Chemical burns

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Objectives: To report a burn unit's experience with chemical burns and to discuss the fundamental principles in managing chemical burns.

Design: A chart review.

Setting: A burn centre at a major university-affiliated hospital.

Patients: Twenty-four patients with chemical burns, representing 2.6% of all burn admissions over an 8-year period at the Ross Tilley Regional Adult Burn Centre. Seventy-five percent of the burn injuries were work-related accidents. Chemicals involved included hydrofluoric acid, sulfuric acid, black liquor, various lyes, potassium permanganate and phenol.

Results: Fourteen patients required excision and skin grafting. Complications were frequent and included ocular chemical contacts, wound infections, tendon exposures, toe amputation and systemic reactions from absorption of chemical. One patient died from a chemical scald burn to 98% of the body surface area.

Conclusions: The key principles in the management of chemical burns include removal of the chemical, copious irrigation, limited use of antidotes, correct estimation of the extent of injury, identification of systemic toxicity, treatment of ocular contacts and management of chemical inhalation injury. Individualized treatment is emphasized.

Objectifs : Décrire l'expérience d'un service de traitement des brûlés à l'égard des brûlures chimiques et discuter des principes fondamentaux du traitement de celles-ci.

Conception : Étude de dossiers.

Contexte : Centre de traitement des brûlés d'un important hôpital affilié à une université.

Patients : Vingt-quatre patients victimes de brûlures chimiques qui ont représenté 2,6 % du total des brûlés admis en 8 ans au Ross Tilley Regional Adult Burn Centre. Soixante-quinze pour cent des brûlures ont été causées par des accidents de travail. Les produits chimiques en cause comprenaient l'acide fluorhydrique, l'acide sulfurique, la liqueur noire, diverses soudes, le permanganate de potassium et le phénol.

Résultats : Quatorze patients ont dû subir des excisions et des greffes de peau. Les complications ont été fréquentes et ont compris la présence de produits chimiques dans les yeux, des plaies infectées, des tendons exposés, l'amputation d'orteils et des réactions générales causées par l'absorption de produits chimiques. Un patient est mort des suites de brûlures causées par projection de produits chimiques sur 98 % de la surface corporelle.

Conclusions : Les principes clés du traitement des brûlures chimiques comprennent l'enlèvement du produit chimique, une irrigation abondante, l'utilisation limitée d'antidotes, une estimation exacte de l'étendue de la blessure, la définition de la toxicité systémique, le traitement des yeux et le traitement de l'inhalation de produits chimiques. On insiste sur le traitement personnalisé.

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The chemical burn stands alone among burn injuries because of its unique and unpredictable nature. Although chemical injuries account for only a fraction of all burns seen in burn units, careful, individualized attention is necessary for each one. This report presents our experience with chemical burns at the Ross Tilley Regional Adult Burn Centre in Toronto. The fundamental principles of chemical burn management are discussed.

Patients and method

The charts of all 932 patients admitted with to the Ross Tilley Regional Adult Burn Centre between Apr. 1, 1984, and Dec. 31, 1992, were reviewed to identify those who had chemical burns.

Findings

Twenty-four patients (22 men, 2 women) were admitted with chemical burns, representing 2.6% of all burn admissions. The mean age of the patients was 39 years (range from 21 to 74 years). Fifteen (62%) injuries were work-related and 2 injuries occurred in the home. The two women and one man sustained burns from assaults involving chemical agents (Table I).

A wide variety of chemicals from many different settings were encountered (Table I). In two instances, the chemical agent was unknown: an unknown alkali in one and a Chinese herbal remedy in the other. Three patients sustained uniquely "Canadian" chemical burns from black liquor, a wood softener used in the pulp and paper industry.[1] Black liquor is a potent, heated mixture of sodium carbonate, sodium hydroxide, sodium sulfide, sodium thiosulfate and sodium sulfate.[1]

The mean body surface area (BSA) involved was 21% (range from 1% to 98%). Fourteen patients (58%) with a mean full-thickness involvement of 7.5% BSA required excision and skin grafting.

Early management at the scene of the accident was reviewed in each case. Fourteen patients (58%) had involved clothing removed and immediate shower irrigation, whereas five patients did not. Five patients presented late, with a mean delay of 4.9 days, (range from 12 hours to 6 days). Interestingly, all of the patients with delayed presentation had deep burns that required excision and grafting.

Five of eight patients with ocular chemical contact received immediate eye irrigation at the scene, but, surprisingly, three did not.

