6 Chemical Skin Burns

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6.1 Introduction

Chemical skin burns are particularly common in industry, but they also occur in non-work-related environments. Occupationally induced chemical burns are frequently noticed when visiting and examining workers at their work sites. Corrosive chemicals used in hobbies are an increasing cause of skin burns. Disinfectants and cleansers are examples of household products which can cause chemical burns. However, in most cases, the cause of a chemical burn is obvious to the affected persons and damage is minimal and heals without medical care, so medical attention is not sought. Sometimes the chemical burns are severe and extensive with the risk of complications and long-term disability. In the acute stage, there is a varying risk of systemic effects, including a fatal outcome, depending on exposure conditions and the incriminating agent. For these reasons it is important for the physician to have knowledge of corrosive chemicals as well as of chemical burns with regard to their clinical manifestations, specific medical treatments, and preventive measures.

6.2 Definition

A caustic burn (chemical burn) is an acute, severe irritant reaction by which the cells have been damaged to a point where there is no return to viability; in other words, a necrosis develops [7, 43, 45]. One single skin exposure to certain chemicals can result in a chemical burn. These chemicals react with intra- and intercellular components in the skin. However, the action of toxic (irritant) chemicals varies causing partly different irritant reactions morphologically. They can damage the horny layer, cell membranes, lysosomes, mast cells, leukocytes, DNA synthesis, blood vessels, enzyme systems, and metabolism. The corrosive action of chemicals depends on their chemical properties, concentration, pH, alkalinity, acidity, temperature, lipid/water solubility, interaction with other substances, and duration and type (for example, occlusion) of skin contact. It also depends on the body region, previous skin damage, and possibly on individual resistance capacity.

Many substances cause chemical burns only when they are applied under occlusion from, for example, gloves, boots, shoes, clothes, caps, face masks, adhesive plasters, and rings. Skin folds may be formed and act occlusively in certain body regions, e.g., under breasts and in the axillae. Many products, which under ordinary skin exposure conditions cause weak irritant reactions or irritant contact dermatitis, can under occlusion cause chemical burns, e.g., detergents, emulsifiers, solvents, plants, woods, topical medicaments, toiletries, insecticides, pesticides, preservatives, cleansers, polishes, paint, plastic monomers, and Portland cement. Wet cement can usually be handled without causing a chemical burn, but when present under occluding clothes for some hours, it can cause severe skin damage, e.g., on knees. White spirit causes only slight dryness at open application, but causes blisters under occlusion.

There are different mechanisms for reactions between skin components and agents causing chemical and thermal burns. Chemical agents cause progressive damage until either no more chemical remains unreacted in the tissue or the agent is inactivated by treatment, while thermal damaging effects cease shortly after removal of the heat source.

Acids
Acetic acid
Acrylic acid
Benzoic acid
Boric acid
Bromoacetic acid
Chloroacetic acids
Chlorosulfuric acid
Fluorophosphoric acid
Fluorosilicic acid
Fluorosulfonic acid
Formic acid
Fumaric acid
Hydrobromic acid
Hydrochloric acid
Hydrofluoric acid
Lactic acid
Nitric acid
Perchloric acid
Peroxyacetic acid
Phosphonic acids
Phosphoric acids
Phthalic acids
Picric acid
Propionic acid
Salicylic acid
Sulfonic acids
Sulfuric acid
Tartaric acid
Toluenesulfonic acid

Alkalis
Amines
Ammonia
Barium hydroxide
Calcium carbonate
Calcium hydroxide
Calcium oxide
Hydrazine
Lithium hydroxide
Potassium hydroxide
Sodium carbonate
Sodium hydroxide
Sodium metasilicate

