

MANAGEMENT OF CHEMICAL INJURIES TO THE UPPER EXTREMITY

Debra A. Reilly, MD, and Warren L. Garner, MD

Chemical burns are unique injuries that need specialized knowledge and management principles to obtain the best outcome. Because of the ubiquity of various chemicals in common household and industrial use, the products containing chemicals capable of causing a cutaneous injury number more than 25,000.⁹ Contact with these chemicals occurs at work,⁶ accidentally, because of assault, and during acts of war.²⁵ Alkalis are the most common chemical involved in cutaneous burns, but the most frequent single chemical agent involved is sulfuric acid.³⁶

As with thermal injuries, the severity of the chemical injury depends on time and duration of exposure to the noxious agent; specifically, (1) concentration of the agent, (2) duration of exposure, (3) volume of substance contacting the skin, (4) local skin characteristics, and (5) physical properties of the chemical. Duration of contact with the skin is the major determinant of the chemical injury. Ongoing tissue destruction will occur for as long as the chemical remains active and in contact with the skin. Delay in the treatment of an acute chemical contact will both produce a

more severe cutaneous response and may initiate a systemic response. This article deals with both the initial treatment of chemical exposures as well as definitive therapies.

Patients with burns caused by exposure to chemical substances constitute a minority of the admissions to burn centers. Of these admissions, more than 60% are the result of work-related injuries. Many of these injuries involve the upper extremity and specifically the digits. Although the traditional measure of burn severity—percentage body surface area burned (%TBSA)—is low compared with the usual burn population, these injuries have a significant morbidity because the percentage of full-thickness burn is high and the patient's hospital stay is long.²⁵

Treatment of chemical burns can be difficult. Minor injuries can be treated safely and effectively by a surgeon with interest and experience. Serious chemical burns are much more difficult to treat optimally. Both the American College of Surgeons and the American Burn Association recommend that such injuries be referred to a Burn Center specializing in the care of complex wounds.

From the Division of Plastic and Reconstructive Surgery, University of Southern California (DAR, WLG); the Department of Burn Reconstructive Surgery, Rancho Los Amigos National Rehabilitation Center (DAR); and the Los Angeles County and University of Southern California Burn Center (WLG), Los Angeles, California

HAND CLINICS

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MECHANISMS OF ACTION

Acids—Sulfuric Acid, Hydrochloric Acid

Acids are proton donors, which decrease pH from 7 to 0 as hydrogen ions are released. The hydrogen ions from the acid produce amide bond hydrolysis. This causes protein structural collapse. Some acids are exothermic, producing heat as they dissociate, combining their destructive chemical effects with a thermal injury. Necrosis of the soft tissues and bone is the end result. Tissue damage depth is limited by the buffering capacity for acids contained in the damaged tissue.

Alkalis—Ammonia, Lime, Sodium (Caustic Sodium) and Potassium Hydroxide (Potash)

Bases accept protons and strip hydrogen ions from amide bonds of the protein backbone, enabling hydroxylation. This hydroxylation also promotes protein hydrolysis. Further injury may be caused when the bases form salts and soaps, resulting in additive tissue damage. This tissue injury, commonly known as *liquefaction necrosis*, allows for a deeper injury compared with acid burns because the alkaline injury produces a continued basic wound milieu. Because damaged tissue has a limited capacity to buffer alkalis, there is further penetration of the lipophilic alkali, which results in deeper tissue injury. The saponification mechanism is also exothermic, which can contribute to the surrounding tissue destruction.

Reduction Reactions—Hydrochloric Acid, Alkyl Mercury Agents, Nitric Acid

Reducing agents bind free electrons in tissue proteins, which denatures proteins. With this class of chemical, water irrigation produces excess heat. Neutralization with soda lime, liquid skin soap, or magnesium solutions should precede water dilution.