On arrival to the burn centre, all burns were liberally showered over a hydrotherapy tank with tap water for 45 minutes to 1 hour, regardless of the irrigation done before admission. Patients were not immersed in the tub as part of our irrigation protocol. The eyes were copiously irrigated with normal saline if contact had occurred. The burn wounds were managed with tetanus prophylaxis, daily tap-water shower irrigation, débridement and topical application of silver sulfadiazine. Early excision and grafting was performed for any burn that did not appear likely to heal within 3 weeks of injury.

Fourteen patients (58%) sustained complications (Table II). All who presented late sustained one or more wound-related complications (burn-wound infections, tendon exposure, loss of skin grafts and toe amputation). One 23-year-old man died. He sustained 98% BSA full-thickness chemical scalding burns after a black liquor explosion. This patient was not resuscitated and was managed with compassionate care only, because of the magnitude of the injury. He died within 24 hours of the injury from hypotension-induced cardiac arrest.

The ocular complications deserve

Table I

Etiology of Chemical Burns in 24 Patients

Chemical	No. of patients	Etiology
Protoplasmic poisons		
Hydrofluoric acid	2	Industrial cleaning, tile etching
Dessicants		
Sulfuric acid	4	Food industry, assaults, farming
Corrosives		
Black liquor	3	Pulp and paper industry
Potassium hydroxide	1	Restaurant kitchen (cleaning solution)
Sodium hydroxide	7	Industrial cleaning, film processing, glue industry, environmental detoxification, home, industrial laundry cleaning
Phenol	2	Industrial cleaning, chemical industry
Aqueous ammonia	2	Refrigeration (meat packing)
Unknown alkali	1	Assault
Oxidizing agents		
Potassium permanganate	1	Stag party
Unknown agent	1	Chinese herbal remedy

special note. Eight patients were splashed in the eyes with chemicals; all had conjunctivitis. In three cases, this was prolonged and lasted up to 5 months. In each case associated with prolonged conjunctivitis, no primary eye irrigation had been provided as part of the first aid. Six patients suffered corneal erosions; in three eye lavage had not been provided at the scene of the injury. One patient sustained a very deep corneal ulceration from black liquor, resulting in unilateral blindness. Although this patient's eyes had been flushed at the scene, the overwhelming potency of the corrosive was probably to blame.

Discussion

Chemical burns are most often occupation-related and account for only 2% to 4% of burn admissions.[2–4] Our findings are consistent with this pattern.

Thousands of chemicals, used commonly in industry and in the home, are capable of producing cutaneous injury.[5] Therefore, it is helpful to classify chemicals into groups based on their activity and mechanism of injury.[2,6] A chemical may belong to more than one group. For example, hydrochloric acid is classified as an acid and as a protoplasmic poison, and in high concentration (muriatic acid) it also acts as a dessicant.

Oxidizing agents denature tissue proteins and are themselves oxidized during this process. Frequently, the oxidized agent is cytotoxic and causes further cell damage.[2] Protoplasmic poisons act by forming salts with cellular proteins (e.g., hydrofluoric acid liberates fluoride, which readily binds calcium ions).[2] Dessicants dehydrate cells and usually produce exothermy in doing so.[2] Vesicants incite a chain of physiologic reactions with release of tissue amines.[2] Acids with a pH less than 2 can produce coagulation necrosis on contact with the skin. Alkalis with a pH greater than 11.5 produce severe tissue injury through liquefaction necrosis.[2] Liquefaction loosens tissue planes and allows deeper penetration of the agent. For this reason, alkali burns tend to be more severe than acid burns. It should be noted that all of these reactions may be accompanied by exothermy, which contributes to tissue injury.[2]

This classification, although useful as an overview, is an oversimplification. Several chemicals encountered in this series have complex and incompletely understood pathophysiologic characteristics.

