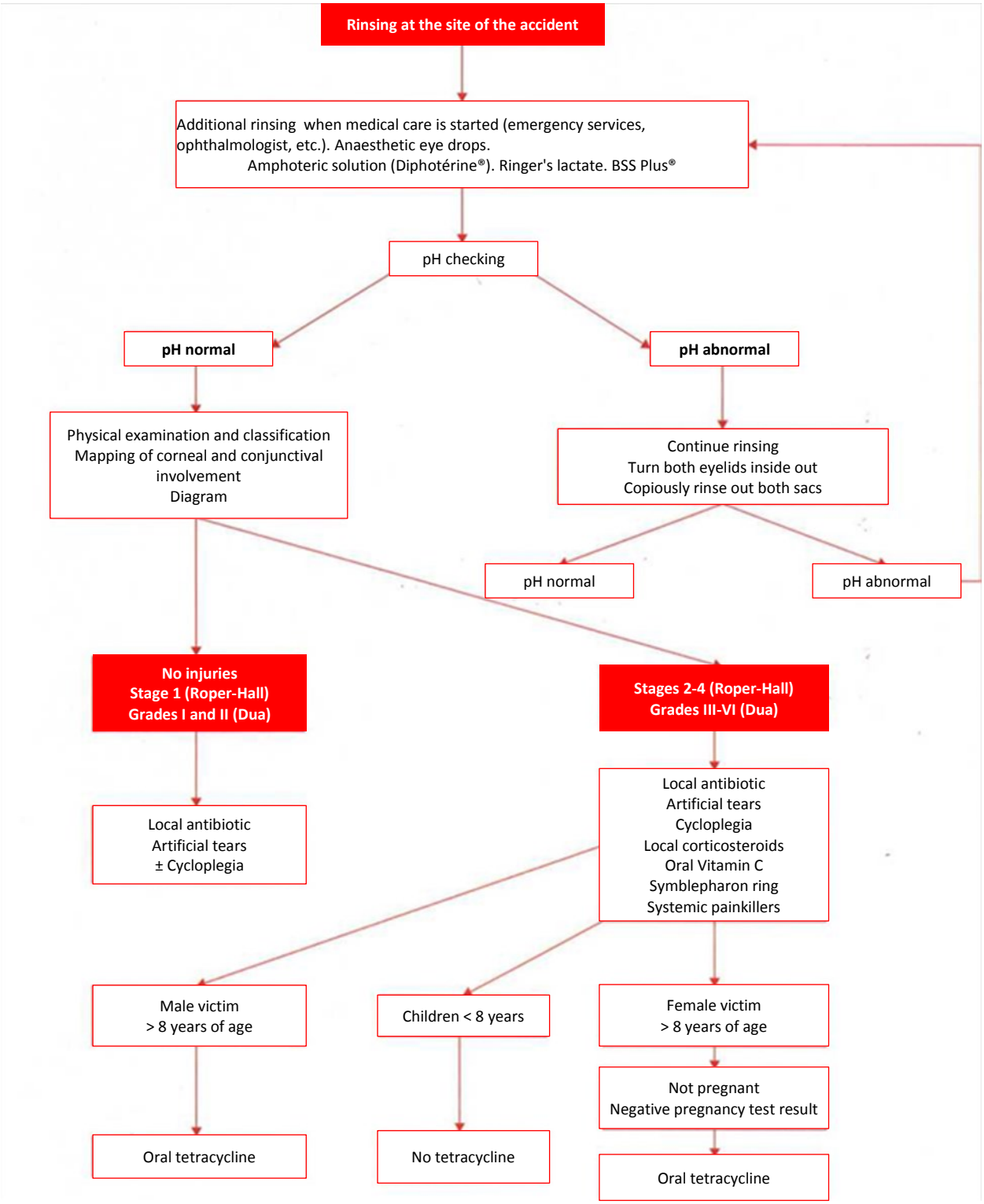

Fig. 5-1-38 ***Emergency care for ocular burns.***

TENONPLASTY

For severe ocular burns with complete loss of all limbal vessels, there is, in addition to the low likelihood of any secondary re-epithelialisation, an immediate risk of necrosis in the anterior segment. In order to restore limbal circulation and block further necrosis or aseptic ulceration, tenonplasty can be carried out. This involves creating advanced Tenon's sheets and placing them up to the limbus [40]. This procedure must be carried out early, as soon as necrotic tissue has been ablated.