Miscellaneous
Acethyl chloride
Acrolein
Acrylonitril
Alkali ethoxides
Alkali methoxides
Allyl diiodine
Aluminium bromide
Aluminium chloride
Aluminium trichloride
Ammonium difluoride
Ammonium persulfate
Ammonium sulfide
Antimone trioxide
Aromatic hydrocarbons
Arsenic oxides
Benzene
Benzoyl chloride
Benzoyl chlorodimethylhydantoin
Benzoyl chloroformiate
Borax
Boron tribromide
Bromine
Bromotrifluoride
Calcium carbide
Cantharides
Carbon disulfide
Carbon tetrachloride
Chlorinated acetophenons (tear gas)
Chlorinated solvents
Chlorobenzene
o-chlorobenzylidene malononitrile (tear gas)
Chlorocresols
Chloroform
Chlorophenols
Chromates
Chromium oxichloride
Chromium trioxide
Creosote
Cresolic compounds
Croton aldehyde
Dichloroacetyl chloride
Dichromates
Dimethyl acetamide

Miscellaneous
Dimethyl formamide
Dimethyl sulfoxide (DMSO)
Dioxane
Dipentene
Dithranol
Epichlorohydrine
Epoxy reactive diluents
Ethylene oxide
Ferric chloride hexahydrate
Fluorides
Fluorine
Fluoro silicate
Formaldehyde
Gasoline
Gentian violet
Glutaraldehyde
Halogenated solvents
Hexylresorcinol
Iodine
Isocyanates
Kerosene fuel
Limonene
Lithium
Lithium chloride
Mercury compounds
Methylchloroisothiazolinone
Methylenedichloride
Methylisothiazolinone
Morpholine
Perchloroethylene
Peroxides
Benzoyl
Cumene
Cyclohexanone
Hydrogen
Methylethylketone
Potassium
Sodium
Phenolic compounds
Phosphorus
Phosphorus bromides
Phosphorus chlorides
Phosphorus oxichloride

Miscellaneous
Phosphorus oxides
Piperazine
Potassium
Potassium cyanide
Potassium difluoride
Potassium hypochlorite
Potassium permanganate
Povidone iodine
Propionic oxide
Propylene oxide
Quaternary ammonium compounds
Reactive diluents
Sodium
Sodium borohydride
Sodium difluoride
Sodium hypochlorite
Sodium sulfite
Sodium thiosulfate
Styrene
Sulfur dichloride
Sulfur dioxide
Sulfur mustard
Thioglycollates
Thionyl chloride
Tributyltin oxide
Trichloroethylene
Turpentine
Vinyl pyridine
White spirit
Zinc chloride

Table 1. Agents causing chemical burns. The chemicals listed are the most common reported to cause chemical burns in industries, hobbies, and households. The list feature strong corrosive substances and also less irritating compounds that require special conditions, for example occlusion, to cause chemical burns.

The most commonly reported chemicals that can cause chemical burns are listed in Table 1. Acids and alkalis have been grouped separately, as the corrosive effect within the respective group is exerted through the same mechanism. These groups contain both strong and weak acids and alkalis, respectively. The other compounds are listed together although their corrosive effects are mediated through different mechanisms. Most of these compounds are neutral. However, some are weak acids or alkalis but are considered to be corrosive due to properties other than acidity or alkalinity, respectively.

6.3 Diagnosis

It is usually easy to arrive at a diagnosis of chemical skin burn as the symptoms are easily recognized and the exposure to a corrosive agent obvious. However, sometimes the exposure is concealed, at least initially. For example, hospital personnel may be exposed to ethylene oxide which may remain in gowns and straps after sterilization [5], and cleaners may occasionally be exposed to a corrosive agent contaminating nonhazardous objects in a laboratory. Corrosive substances under occlusion may also, at least initially, confuse and delay the diagnosis [10]. Chemical skin burns caused by skin preparations can be misdiagnosed as electrical burns or pressure sores [32]. Occasionally, a chemical burn can mimic other dermatoses, e.g., ethylene oxide can mimic bullous impetigo.

6.4 Clinical Features

Not only the skin but also the eyes, lips, mouth, esophagus, nose septum, glottis, and lungs can be directly affected. As a result of resorption toxic chemicals can damage the blood, bone marrow, liver, kidneys, nerves, brain, and other organs. The most common locations of chemical burns on the skin are the hands and face/neck, but the whole body can be affected. The exposure usually occurs by accident. However, occasionally, a chemical burn is the result of malingering. The major symptoms are burning and smarting. Morphologically, chemical burns are characterized by erythema, blisters, erosions, ulcers, and necrosis with surrounding erythema. Usually, the symptoms develop immediately or in close connection to exposure, but certain chemicals, such as phenols, weak hydrofluoric acid, and sulfur mustard gas can give delayed reactions which first appear several hours, or even a day, after the exposure.