Two burns resulted from contact with hydrofluoric acid. These burns are characterized by intense pain and progressive tissue destruction.[7] The initial tissue burn is a caustic injury caused by the high concentration of hydrogen ion.[8] Once the epidermis is penetrated, hydrofluoric acid, which is highly permeable, diffuses throughout the dermis and into deeper tissue, including bone.[8] The acid easily dissociates to liberate free fluoride ions, which produce intense liquefaction necrosis. Both intra- and extracellular calcium and magnesium are depleted, because they readily bind with free fluoride, disrupting numerous biochemical processes.[8] Life-threatening cardiac arrhythmias can result from systemic hypocalcemia after contact with concentrated hydrofluoric acid to as little as 2.5% BSA.[9] The fluoride ion itself is

Table II

Complications of Chemical Burns in 24 Patients

Complication	No. of patients	Chemical
Wound		
Burn wound infection	3	Hydrofluoric acid, sodium hydroxide, unknown alkali
Skin graft loss (tendon exposure)	1	Sodium hydroxide
Severe hypertrophic scars	1	Sulfuric acid
Keloid	1	Sodium hydroxide
Toe amputation	1	Potassium hydroxide
Ocular		
Prolonged conjunctivitis	3	Aqueous ammonia, sodium hydroxide
Corneal erosion/ulceration	6	Aqueous ammonia, sodium hydroxide
Permanent loss of vision	1	Black liquor
Pulmonary		
Inhalation injury	2	Black liquor, aqueous ammonia
Pneumonia	2	Phenol, aqueous ammonia
Sepsis		
Staphylococcus aureus, Escherichia coli	1	Phenol
Enterococcus, *Klebsiella* sp	1	Black liquor
Systemic toxicity		
Pulmonary edema and acute renal failure	1	Phenol
Metabolic acidosis	1	Sulfuric acid

toxic and may induce respiratory arrest and ventricular arrhythmias.[8]

Personnel rendering treatment should protect themselves from contact with hydrofluoric acid by wearing gloves, eye shields and plastic aprons. Surface irrigation itself is inadequate therapy. The penetrating free fluoride ions must be neutralized. Minor exposures may occasionally be managed by irrigation and topical application of 2.5% calcium gluconate jelly, taking pain resolution as the end point of treatment.[7,8] However, deeper absorption of topical calcium gluconate is variable, and many authors recommend subeschar injection of 10% calcium gluconate.[7,8,10] Shewmake and Anderson[10] suggested a sublesional injection of 0.5 mL of 10% calcium gluconate/cm^2 of involved tissue, taking pain resolution as the therapeutic end point. Intra-arterial infiltration of dilute calcium salts for hand and digital exposures has been reported.[8,11] Finally, prompt excision of involved tissue may help to limit systemic toxicity.[8] Frequent monitoring and supplementation of serum calcium and magnesium are necessary. Hypercalcemia has been reported with overly aggressive calcium administration.[12]

Phenol was implicated in two cases. Although the phenol burn itself often appears innocuous, systemic absorption of phenol can have life-threatening consequences.[13] One 40-year-old man with a 40% BSA phenol injury, suffered phenol-induced renal failure within 48 hours of injury (Table II). This patient had been showered with tap water both at the scene and on arrival at the burn unit. Polyethylene glycol was not used. After 48 hours oliguria developed, with elevation of the serum creatinine level to 345 µmol/L. By the 4th day after injury, oliguria persisted and the serum urea nitrogen and serum creatinine levels had risen further to 20.1 mmol/L urea and 810 µmol/L respectively. Continuous arteriovenous hemofiltration (CAVH) was initiated. By the 9th day, normal urine output had returned and the serum creatinine level had dropped to 142 µmol/L; CAVH was discontinued. The patient made a full recovery.

Phenol is readily absorbed through the skin and across the lungs when inhaled as a vapour.[14] The phenol may be excreted in the urine in its free form or as a conjugated or oxidized compound.[14] Systemic toxicity is proportional to the plasma concentration of free phenol. Phenol is a potent central nervous system stimulant causing hyperreflexia and convulsions.[14] Depression of the central nervous system and respiratory arrest ultimately follow.[14] Peripheral nerves are demyelinated and destroyed, with local anesthesia developing at contact sites.[14] Phenol causes not only central cardiac depression but also has a direct negative effect on the myocardium.[14] Erythrocytes exposed to phenol can produce methemoglobin and may undergo lysis.[14] Central lobular necrosis may be seen in the liver after phenol absorption.[14] Finally, renal failure is produced by direct damage to glomeruli and tubules by phenol, as well as by tubular precipitation of hemoglobin.[14]

At a minimum, contact sites should be aggressively showered with tap water to remove phenol from the skin. Gentle lavage or swabbing only spreads the phenol over a wider area, increasing potential for absorption.[14] Because phenol is water insoluble, a solvent such as polyethylene glycol, molecular weight 400 (PEG 400), is a useful adjunct. Swabbing with a 50% PEG 400 solution, combined with high-density shower is ideal.[13,14] In this case, mannitol diuresis and urine alkalinization were not used, but these manoeuvres, combined with aggressive intravenous fluid resuscitation, may prevent renal failure.