PREVENTION OF SYMBLEPHARON FORMATION

Preventing symblepharon must be considered whenever there is extensive burning of the conjunctiva. A number of methods are available: regular release of adhesions using a glass rod or a swab under a local anaesthetic; installation of scleral glasses or rings made of polymethylmethacrylate (Fig. 5-1-39). Prevention also depends on the patient performing duction and version movements.

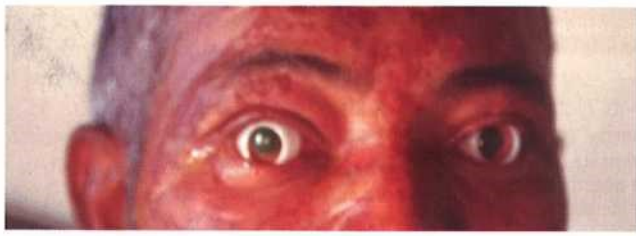

Fig. 5-1-39 ***Severe burn by base.***
Polymethylmethacrylate rings deployed in both eyes to prevent symblepharon.

SECTORAL EPITHELIECTOMY

Sectoral epitheliectomy or directed wound healing involves removing the conjunctival epithelium that covers the cornea faster than the corneal epithelium growing out from LSCs. This is carried out under local anaesthesia with biomicroscopic guidance. Conjunctival epithelium is removed using a Desmarres scarifier or Bonn forceps. It can be repeated every 24-48 hours [41].

AMNIOTIC MEMBRANE TRANSPLANTATION

The amniotic membrane is a tissue located at the interface between placenta and amniotic fluid. It consists of a single-layer epithelium, a basement lamella and avascular stroma. It contains many growth factors that promote re-epithelialisation by attenuating inflammation and scarring. It promotes migration of epithelial cells and adhesion of basal cells. It acts as a genuine substitute basement membrane and promotes epithelial phenotypic expression. Without any Class II human leukocyte antigens (HLAs), the amniotic membrane does not induce rejection. It also has antibacterial, anti-angiogenic and analgesic properties [42]. Its epithelial surface is implanted facing down (patch or inlay) to deliver a maximum of growth factors (which are concentrated in the epithelium) to the ocular surface. It is attached to the de-epithelialised cornea with separate Nylon 10/0 sutures. Several layers can be placed on top of one another. The amniotic membrane is covered by the corneal epithelium, integrated into the stroma and is then reabsorbed.

Amniotic membrane should be transplanted within a week of the accident. It is important to cover the entire cornea as well as areas where ischaemic conjunctiva will have been ablated. In partial stem cell deficiency, re-epithelialisation should exceed 75% within 15 days. Visual acuity improves in 77% of cases and symblepharon is rare. If it is carried out any later, outcomes can still be satisfactory. However, amniotic membrane transplantation is less effective for severe burns characterised by complete LSC deficiency. In consequence, amniotic membrane transplantation is not sufficient to compensate for severe LSC deficiency. In this case, it is necessary to combine it with a limbal graft.

Amniotic membrane can also be used to reconstruct conjunctival sacs after symblepharon excision. Available in the United States, ProKera® is a device consisting of cryopreserved amniotic membrane attached to a concave ring (like an anti-symblepoharon ring). Since it does not require suturing, it can be put in place and removed with ease [43].

LIMBUS AND LIMBAL STEM CELL TRANSPLANTATION

Autologous limbus transplantation as reported by Kenyon & Tseng in 1989 is the treatment of choice for repair of a destroyed corneal limbus and attendant complications [44, 45]. It is indicated in unilateral limbal insufficiency when the other eye is healthy. The whole conjunctival pannus that covers the burned cornea is ablated as far as the underlying corneal stroma and for about 3 mm beyond the limbus. Corneal neovessels should be electrocoagulated. The graft is explanted through a corneal incision located 1 mm in front of the limbus followed by centrifugal tunnelling for about 2 mm behind. In order to preclude iatrogenic limbal insufficiency in the donor eye, the length of the graft should not exceed 180°. The graft is attached to the receiving cornea with separate Nylon 10/0 sutures and to the conjunctiva with resorbable 8/0 thread. Autologous limbal transplantation induces high-quality corneal re-epithelialisation in 75-100% of cases and creates a barrier to phenomena of neovascular scarring originating in the conjunctiva [44, 46]. Autologous limbal transplantation cannot be carried out until at least six months after the accident and the eye must be free of inflammation. However, some people recommend performing the procedure earlier, i.e. before complications develop when the LSC deficiency is profound [9, 46].