Strong acids coagulate skin proteins, and further penetration is decreased by the barrier formed. Some common toxic chemicals affect the skin in a special way [26]. Principally, all strong acids give the same symptoms and major features, including erythema, blisters, and necrosis. Some acids discolor the skin, e.g., producing a yellow color from nitric acid. The action of hydrofluoric acid in the skin differs from other strong acids [24, 48]. It causes liquefaction necrosis, and the penetration may continue for days. When an area above 1% of the total body surface is affected, systemic effects can arise. In the skin, this acid causes much stronger pains than other acids. Diluted hydrofluoric acid can cause pain starting several hours or even a day after the exposure. For example, when bricklayers use this acid at a concentration of 10%–30% for rinsing brick walls, it may penetrate into their nail beds and, there, cause severe pain after several hours. The strong pain is due to the capacity of fluorine ions to bind calcium in the tissue, which affects the nervous system. Hydrofluoric acid can penetrate to the bone and cause decalcification there. Also, fluorides and fluorosilicic acid can give the same types of symptoms.

Alkalis often cause more severe damage than acids, except hydrofluoric acid [4, 20, 51]. The necrotic skin first appears dark brown and then changes to black. Later, skin becomes hard, dry, and cracked. Generally, no blisters appear in the skin. Alkalis split proteins and lipids, and there is a saponification of the released fatty acids. The emulsifying effect of the soap formed facilitates further penetration of the alkali into deeper layers of the skin. Chemical burns from alkaline chemicals are more painful than from acids, except from hydrofluoric acid. Because of its alkalinity, cement mixed with water can cause acute ulcerative damage [1, 18, 27, 30, 31, 34, 42, 44, 47]. Severe skin damage has involved the lower limbs, often after kneeling on wet concrete or when it gets inside boots or shoes. Sometimes, necrotic skin appears 8–12 h after exposure. Rarely, hands can also be affected, particularly when the insides of gloves have been contaminated. The alkalinity can also vary considerably between batches from the same cement factory.

Phenolic compounds such as phenol, cresol, chlorocresol, and unhardened phenolic resins penetrate the skin easily and can damage peripheral nerves, resulting in insensibility. Sometimes, peripheral nerves can be affected without visible damage to the skin. After exposure to phenolic compounds, the local blood vessels become constricted, which can contribute to the development of the necrosis. Shock and renal damage can appear after absorption of phenolic compounds [21, 28, 39].

Sulfur mustard, 2,2'-dichlorodiethyl sulfide, is

a chemical warfare agent [33, 36, 40]. It has been dumped into the sea, and fishermen have been injured when leaking containers get in their nets. The chemical is a viscous liquid below and a gas above 14°C. On the skin, the liquid causes blisters and necrosis 10–12 h after skin exposure. The gas attacks mainly the eyes and the respiratory organs. Sometimes the skin is also affected by direct contact with the gas, and the chemical burn then clinically appears 3–6 h after exposure; initial redness is followed by blisters and ulcers.

Tear gas (o-chlorobenzylidene malonitrile) (CS) dispersed by means of a pyrotechnic mixture can give a bullous dermatitis [53]. CS incapacitant spray can cause chemical burns under special circumstances. The CS is dissolved in methyl isobutyl ketone which may contribute to the injury [41].

Ethylene oxide gas used for sterilization of surgical instruments, textile, and plastic material can remain in these objects for several days if not ventilated well enough [5, 19]. Thus, when hospital personnel handle such objects, there is a possible exposure to ethylene oxide, which is not obvious, and the symptoms, including erythema, edema, and large bullae, may therefore be misdiagnosed as another skin disease.

Accidental skin exposure to chemicals under high pressure, for example hydraulic oil, can result in deep penetration into the skin, where a chemical burn with necrosis can develop.