Oxidation Reactions—Chromic Acid, Sodium Hypochlorite (Chlorox), and Potassium Permanganate; Industrial Cleansers

Oxidizing agents denature tissue proteins. In the process they may be oxidized and the end product itself is cytotoxic, causing further tissue damage. Neutralization with milk, egg white, or starch paste is adequate initial treatment prior to copious water lavage.

Corrosives—White Phosphorous, Potassium and Sodium Hydroxides, Phenol (Cresols), Aqueous Ammonia; Pulp and Paper Industries, Industrial Cleaning

Corrosive agents denature tissue proteins. Phenols act as solvents that disrupt the protein structure.

Protoplasmic Poisons—Salt Formers; Tungstic Acid, Tannic Acid, Trichloroacetic Acid, Acetic Acid, Formic Acid Metabolic Competitor or Inhibitor; Hydrofluoric Acid, Oxalic Acid; Industrial Cleaning and Tile Etching

Protoplasmic proteins form salts and directly bind with cellular proteins or bind calcium and other organic ions.

Desiccants—Sulfuric Acid, Muriatic Acid; Food Industry, Farming and Fertilizers

Desiccants dehydrate cells and the process is exothermic. Concentrated solutions of sulfuric acid should first be irrigated with a soap (skin soap) solution to bind the acid. Minimal water irrigation may produce further heat injury so copious water irrigation is necessary.

Vesicants—Mustard Gas, Dimethyl Sulfoxide, Cantharides, Lewisite; War, Pharmaceuticals

Vesicants produce subepidermal blisters and edema that also liberate histamine and serotonin. Ischemic necrosis results.

CARE OF THE ACUTE INJURY

The essential activity in the initial care of a chemically injured patient is to stop the burning process. This demands immediate removal of any gloves, shoes, or clothing contaminated with the chemical, brushing away residual dry chemical, and water irrigation at the scene. Water is the appropriate emergent treatment for most chemical contact spills. Although precise identification of the causative agent is necessary for definitive treatment, this prompt treatment follows the advanced burn life support (ABLS) principles of "stop the burning process." Irrigation is an emergency treatment that should be initiated even before medical assistance is sought, and should be continued for 20 to 30 minutes. Water lavage has as several benefits: (1) diluting the chemical agent away from the victim, thereby decreasing the chemical load to the skin, (2) decreasing the rate of the chemical reaction, (3) decreasing tissue metabolism, and (4) restoring normal skin pH. Failure to perform irrigation at the scene of contamination causes potential significant harm to the victim by increasing the %TBSA exposed to the chemical as well as the depth of the injury.

Irrigation must be continued until the chemical is diluted to concentrations no longer harmful to the skin. Both animal studies⁴⁰ and human experience confirm the essential fact that copious and continuous water irrigation immediately following chemical exposure is critical to limiting the injury process.¹⁷ A delay of only 15 minutes following contact with a chemical may allow the epidermis to be destroyed. The more permeable dermis is then exposed to the deleterious effects of a toxic chemical. By starting the washing process before the tissue pH level reaches an extreme, subsequent pH aberrations are minimal.³⁹ Care must be taken with the drainage water used in the irrigation, as the water used to flush the wound will also contain the chemical agent. Large volumes of water are recommended and continued until the skin pH achieves a neutral 7. Checking the pH of the lavage water also confirms appropriate neutralization of the chemical agent.

Exceptions to the protocol of water irrigation are rare and include injuries from ele-

mental sodium, potassium, and lithium. Exposure of these elements to water causes ignition and additional thermal injury. Mineral oil may be used to first coat the patient and water irrigation used later to remove the particles of chemical imbedded in the skin.