The objective of allogeneic limbal transplantation, like that of autologous grafting, is to restore the normal phenotype of the corneal epithelium. Allogeneic limbal transplantation is indicated for extensive bilateral injuries or unilateral injuries affecting just one of the eyes. Tissue is taken from a corneal graft or from an eye being stored in a tissue bank. Transplantation must be performed with the same safety conditions as corneal transplantation. Allogeneic transplantation is associated with a high risk of rejection and prolonged immunosuppression is indispensable (oral corticosteroids, ciclosporin, etc.). Outcomes are not nearly as good as with autologous transplantation with only 10-45% of eyes having a visual acuity of 1/10 or better after five years [46]. An allogeneic graft from a related donor provides fresh tissue without the need for preliminary preservation as well as closer HLA compatibility but systemic immunosuppressive treatment is still required [46]. Allogeneic transplantation of LSCs grown on amniotic membrane is a recent technique. A 1 x 2 mm fragment of limbal tissue is taken from the healthy eye (which does not entail any risk of LSC deficiency). This is then cultured for three weeks on amniotic membrane to yield a graft with a diameter of about 2 cm. The epithelial tissue is grafted onto the receiving cornea together with the amniotic membrane [47].

KERATOPLASTY

Transfixing keratoplasty (TK) with a diameter of 11-12 mm affords two advantages: that of optic or tectonic TK coupled with that of boosting the number of LSCs. However, it carries a significant risk of rejection which, in practice, compromises the results. It is advantageously substituted with preliminary LSC transplantation followed, after 1-13 months, by TK of classic diameter. The overall risk of TK rejection is 10% but it is higher for chemical burns, largely because of the frequency and extent of stromal neovascularisation in the receiving cornea. TK can also be carried out at the same time as allogeneic limbal transplantation.

Exceptionally, corneal transposition (auto-TK) can be combined with autologous limbal transplantation as illustrated in Figure 5-1-40: this was a patient with sight only in his left eye who had sustained a Stage 4 burn of said eye with a strong base. Two TK procedures ended in failure as a result of successive rejections. Visual acuity in the left eye was reduced in a brightly lit environment. His cornea was white, ulcerated and heavily neovascularised. Limbal insufficiency was total. His right eye had not worked since childhood due to closed contusion. At the same time, we took a fragment of corneal tissue from the right