6.5 Treatment

Rinsing with water is the first-aid treatment; preferably, tepid, running tap water should be used. Irrigation should not be done at high pressure, as the corrosive agent may be splashed onto other parts of the body or on the persons treating the burn. It is important that the treatment starts immediately after exposure and that copious volumes of water be supplied, sometimes for hours. Occasionally, chemical burns are caused by corrosive substances insoluble in water; therefore, a solution of water and soap should frequently be used instead. However, sometimes specific antidotes for certain types of chemical burns are required. Clothes, watches, rings, shoes, etc., can be contaminated with the corrosive agent, so they should be removed.

Theoretically, neutralizing solutions should be an alternative treatment to water after exposure to acids and alkalis [12, 17, 52]. However, neutralization of the corrosive agent with weak acids/bases is not recommended for two reasons: (1) irrigation should not be delayed while waiting for a specific antidote—immediate irrigation provides the best removal of the agent, and (2) neutralization of the corrosive agent may produce an exothermic reaction, and the heat can cause further damage [37].

Heat is generated when strong sulfuric acid and phosphoric acid are exposed to water; hence, a thermal burn can add to the chemical burn. To prevent this, it is important that copious volumes of running water be applied. However, water is contraindicated in extinguishing burning metal fragments of sodium, potassium, and lithium, because a chemical burn can be caused by hydroxides formed when water is added to hot metals. These metals spontaneously ignite when exposed to water. To extinguish the burning metal, sand can be used. The burn should then be covered with cooking or mineral oil to isolate the metal from water. Metal pieces should be mechanically removed. Embedded pieces should be removed surgically. First, though, the area should be irrigated with water to prevent an alkali burn from the hydroxides already formed from the metal and water naturally present in the skin.

Skin exposed to hydrofluoric acid should be carefully irrigated with copious volumes of running tap water, then treated with calcium gluconate gel (2.5%) by massaging into the burned skin for at least 30 min (K-Y Jelly, Johnson & Johnson Products, Inc., New Brunswick, NJ, USA) [3, 9, 13, 25, 38]. The calcium gluconate gel can also be made by mixing 3.5 g calcium gluconate with 150 g of a water-soluble lubricant. A variation of this treatment is suggested—ten 10 g tablets of calcium carbonate (648 mg) are crushed to a fine powder. The powder is mixed with 20 ml of a water-soluble lubricant to create a slurry. This calcium preparation is applied repeatedly to the skin until the pain has disappeared. Necrotic tissue should be excised, blisters debrided, and the underlying tissue treated with the calcium preparation. Nails should be removed if the acid penetrates to the nail bed and matrix and causes severe pain there. If there is no effect of the topical treatment within 2 h, 10% calcium gluconate (0.5 ml/cm^2) should be injected into and under the lesions. No anaesthetics should be given, since the disappearance of pain is a sign of successful treatment. Without treatment, the burn can increase in depth for several weeks.

Superficial chemical burns from *chromic acid* with an area greater than 1% of the total body surface imply a high risk of systemic damage to many organs, including erythrocytes [46]. Therefore, immediate irrigation of the burn with copious volumes of water is necessary. Thereafter, and within 2 h after the exposure, all burnt tissue must be excised. To remove circulating chromium, peritoneal dialysis has to be carried out during the first 24 h.

Solid particles of lime, cement, and phosphorus, for example, tend to fix to the skin and should be mechanically removed before or during irrigation.

Phosphorous, above all white phosphorous, is oxidized by air and can ignite spontaneously, thus causing thermal burns [14–16, 23]. In water, oxidized phosphorous is transformed into phosphoric acid which can cause a chemical burn, therefore, it is important to remove particles mechanically before washing with soap and water. The skin is then washed with 1% copper (II) sulfate in water, which reacts with phosphorous to form black copper phosphide, which makes any remaining phosphorous visible and thus easily removable. Wet dressings of copper sulfate should never be applied to wounds because of the risk of systemic copper poisoning. To minimize the copper absorption, a water solution of 5% sodium bicarbonate and 3% copper sulfate suspended in 1% hydroxyethyl cellulose can be used for irrigation instead of the 1% copper sulfate solution. However, it should be stressed that copper is a potentially toxic substance, which can cause systemic effects. Copper sulfate must therefore be used only for a few minutes in order to visualize phosphorous and, after mechanical removal of the phosphide, it is important to irrigate the skin with water.