Surface chemical should be irrigated away before there is time for the chemical to penetrate to deeper tissue planes where it is not available to dilution. The duration of irrigation varies with the agent and its concentration. One hundred twenty minutes is reasonable for most acid exposures. Alkalis, however, may need up to 12 hours of irrigation to neutralize the pH.^{17, 38} Skin pH measurements using litmus paper gives a rough guide to neutralization of the surface skin but does not provide an accurate estimate of the pH of the deeper tissues, where the damage may be ongoing. Pain may be a useful guide but is not completely reliable because some of the chemicals (phenols) demyelinate nerves, making the wound anesthetic.¹⁸ Shower irrigation is preferred to bathtub immersion so that contaminated water does not remain in contact with the patient's skin. Delays in presentation and treatment have been shown to increase the depth and severity of the injury. There is also a higher likelihood of the need for tangential excision and split-thickness skin grafting in injuries not treated immediately.^{6, 27, 25}

Estimating the depth of injury is more difficult with chemical burns. Chemical vesicants usually have a delay in their presentation and at first present with soft signs of erythema and blistering. Other chemicals such as corrosives produce liquefaction eschars that are moist on the top. Still others, the protoplasmic poisons, form dry, hard eschars, hiding the underlying necrosis. The thickness of the epidermis and the overall quality of the skin also influence minimal blistering time. Skin problems such as maceration, abrasions and pressure also sensitize the deeper skin to damage.

Histologic sections of chemically injured skin demonstrate a decrease in the collagen and acid mucopolysaccharide content. Up to one third of the dermal collagen is lysed within 8 hours of a significant chemical exposure. The loss of hydroxyproline in acid burns

explains the loss of the epidermis, causing exposure of dermis and leading to full-thickness dermal damage. There is also considerable coagulation necrosis and thrombus formation in the microvasculature in the injured area.

WOUND CARE

Following adequate irrigation of the injured part, blisters, bullae, and necrotic tissue are debrided because they may contain residual concentrations of the chemical and act as a barrier to water irrigation. Daily wound care should be provided until healing for partial-thickness injuries or surgical intervention for full-thickness injuries. If the initial care is provided by a referring physician, it is simpler to cover the burn with a dry gauze dressing until the wound is evaluated by the treating burn specialist. Serial wound evaluations are often needed to determine the healing potential of the injured skin. Tissue biopsies can be used to determine the histologic depth of the injury,¹⁰ but are seldom clinically useful. The early visual clues traditionally used for superficial versus deep second- to third-degree thermal burns are not equivalent for chemical injuries.

A variety of topical agents are now at the disposal of the treating burn physician. In most situations, the practitioner is well served by treating the wound with either once-daily gauze and 1% silver sulfadiazine dressing or an Acticoat (Westaim Biomedical, San Clemente, CA) dressing. The traditional daily Silvadene dressing is safe and effective but sometimes painful and demands personnel time. A new dressing material, Acticoat (a dry silver-coated gauze), is an alternative material that does not require daily changes. This results in less pain and nursing effort. There are many other alternative dressings and topical antimicrobial agents available. Their use should be based on personal experience and individual wound cultures. The use of artificial skin¹⁶ substitutes is to be delayed until the depth of injury is well documented and the wound is free from infection (defined as 10^5 or fewer bacteria per gram of tissue).

As with other forms of burn injury, wounds of first- or second-degree depth, expected to heal within 14 days, need only gentle daily wound cleansing and expectant management. Deeper wounds, of second or third degree, are better served with timely excision and wound closure using split skin, artificial skin, or flap, depending on the clinical situation. Rehabilitation proceeds as indicated by pre-morbid life and work style and postsurgical deficit.

SPECIAL CONCERNS FOR THE UPPER EXTREMITY

The hand and upper extremity are frequently injured in minor chemical spills.³⁶ Although universally recommended when working with toxic chemicals, gloves are sometimes not worn or are used despite tears in the material.¹² Many of the industrial-related accidents involve chemicals used for cleaning equipment, a situation in which the hands are at constant risk for exposure.