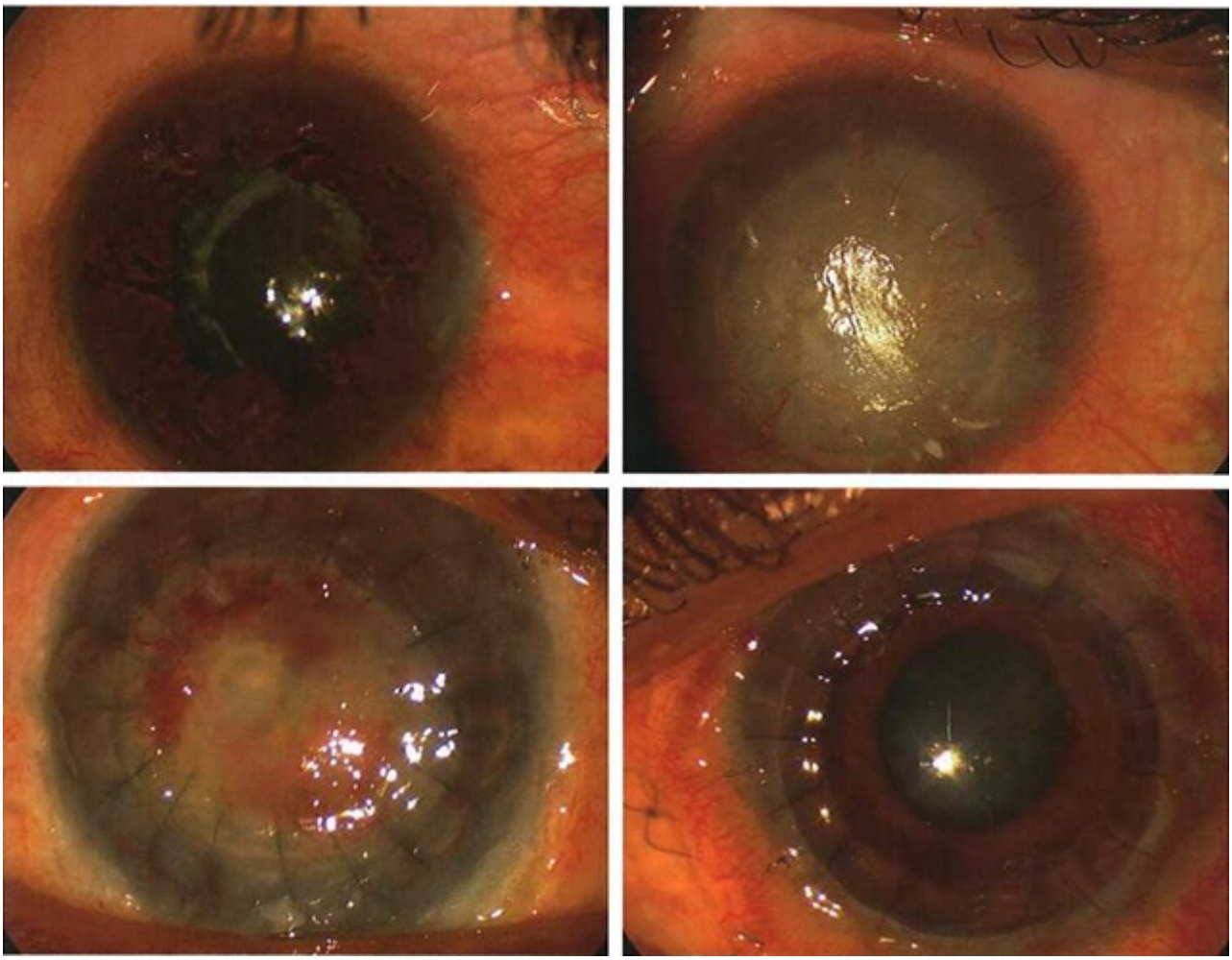

Fig. 5-1-40 ***Before (a, b) and 1 month after (c, d) corneal transposition surgery after burn.***

a. Right eye. No light perception. Cornea clear, anterior segment calm, old posterior synechiae, surgical extracapsular aphakia. Ocular tone normal. **b.** Left eye. Visual acuity restricted to good light orientation. Cornea white, oedematous, ulcerated and neovascularised. Total limbal insufficiency due to 360° destruction of the limbus. **c.** Right eye. Degenerative cornea in the left eye sutured with 16 separate Nylon 10/0 stitches. **d.** Left eye. Clear cornea of the right eye sutured with 16 separate Nylon 10/0 stitches. 360° autologous transplantation of limbus material taken from the right eye and attached with 8 separate Nylon 10/0 stitches on the corneal side and 8 separate Vicryl® 8/0 sutures on the conjunctival side.

eye (8 mm trepanation) and material from the limbus over 360°. After ablation of the conjunctival pannus that was covering the limbus and cornea of the left eye, we transplanted the cornea and limbus taken from the right eye.

Deep lamellar keratoplasty (LK) is indicated for corneal burns that have spared Descemet's membrane and the endothelium. Compared with TK, the risk of rejection is lower even if the cornea is highly neovascularised [48]. Large-diameter LK was conceived by Vajpayee in 2000. This brings in LSCs and yields a stably re-epithelialised surface. It is indicated when the burning has spared deep layers of the cornea [49].

The depth of conjunctivalisation of the cornea can be assessed by means of optical coherence tomography (OCT) which is useful in guiding surgical strategy. *In vivo* confocal microscopy and full-field OCT can help with investigation of LSC niches. Involvement of corneal stroma indicates:

- simple limbal transplantation if it is less than a third in depth;
- limbal transplantation together with LK or deep LK if it is less than two-thirds;
- limbal transplantation and TK if the entire depth is involved [50].