Skin contaminated with bromine or iodine should be washed frequently with soap and water and treated with 5% sodium thiosulfate, which reacts with bromine and iodine, forming ions less hazardous to the skin [11, 49].

Skin contaminated with phenolic compounds can initially be washed with soap and water, and as early as possible treated with undiluted polyethylene glycol 300 or 400, or with 10% ethanol, which all dissolve phenolic compounds [21, 28, 39]. Tissues with deep damage from phenolic compounds should be excised immediately, as the compounds easily penetrate further with subsequent damage of, for example, nerves.

Skin contaminated with sulfur mustard liquid should be treated with a mixture of 75% calcium hypochlorite and 25% magnesium sulfate for some minutes before washing with soap and water. Contaminated objects should also be treated with this mixture [33, 36, 40].

Studies in pigs have shown the usefulness of mechanical dermabrasion to accelerate the rate of healing of induced injuries from sulphur mustard vapor [35].

Hot tar, pitch, and asphalt cause burns mainly due to the heat. They stick to the skin and should not be removed mechanically, as the skin can be further damaged and thus increase the risk of secondary infection. The material will fall off spontaneously in due time.

Generally, an antibacterial cream should be given to chemical skin burns to protect the surface and to prevent secondary infection. If there is a significant element of inflammation in nonnecrotic areas, a mild topical corticosteroid preparation can be used. Frequent examinations of primarily superficial and limited burns are also advisable, as they can become deeper in a few days.

Surgical treatments, such as excision, debridation of blisters, transplantation, and removal of nails can be of great value. When a limb is affected circumferentially, there is a risk of blood-vessel compression. The best method for treating the black, adherent necrotic tissue caused by cement and other toxic compounds is excision. For example, the healing time of cement burns on knees can be diminished from 8–10 weeks to 3 weeks if the necrotic tissue is excised.

Several chemicals can also produce systemic effects without severe skin injury, e.g., phenolic compounds, hydrofluoric acid, chromic acid, sulfur mustard, and gasoline [2, 8]. When the chemical burn is not minimal, there is a risk of systemic damage, and an analysis including hematological screening, liver and kidney function, should be made both at the first examination and then later in the course of treatment, depending on the intensity and extension of the chemical burn as well as on the results of laboratory investigations. These analyses are performed mainly to enable precautions and measures necessary to prevent and diminish damage on internal organs, but also partly for legal reasons.

Patients with severe and extensive skin damage and/or with systemic symptoms after exposure to corrosive agents should be treated in intensive care units. It should be noted that hydrofluoric acid or chromic acid exposure affecting only 1% of the total body surface of a person means risk of severe systemic effects. Hospitalization is also recommended for persons who have concurrent illnesses, implying that they are high-risk patients, as well as for persons with chemical burns on the hands, feet, and perineum [2, 8].

6.6 Complications

Chemical skin burns can cause hyper-or hypopigmentation. Chemical burns involving deeper parts of the skin heal with scarring. Tumors of both malignant and benign types may rarely develop in scars. In

the acute stage of chemical burns from, for example, phenolic compounds and hydrofluoric acid/fluorides, the sensory nerve system is frequently affected.

Many contact sensitizers also have irritant properties. Patch testing with such sensitizers at too high concentrations can cause an irritant reaction or a chemical burn, which seems to facilitate active sensitization. However, only a few sensitizers can cause chemical burns without occlusion e.g., formaldehyde, chromic acid, amines, chloroacetophenone, some plastic monomers, and methylisothiazolinones. Even one single contact with these chemicals can both cause a chemical burn and induce sensitization with a subsequent possible development of an allergic contact dermatitis [6, 22] (Table 2). Therefore, when a potential sensitizer has caused a chemical burn, the patient should be patch tested with the sensitizer after healing of the burn, independent of any subsequent development of an eczema.