In contrast, deliberate assaults with chemicals and warfare-related exposures frequently involve additional body areas, such as the face and torso.²⁵ When the injury spreads onto the arm it is usually not in a uniform pattern of distribution, but appears as streaks from the spill or a run-down effect of the chemical dripping onto the skin. A chemical pattern of this sort should alert the burn team that abuse is the cause²³ of the accident and appropriate questioning should commence to determine the potential to the patient for subsequent danger.

Nail Beds

Persistent chemical contact beneath the nail bed can be difficult to treat because the overlying nail prevents access to the water used for dilution. A simple drainage hole is usually insufficient to adequately remove the agent. The alternatives are complete removal of the nail (for large exposures), removal of a portion of the nail, or incision through the lateral pulp and irrigation through the subcutaneous tissues. Ultimately, this decision is based on

the surgeon's experience with late care of the nail bed. The potential for nailbed deformity following nail removal is balanced with the scarring expected from a partially treated nail-bed burn. Of special concern are hydrofluoric acid exposures. Because the treatment for hydrofluoric acid in the soft tissues is often injection of a liquid, adequate access to the involved nail bed is important.

Finger Pulp

Blisters on the finger pads are best débrided. This serves both to remove any remaining chemical adherent to the skin and to allow better dilution of the deeper tissues for complete removal of any chemical that has penetrated deeper into the tissues. Burns caused by agents such as hydrofluoric acid, which are best treated with injection of a neutralizing agent, have the added problem of causing a compartment syndrome of the digit. Regional blocks are recommended prior to aggressive debridement or the injection of large quantities (0.5 mL/cm²) of neutralizer. The surgeon must be careful, however, to recognize that the pain relief from the anesthetic may eliminate the protective mechanism of pain as a measure of ongoing chemical injury and inadequate early wound care. Lateral incisions of the pulp may be the best approach to obtain access to the deep soft tissues of the fingers.

Fascial Compartments

The fascial compartments of the upper extremity (forearm and carpal tunnel) are at risk for edema formation, as they are with a thermal burn. Because the magnitude of a chemical burn is often underdiagnosed in the early hours following a chemical exposure, compartment syndromes may be missed in their initial stages unless careful surveillance of the neurovascular status of the upper extremity is carried out. Surgical release of the fascial compartments and the carpal tunnel are performed in a standard fashion and treated as with the open burn areas. Secondary delayed primary closure or split-thickness

skin graft of the defect is performed, depending on the patient's condition and the surrounding tissue damage.

SYSTEMIC CONCERNS

If the upper extremity or hand is part of a larger %TBSA injury, the patient is at risk for severe systemic toxicity. In these cases, the total body pH can be altered, resulting in pulmonary and renal damage or damage to the organ of elimination (liver, lung, or kidney) by the byproducts of degradation.^{7, 14, 26} Early and serial testing of arterial blood gases for pH, serum chemistries, serum concentrations of metals,²⁶ and toxic urinary byproducts is essential until these events subside.

LATE SEQUELAE

Because many chemical burns are deep injuries, the outcome of their treatment is not ideal. The inflammation that results from the chemical agent induces significant scar formation and increased morbidity from both poor wound healing and impaired mobility. Skin contour irregularities, pigment discoloration, and contractures are common. Scar management therefore should be instituted immediately after the acute wound healing phase in the compliant patient. Occupational and physical therapy, including modalities for splinting, stretching, desensitization, and compression, are important aspects of the rehabilitation phase of wound care. Surgical reconstruction and releases are performed as indicated by the late results of wound healing.

Contact dermatitis occurs commonly following prolonged exposures to a chemical, as is seen in occupation-related exposures.^{1, 19} Even a single exposure to a high concentration of chemical may be enough to induce an antigenic sensitization. Although not a traditional burn injury, the resultant skin symptoms of urticaria, keratin slough, puritis, and eczema may be similar to postburn symptoms. The risk of cutaneous malignancy is not increased following recovery from a chemical cutaneous burn.