Black liquor commonly produces a combination thermal and alkali chemical burn. It has a pH of 11 to 13 and is usually used at temperatures of 85 °C to 90 °C.[1] In two cases, the black liquor was released under pressure, resulting in inhalation of aerosolized chemical in one patient and ocular contact in another. Black liquor contacts should be initially managed with copious tap water lavage.

Because of their unique nature, chemical burns must be managed in a careful individualized manner. The following principles should be employed in treating chemical burns (Table III[6]).

Remove the chemical

The duration of the chemical's contact with the skin is the major determinant of injury severity. Chemical burns are characterized by ongoing tissue destruction for as long as the inciting agent is present.[3,15,16] The dermis is more permeable to toxins than the epidermis, and absorption occurs extremely efficiently once the epidermis is destroyed.[15] Hence, immediate removal of the chemical is of paramount importance. This includes removal of involved clothing and footwear, brushing away dry chemical, and immediate water lavage at the scene.[17,18] In this series, it is notable that all five patients who presented late had deep injuries requiring grafting. Presumably this was directly related to prolonged contact with the chemical.

Irrigate! Irrigate! Irrigate!

This is of such fundamental importance that it demands repetition. Water lavage not only dilutes but also removes the chemical. There is ample experimental and clinical evidence supporting immediate irrigation. Gruber, Laub and Vistnes[19] demonstrated less pronounced changes in tissue pH

when sodium hydroxide and hydrochloric acid burns were irrigated immediately. Leonard, Scheulen and Munster[20] have shown that the severity of burn and length of hospital stay are both reduced when water irrigation is initiated in the field.

In addition to lavage at the scene, shower irrigation should be repeated when the patient arrives at the burn centre, even if the presentation is delayed.[21] Although copious tap-water lavage should be used for virtually all chemical burns, there are a few notable exceptions.

Some chemicals create significant exothermy when combined with water, and other chemicals are insoluble in water.[3] Dry lime contains calcium oxide, which reacts with water to form calcium hydroxide, an injurious alkali.[20] Therefore, dry lime should be dusted off the skin prior to lavage.[20] As already discussed, phenol is insoluble in water and should first be wiped off the skin with sponges soaked in solubilizing agent such as 50% polyethylene glycol.[14,16] Muriatic acid and concentrated sulfuric acid produce extreme heat when combined with water. These agents should be neutralized with soap or lime water before lavage.[3,6] Finally, there is no role for immersion therapy in a burn tank. Hydrotherapy by immersion carries the risk of nosocomial bacterial dissemination, electrolyte imbalances and hypotension.[22,23] Shower lavage by a hand-held unit over a hydrotherapy tank is preferable to immersion. The risk of nosocomial infection may be reduced by using sterile solutions (e.g., normal saline) instead of tap water for subsequent hydrotherapy of the burn wound.[22,23]

The question of antidotes

In most instances, antidotes should be avoided. Dilution, not neutralization, is the key to therapy.[16] Problems associated with antidotes include delay of hydrotherapy while the antidote is sought, exothermy from the neutralization reaction and toxicity from the antidote itself.[16] Again, however, there are a few exceptions. As noted, phenol burns should be swabbed with polyethylene glycol sponges prior to a high-density shower.[14,19] Muriatic and sulfuric acid burns should be neutralized with soap.[3,6,17] The exquisitely painful hydrofluoric acid burn is treated with a combination of water lavage and subcutaneous injection of 10% calcium gluconate to neutralize free fluoride ions, as mentioned previously.[2,10]

White phosphorous, used in the military, in fireworks and in some insecticides, may ignite spontaneously on exposure to air.[6,17] Recommended treatment includes lavage with 1% to 2% copper sulfate, copious water lavage or immersion and meticulous