Table 2. Chemicals which can both cause a chemical burn and induce sensitization after one single skin contact

Epoxy resin system (consisting of epoxy resin and the hardener diaminodiphenylmethan)
Polyfunctional aziridine
Methyl acrylate
Phenol-formaldehyde resin
Methylchloroisothiazolinone/methylisothiazolinone
Omega-chloroacetophenone
o-Chlorobenzylidene malonitrile

Another type of eczematous dermatitis that can follow after a chemical burn is "posttraumatic eczema" [29]. It can present as discoid eczema and is a poorly understood complication of skin injuries [50]. It can appear after either physical or chemical skin injuries, including chemical burns, and is always unrelated to infection and topical treatment.

6.7 Prevention

Employees should be informed of the risks of exposure to corrosive agents and be well trained to handle the chemicals as well as to act when they have been exposed. Showers for rapid irrigation with water should be easily accessible. A 1% copper sulfate solution, polyethylene glycol 300 or 400, 5% sodium thiosulfate solution, and a proper calcium preparation should be present in the first-aid kit. A calcium preparation for topical treatment should also be present near any employees' work site where hydrofluoric acid or fluorides are used. Workers at risk should wear proper protective equipment, which may include eye glasses, face masks, gloves, boots, and safety dresses.

In industries in which corrosive chemicals are handled, certain procedures frequently lead to accidents, resulting in exposure to the chemicals. Such procedures include the repairing as well as charging and discharging of procedure vessels, during which chemicals can be spilt and splashed. Accidents can be caused by breakage of hoses or connections with snap couplings. A nonaccidental but unintended exposure may occur due to material sterilized with ethylene oxide; thus, the material should be well ventilated and not used until a week after the sterilization procedure. For these reasons, it is important to prevent chemical burns via careful planning and supervision of the working environment.

6.8 Summary

Thousands of chemicals and products can cause chemical skin burns, some only under special circumstances, for example occlusion. Most chemical burns are due to accidents and the majority are occupationally induced, but chemical burns also frequently occur in households and as a result of activities related to hobbies. Clinically, a chemical burn is characterized by erythema, blisters, and necrotic skin. Some corrosive chemicals, such as phenolic compounds, sulfur mustard, chromic acid, hydrofluoric acid, and gasoline may cause systemic effects that require hospitalization. Other chemical burns, particularly those affecting hands, feet, and perineum, may also require hospitalization. To prevent and diminish the damage after exposure to corrosive agents, it is important to administer immediate treatment. Irrigation with copious volumes of water is a universal remedy, except for treatment of burning metal fragments of sodium, potassium, and lithium. First-aid treatment after exposure to water-insoluble corrosive agents consists of washing with soap and water. Sometimes specific antidotes are needed, as for chemical burns from hydrofluoric acid, phenolic compounds, phosphorous, iodine, bromine, and sulfur mustard (Table 3). Surgical intervention may be required for certain chemical burns. A few corrosive compounds are potential sensitizers, and one single exposure to such a compound may both cause a chemical burn and induce sensitization with subsequent allergic contact dermatitis.

To prevent chemical burns, it is important to use as few corrosive agents as possible and, when unavoidable, to use the weakest ones possible, particularly in households and while engaged in hobbies. In the working environment, well-informed workers, access to first-aid treatment, careful planning, and supervision are required to prevent chemical burns.

Table 3. Treatment for chemical skin burns caused by some specific chemicals

Chemical	Treatment
Hydrofluoric acid	Calcium gluconate gel (2.5%)
Phosphorous	Copper (II) sulfate in water (1%)
Bromine, iodine	Sodium thiosulfate in water (5%)
Phenolic compounds	Polyethylene glycol 300 or 400
	Ethanol in water (10%)
Sulfur mustard liquid	Mixture of 75% calcium hypochlorite and 25% magnesium sulfate