MOST COMMON CHEMICALS AND THEIR INJURY PATTERN

The following is a selection of chemicals most commonly encountered in emergency room injuries (Table 1). It is not meant to be comprehensive and physicians encountering other chemical compounds are encouraged to consult the Materials Safety Data Sheet and local hazardous materials authorities for treatment recommendations. Commercial products have toll-free numbers on their labels to call with questions in the event of an exposure.

Hydrofluoric acid is prevalent in laboratories (photography), industrial processes (glass etching), and household products (rust removers). Exposures usually cover a small surface area but are among the most painful of the chemical injuries. The mechanism of action is bimodal. First the H^+ ion is released, as with an acid burn, and must be neutralized with dilution and necrotic tissues. Then the powerful Fl^- ion is released and binds with cations in the tissues—specifically, calcium and magnesium. Decalcification of bone results if the ion is not neutralized quickly. Tissue damage continues until all of the free fluoride ion is neutralized (Fig. 1).⁸ Treatment therefore is directed at absorption of the negative ions. Calcium and magnesium salts are chosen. The wound initially is irrigated with water and then a topical 2.5% calcium gluconate gel is applied. Adequate treatment is best demonstrated by the cessation of pain. The use of dimethylsulfoxide may enhance penetration³³ of the gel, but its use in human protocols is not standardized.

If symptoms are not alleviated quickly, as is the case with most injuries of 20% to 50% concentrations, sublesional injection of 10% calcium gluconate or magnesium sulfate solutions is next. The recommended volume is 0.5 mL/cm². If this does not alleviate the pain, then careful consideration is given to intra-arterial injection of calcium, as directed by arteriogram anatomy. Two compounds provide emergent care if calcium gel is not available. Mylanta II (Merck Pharmaceutical, Fort Washington, PA) contains enough Mg^{2+} to neutralize small quantities of HFl. Iced Zephiran (Sarofi Winthrop, New York, NY) (benzalkonium chloride) is also effective for small contaminations (<20% concentrations).⁵

Cement burns are traditionally considered alkali burns because the lime (calcium oxide) component of cement has a pH as high as 12.9.^{24, 29} More accurately, the lime mixes with the added water to form complex silicates in an exothermic reaction. The silicates further react with water to form a slaked lime solution with a pH of 10 to 12. People come in contact with this slake during the smoothing and finishing phase of concrete laying. There is usually no initial pain, so the injury remains unrecognized and untreated for hours. The end result usually manifests itself as a full-thickness injury that is very slow to heal.³⁷

White phosphorus burns were frequently encountered during the Vietnam conflict.⁹ Phosphorous is also used in the illegal manufacture of methamphetamines and fireworks.¹³ Explosions (as with grenades) shower the phosphorous particles into the skin. They will then smoke as they are oxidized by air. Treat-

Table 1. COMMON CHEMICALS

Commercial Types	Chemical Compound	Treatment
Batteries	Sulfuric acid, Li^{2+}	Water irrigation
Toilet bowl cleaners	HSO_4 , HCl (muriatic acid)	Water irrigation
Pool cleaners	HCl	Magnesium oxide, soaps
Rust removers	HFl (H^+ / Fl), chromic acid	Water irrigation, calcium/magnesium ($Ca^{2+}Mg^{2+}$) slurry
Petroleum solvents	Organics	Dilute soaps, water irrigation
Bleach	Sodium hypochlorite	Water irrigation
Drain uncloggers, oven cleaners	Lye (sodium hypochlorite), NaOH	Water irrigation
Tile cleaners*	Ammonium chloride (alkali)	Water irrigation
Cement	Lye	Water irrigation

*Brand-name companies make multiple products—some acid, some alkali. It is not enough to have the patients tell you what brand-name chemical they came into contact with, but the exact product name must be known to adequately treat their injury.