OTHER SURGICAL TREATMENT MODALITIES: CONJUNCTIVAL TRANSPLANTATION OF BUCCAL OR NASAL MUCOSAL TISSUE, KERATOPROSTHESIS

Conjunctival transplantation keeps indications in the repair of conjunctival sacs that have been remodelled by fibrotic scarring.

Buccal mucosa, usually taken from the posterior side of the upper or lower lip, can be used to repair symblepharon, trichiasis, distichiasis, entropion or keratinised patches on the conjunctiva or the edge of the eyelid. A graft of nasal mucosa is taken from the septum or the lower or middle turbinate. The advantages of grafting nasal mucosa is that the graft can be big as well as the fact that intraepithelial mucus cells get transplanted [51].

Keratoprosthesis remain the last surgical resort for bilateral corneal blindness when neither TK nor LSC transplantation are possible. Although it is a demanding procedure, it is still used because outcomes are sometimes very encouraging.

Preventive treatment

Prevention is mainly relevant to the world of industry, in all activity sectors. It is particularly relevant in the chemicals industry because high concentrations of substances are handled and the risk of accident persists from the reception of raw materials through the dispatch of finished products. In addition to thorough ventilation of work premises and judicious installation of machinery, people protection is key. This relies on: training on chemical risks; the establishment of a standard protocol for what to do in the event of a burn and familiarisation for all employees; safety goggles; provision of eye rinsing solutions at all potentially hazardous sites; banning lone working in hazardous places; possibly all employees to carry an individual rinsing solution with them.

The labelling of hazardous chemical substances and preparations is often the first information. This is essential since it provides important information about hazards and what precautions to take when handling dangerous materials. Many burns are associated with the handling of container bottles, especially when opening them. This is often a plastic jerry can that readily deforms without any safety system for opening. This type of packaging is extremely poorly matched to the hazardousness of the contents as Pouliquen showed in 1972 [52].

Hazardous products must be kept away from children and their packaging should be made difficult to open.

Cheap chemical products, instructions for making explosives on the Internet and the importation of illegal fireworks are all potential hazards and it may be that it is time to tighten up the legislation and regulations. Illegal fireworks are dangerous because they do not conform to safety standards, do not give safety directions and often fail to work properly. In fact, the incidence of accidents with fireworks is ten times higher in American states that have banned them [53].

Progress and prognosis

■ NATURAL EVOLUTION OF A CHEMICAL BURN

The natural evolution of chemical ocular burns will depend on the nature of the guilty product although all progress in certain similar ways. After a few minutes or hours, initial shock gives way to a debridement of necrotic tissue, and ultimately a healing phase.

The debridement phase is characterised by an influx of inflammatory cells in response to products of corneal and conjunctival breakdown (free radicals, prostaglandins, leukotrienes, etc.). Tissue burned with alkali releases N-acetyl-proline-glycine-proline and methyl-proline-glycine-proline which attract neutrophils and stimulate their proliferation. These inflammatory cells secrete various enzymes such as metalloproteases (collagenase, gelatinase, stromelysin, membrane-type protease and matrilysin) that help clean the site up by breaking collagen down. However, this exacerbates the destruction of ocular structures. Over debridement entails a risk of perforation whereas inadequate debridement brings a risk of infection.

Healing depends on the persistence of healthy tissue around the damaged part. This concerns ischaemic lesions secondary to destruction of the vascular network and injuries to corneal and conjunctival cells. Oxygen-starved cells produce factors like VEGF, TGF and fibroblast growth factor (FGF) which stimulate new vessel formation in burned tissue. Although such neovascularisation is beneficial in the conjunctiva, it is deleterious in the cornea and anterior segment. The corneal epithelium heals by virtue of cell turnover in epithelial crypts around the limbus [54]. The healing sequence has been described by Thoft, based on three axes (x, y and z) and the migration of LSCs:

- x: anterior migration;
- y: centripetal migration;
- z: migration out towards the surface.