References

1. Adams RM. Cement burns. In: Adams RM (ed) Occupational skin disease, 2nd edn. WB Saunders, Philadelphia, 1990; pp 15–16
2. Andersen KE. Systemic toxicity from percutaneous absorption of industrial chemicals. In: Adams RM (ed) Occupational skin disease, 2nd edn. WB Saunders, Philadelphia, 1990; pp 73–88
3. Anderson WJ, Anderson JR. Hydrofluoric acid burns of the hand: Mechanism of injury and treatment. J Hand Surg 1988; 13:52–57
4. Beausang E, Herbert K. Burns from a dust explosion. Burns 1994; 20:551–552
5. Biro L, Fisher AA, Price E. Ethylene oxide burns. Arch Dermatol 1974; 110:924–925
6. Bruze M, Dahlquist I, Gruvberger B. Chemical burns and allergic contact dermatitis due to Kathon WT. Am J Cont Derm 1990; 1:91–93
7. Cartotto RC, Peters WJ, Neligan PC, Douglas LG, Beeston J. Chemical burns. Can J Surg 1996; 39:205–211
8. Chan TC, Williams SR, Clark RF. Formic acid skin burns resulting in systemic toxicity. Ann Emerg Med 1995; 26:383–386
9. Chick LR, Borah G. Calcium carbonate gel therapy for hydrofluoric acid burns of the hand. Plast Reconstr Surg 1990; 86:935–940
10. Chockalingam S, Dega R, Goodier D. Skin burn associated with paint on synthetic plaster cast. Injury 1999; 30:301–302
11. Corazza M, Bulciolu G, Spisani L, Virgili A. Chemical burns following irritant contact with povidone-iodine. Contact Dermatitis 1997; 36:115–116
12. Dunn BJ, MacKinnon MA, Knowlden NF, Billmaier DJ, Derelanko MJ, Rusch GM, Naas DJ, Dahlgren RR. Hydrofluoric acid dermal burns. An assessment of treatment efficacy using an experimental pig model. J Occup Med 1992; 34:902–909
13. Dunn BJ, MacKinnon MA, Knowlden NF, Billmaier DJ, Derelanko MJ, Rusch GM, Naas DJ, Dahlgren RR. Topical treatment for hydrofluoric acid dermal burns. Further assessment of efficacy using an experimental pig model. J Occup Environ Med 1996; 38:507–514
14. Eldad A, Simon GA. The phosphorous burn—a preliminary comparative experimental study of various forms of treatment. Burns 1991; 17:198–200
15. Eldad A, Chaouat M, Weinberg A, Neuman A, Ben Meir P, Rotem M, Wexler MR. Phosphorous pentachloride chemical burn—a slowly healing injury. Burns 1992; 18:340–341
16. Eldad A, Wisoki M, Cohen H, Breiterman S, Chaouat M, Wexler MR, Ben-Bassat H. Phosphorous burns: evaluation of various modalities for primary treatment. J Burn Care Rehabil 1995; 16:49–55
17. Erdmann D, Hussmann, Kucan JO. Treatment of a severe alkali burn. Burns 1996; 22:141–146
18. Fisher AA. Chromate dermatitis and cement burns. In: Fisher AA (ed) Contact dermatitis, 3rd edn. Lea & Febiger, Philadelphia, 1986; pp 762–772
19. Fisher AA. Ethylene oxide (EO) burns. In: Adams RM (ed) Occupational skin disease 2nd edn. WB Saunders, Philadelphia, 1990; pp 17–18
20. Gelmetti C, Cecca E. Caustic ulcers caused by calcium hydroxide in 2 adolescent football players. Contact Dermatitis 1992; 27:265–266
21. Horch R, Spilker G, Stark GB. Phenol burns and intoxications. 1994; Burns 20:45–50
22. Kanerva L, Tarvainen K, Pinola A, Leino T, Granlund H, Estlander T, Jolanki R, Forstrom L. A single accidental exposure may result in a chemical burn, primary sensitization and allergic contact dermatitis. Contact Dermatitis 1994; 31:229–235
23. Kaufman T, Ullmann Y, Har-Shai Y. Phosphorus burns: a practical approach to local treatment. J Burn Care Rehabil 1988; 9:474–475
24. Kirkpatrick JJ, Enion DS, Burd DA. Hydrofluoric acid burns: a review. Burns 1995; 21:483–493
25. Kono K, Watanabe T, Dote T, Usuda K, Nishiura H, Tagawa T, Tominaga M, Higuchi Y, Onnda M.. Successful treatments of lung injury and skin burn due to hydrofluoric acid exposure. Int Arch Occup Environ Health 2000; 73:93–97