Table III

Principles of Emergency Treatment for Chemical Burns

Principle	Action/comment
1 — Removal of chemical	Remove particulate debris, brush off dry chemical, water lavage
2 — Irrigation (dilution)[6]	Copious high-density shower with tap water, do NOT immerse Notable exceptions: Phenol — wipe off with 50% polyethylene glycol sponges before lavage Sulfuric and muriatic acids — soda lime or soap wash, avoid irrigation Chlorox — milk, egg white or 1% sodium thiosulfate wash, then irrigation
3 — Antidotes[6]	Generally to be avoided: DILUTION NOT NEUTRALIZATION Notable exceptions: Hydrofluoric acid — subeschar injection of 10% calcium gluconate until pain is relieved, up to 0.5 mL/cm^2. Monitor calcium and magnesium White phosphorus — lavage with 1% or 2% copper sulfate, immerse in water (note toxicity of copper sulfate)
4 — Extent of burn	Deceptive, be aware of tendency to underestimate extent of burn
5 — Systemic toxicity	Consult nearest poison control centre for information on toxicity of chemical agent
6 — Ocular contact	Water lavage at scene. Irrigate eye(s) with continuous stream of 1–2 L of normal saline via intravenous tubing and 18-gauge angiocatheter. Ophthalmology consultation
7 — Inhalation injury	Be suspicious if chemical is in aerosol form. Consider intubation, supplemental oxygen, bronchoscopy for diagnosis

débridement of phosphorous particles. Excess copper sulfate must be removed, because absorption may cause hepatic and renal damage.[6] It is important to remember that neutralizing agents can themselves cause toxicity. The practitioner is advised to be familiar with the toxic effects of any antidote used.

Estimating the extent of the burn

Estimating the depth and extent of a chemical burn is notoriously difficult because of the unusual tanning and local anesthetic properties of some chemicals. For example, phenol burns are firm, leathery and insensate and may appear brown, dull grey or progress to black gangrene, whereas lye burns typically are erythematous with bullae.[14]

A significant deep burn may appear deceptively superficial. The burning process is insidious and ongoing. Classically, the magnitude of the injury is underestimated and fluid administration is insufficient. Therefore, the tendency should be toward aggressive over-resuscitation with appropriate tailoring of therapy based on careful observation of the patient's response.[2,16] Urine output is the single most important guiding parameter.

Systemic toxicity

The physician managing a chemical burn must be aware of any possible toxicity from systemic absorption of the agent. Bizarre and often fatal reactions may occur.

Hydrofluoric acid toxicity includes hypocalcemia and ventricular fibrillation.[4] Absorption of petroleum distillates may result in pulmonary hemorrhages, cardiac arrhythmias and renal and hepatic injury, leading to toxicity.[24] Formic acid absorption can produce intravascular hemolysis, renal failure and necrotizing pancreatitis.[24] White phosphorous is associated with ventricular arrhythmias and hepatic and renal injury.[24]

Consult a toxicologist

In most cases a toxicologist should be consulted, usually through the regional poison control unit, which is an excellent source of information and literature references regarding the toxicity of virtually any chemical.

Ocular injury

The eye is often involved in chemical burns.[4] Almost half of our patients had ocular involvement. A chemical burn to the eye is an emergency and an ophthalmologist must be consulted immediately.[25] Irrigation with water must start at the scene of the injury and should be continued during transportation to the burn centre. This is most easily accomplished by immersing the face in water and having the patient open and close the eyes continuously.[2] In this series, failure to provide this basic first aid usually resulted in prolonged conjunctivitis or a corneal erosion. Our preferred method of eye irrigation, when the patient arrives in the burn unit, is to run a continuous stream of 1 to 2 litres of normal saline into the eye through intravenous tubing and an 18-gauge angiocatheter, with the eyelid everted. One staff member is specifically designated to perform the irrigation.

Subsequent treatment usually involves administration of cycloplegic and antimicrobial drops. The eye is not patched so that globe mobility is maintained.[2,18,25]

Inhalation injury

Concomitant respiratory injuries may occur when aerosolized chemical or smoke is inhaled. In this series, two inhalation injuries occurred (Table II). The practitioner must be acutely aware of the possibility of inhalation injury in all cases of chemical burns. The diagnosis is usually made with the fiberoptic bronchoscope.

Chemical inhalation injuries, like smoke inhalation injuries, are managed with airway protection and supplemental oxygen, by mechanical ventilation with positive end-expiratory pressure and aggressive chest physiotherapy. The administration of steroids and antibiotic prophylaxis are not indicated.

Conclusions

Chemical burns are unique injuries for which individualized attention and management are necessary. Complications are frequent and must be identified early and treated. The important principles of management include early removal of the chemical and copious water lavage, avoidance of neutralizing agents except in selected instances, appreciation of the extent of the burn, familiarity with the systemic toxicity of the chemical and recognition of ocular and pulmonary involvement.

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