He explains healing of a lesion in the middle of the cornea by centripetal migration of the entire edge, and that of a peripheral lesion by circumferential migration of neighbouring cells [55]. However, other, LSC-independent mechanisms for healing of the corneal epithelium exist. These mediate healing of the central cornea even if the entire limbus has been destroyed. These suggest that stem cells are distributed across the whole corneal surface. Healing of the corneal stroma depends on keratinocytes that can both break down collagen (mainly Type I) and produce it. These account for 80% of stromal cells. Keratinocyte metabolism is controlled by cytokines produced by epithelial and inflammatory cells. Keratinocytes migrate into damaged parts of the cornea from neighbouring regions. In severe burns, rates of Type I collagen synthesis and breakdown peak after about three weeks. This depends on ascorbic acid, the concentration of which in the aqueous humour is reduced during a chemical burn [33]. If all the keratinocytes have been destroyed, fibroblasts synthesise Type III collagen; this is gradually replaced by Type I collagen which helps restore the transparency and strength of the cornea. Healing of the conjunctiva can proceed through transformation of surviving cells into fibroblasts or by the division of stem cells in the fornix. The growth of fibroblastic tissue is what causes symblepharons to form as well as corneal clouding in the event of total destruction of all cornea cells and limbal stem cells. Filling in of the iridocorneal angle with such tissue is complicated by ocular hypertonia. Damage to nerve terminals is a constant feature that compromises corneal sensitivity and affects prognosis.

■ PROGNOSIS ACCORDING TO THE PROPERTIES OF THE CAUSAL AGENT

The seriousness of the injuries will depend on: the nature, concentration, quantity, exposure time, force of impact, pH, the chemical substance and area of contact with the eyeball [9]. Temperature may also play a role: a boiling hot solution will cause more injuries than a cold one. Drops of molten metal, solids and oily substances are not evacuated by blinking and powders tend to accumulate in the conjunctival sacs. Pressure and energy transfer can mechanically disrupt tissue architecture. They favour the intraocular penetration of the liquids. Bases quickly penetrate deeply into the ocular medium. The anion saponifies fatty acids in cell membranes and induces the instant death of epithelial cells. The cation reacts with carboxyl groups on collagen and glycosaminoglycans in the extracellular matrix, thereby promoting penetration of the alkali inside the eye. Ammonium ion ($NH4+$) penetrates fastest of all. The pH in the anterior chamber changes within seconds of exposure to ammonia. In addition to corneal injuries, the iris, iridocorneal angle, ciliary body and lens may also be damaged, depending on

how deeply the substance penetrates. Total destruction of the eyeball is possible [13]. Once the surface of the cornea has returned to a normal pH, the pH in the aqueous humour will normalise within 30 minutes to 3 hours. Above a pH of 11.5, ocular damages induced by bases are rapid and irreversible. Acids penetrate more slowly than bases. Protons (H+) precipitate and denature proteins, thereby killing superficial cells and disrupting the extracellular matrix. The resultant superficial coagulation stops the acid penetrating deeper into the cornea. Once the necrotic epithelium has been removed, the underlying stroma may appear perfectly transparent. However, lesions caused by strong acids are identical to those caused by bases because below a pH of 2.5, the injuries are profound and critical.

■ PROGNOSIS ACCORDING TO THE INITIAL PHYSICAL EXAMINATION

Although Stage 1 and 2 lesions heal fairly quickly leaving few sequelae, healing of more severe involvement (Stages 3 and 4) will depend on the extent of collateral injuries to limbus and conjunctiva. LSC deficiency manifests in histological analysis as invasion of the corneal surface by conjunctiva-like epithelium with caliciform cells. In clinical terms, LSC deficiency is characterised by:

- an irregular epithelial surface which can be clearly discerned under blue light after the instillation of fluoroscein;
- unstable lacrimal film;
- recurrent chronic or sterile epithelial ulcers, stromal clouding, neovascularisation and fibrovascular conjunctival pannus of the cornea (Fig. 5-1-41).

Injuries to the conjunctiva compromise the lacrimal film and often lead to retraction to the origin of symblepharons. In severe burns, apart from necrosis of cornea and conjunctiva, intraocular complications are common. These include cataract, endothelial injuries, retrocorneal membranes, superinfection, intraocular inflammation and anterior or posterior synechiae [10]. Ocular hypertonia is frequent and hypotonia (due to severe injuries to the ciliary bodies) is a very negative prognostic factor. Dryness of the eyes is common. Perforation and phthisis are ultimate complications (Fig. 5-1-42). Eyelid injuries can be complicated by fibrosis of the tarsus, dystrichiasis, entropion or ectropion. Lagophtalmos can be caused by scarring due to massive palpebral oedema or secondary to central nervous system problems induced by sedation. Lagophtalmos compromises the epithelial healing process. For alkali burns, other organs may be damaged by toxic fumes, notably the upper airways and lungs.