26. Kuniyuki S, Oonish H. Chemical burn from acetic acid with deep ulceration. Contact Dermatitis 1997; 36:169–170
27. Lane PR, Hogan DJ. Chronic pain and scarring from cement burns. Arch Dermatol 1985; 121:368–369
28. Lin CH, Yang JY. Chemical burn with cresol intoxication and multiple organ failure. Burns 1992; 18:162–166
29. Mathias CGT. Post-traumatic eczema. Dermatologic Clinics 1988; 6:35–42
30. McGeown G. Cement burns of the hands. Contact Dermatitis 1984; 10:246
31. Morley SE, Humzah D, McGregor JC, Gilbert PM. Cement-related burns. Burns 1996; 22:646–647
32. Nahlieli O, Baruchin AM, Levi D, Shapira Y, Yoffe B. Povidone-iodine related burns. Burns 2001; 27:185–188
33. Newman-Taylor AJ, Morris AJR. Experience with mustard gas casualties. Lancet 1991; 337:242
34. Onuba O, Essiet A. Cement burns of the heels. Contact Dermatitis 1986; 14:325–326
35. Rice P, Brown RF, Lam DG, Chilcott RP, Bennett NJ. Dermabrasion—a novel concept in the surgical management of sulphur mustard injuries. Burns 2000; 26:34–40
36. Ruhl CM, Park SJ, Danisa O, et al (1994) A serious skin sulfur mustard burn from an artillery shell. J Emerg Med 12:159–166
37. Sawhney CP, Kaushish R. Acid and alkali burns: considerations in management. Burns 1989; 15:132–134
38. Seyb ST, Noordhoek L, Botens S, Mani MM. A study to determine the efficacy of treatments for hydrofluoric acid burns. J Burn Care Rehabil 1995; 16:253–257
39. Shibata K, Yoshita Y, Matsumoto H. Extensive chemical burns from toluene. Am J Emerg Med 1994; 12:353–355
40. Smith WJ, Dunn MA. Medical defense against blistering chemical warfare agents. Arch Dermatol 1991; 127:1207–1213
41. Southward RD. Cutaneous burns from CS incapacitant spray. Med Sci Law 2001; 41:74–77
42. Spoo J, Elsner P. Cement burns: a review 1960–2000. Contact Dermatitis 2001; 45:68–71
43. Stewart CE. Chemical skin burns. Am Fam Physician 1985; 31:149–157
44. Stoermer D, Wolz G. Cement burns. Contact Dermatitis 1983; 9:421–422
45. Sykes RA, Mani MM, Hiebert JM. Chemical burns: retrospective review. J Burn Care Rehabil 1986; 7:343–347
46. Terrill PJ, Gowar JP (1990) Chromic acid burns; beware, be aggressive, be watchful. Br J Plast Surg 43:699–701
47. Tosti A, Peluso AM, Varotti C. Skin burns due to transit-mixed Portland cement. Contact Dermatitis 1989; 21:58
48. Vance MV. Hydrofluoric acid (HF) burns In: Adams RM (ed) Occupational skin disease, 2nd edn. WB Saunders, Philadelphia, 1990; pp 18–21.
49. White A, Joseet M. Burns from iodine. 1990; Anasthecia 45:75
50. Wilkinson DS. Discoid eczema as a consequence of contact with irritants. Contact Dermatitis 1979; 5:118–199
51. Winemaker M, Douglas L, Peters W. Combination alkali/thermal burns caused by "black liquor" in the pulp and paper industry. Burns 1992; 18:68–70
52. Yano K, Hata Y, Matsuka K, Ito O, Matsuda H. Effects of washing with a neutralizing agent on alkaline skin injuries in an experimental model. Burns 1994; 20:36–39
53. Zekri AM, King WW, Yeung R, Taylor WR. Acute mass burns caused by *o*-chlorobenzylidene malononitrile (CS) tear gas. Burns 1995; 21:586–589