Conclusion

Burns, especially chemical burns, are common ocular emergencies. They can cause severe, bilateral, irreversible impairment of visual function. The initial physical examination is difficult because the functional problems complicate the procedure. The priority is to classify the lesions, establish prognosis and, most importantly, guide the treatment strategy. The ocular rinsing is absolutely essential and everyone should know how to do it , not only ophthalmologists but also anyone who is likely to have to deal with an ocular emergency (emergency room physicians, general practitioners, occupational physicians, non-medical care-providers, etc.). By virtue of better understanding of physiology of the corneal epithelium, prognosis has improved in severe forms. However,

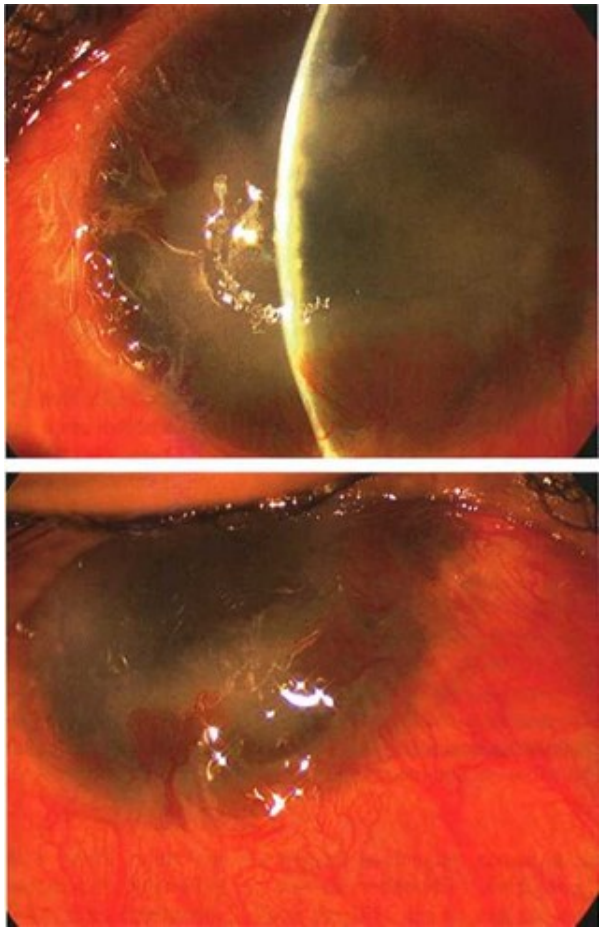

Fig. 5-1-41 ***Burn by strong base , complete cellular deficiency limbal cells***

a. Clouding of the corneal stroma, chronic, sterile ulceration, circumferential neovascularisation. **b.** Fibrovascular conjunctival pannus of the cornea

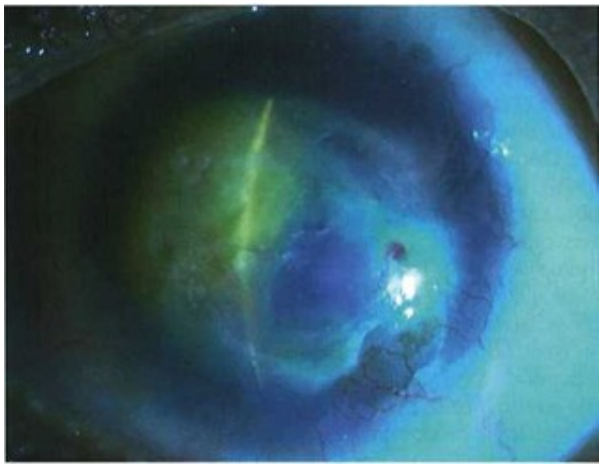

Fig. 5-1-42 ***Burn by strong base, late sequelae***

Total corneal clouding, chronic, sterile ulceration, circumferential neovascularisation.

prevention is essential when it comes to cutting the incidence down because many dramatic cases could be avoided with a minimum of information, training and regulation.

REFERENCES

References can be accessed on-line at the following address: http://www.err. consulte.com/e-complement/475395.