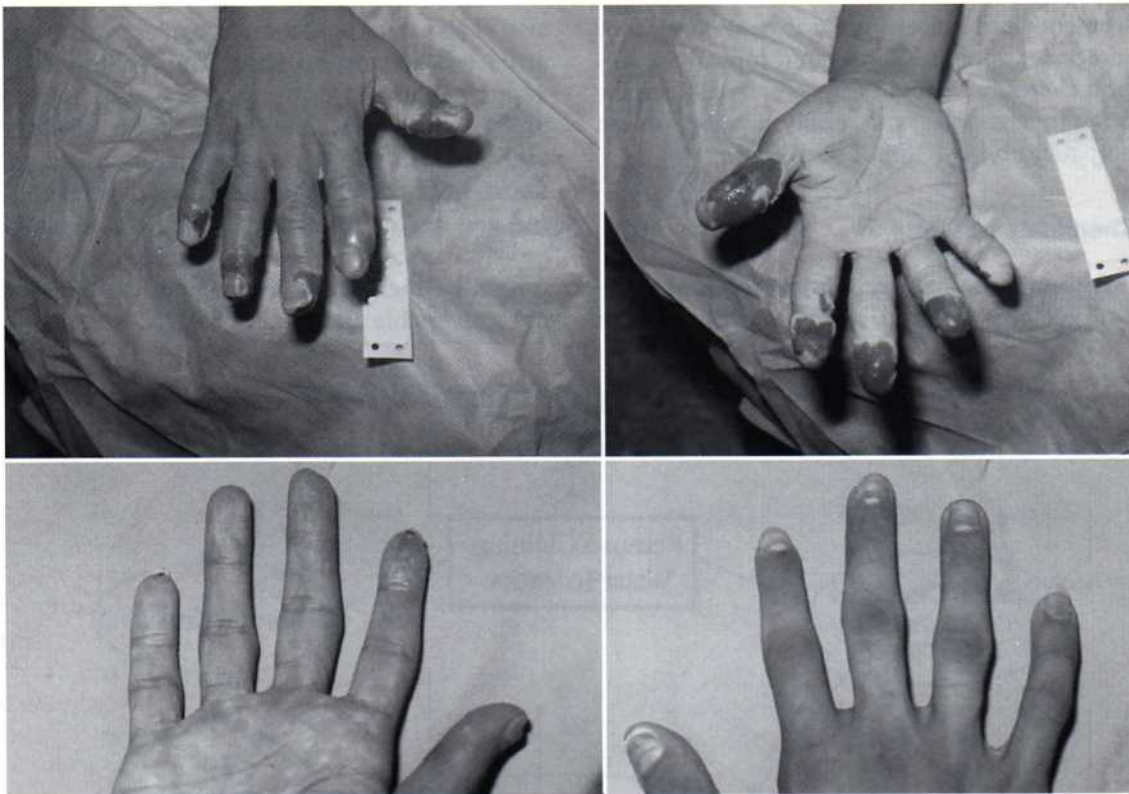


Figure 1. Immediately following hydrofluoric acid exposure (*top*) and 3 months post-injury (*bottom*). Note loss of pulp volume and scarring of index finger after full thickness injury.

ment involves the manual removal of all particles. Irrigation *once* with a dilute 1% to 3% solution of copper sulfate will stain the phosphorus black, making identification and removal easier.²⁰ The patient can then be placed in a water bath and the phosphorous particles removed under water. Contact of the phosphorus with water prior to binding with the copper sulfate (CuSO_4) may cause the phosphorus to ignite in an exothermic process. Immediate treatment decreases the incidence of concomitant renal damage.

Petroleum contact produces another form of poorly recognized skin injury. In addition to the skin irritation and deep dermal slough, the petroleum is absorbed through the skin and has systemic toxicity, particularly to the lungs.

Household cleaners are most commonly composed of multiple chemicals, especially alkalis. When improperly diluted or large spills are encountered (slipping and falling into a bucket of chemical) substantial skin surface area may be injured. Oven cleaners and drain

uncloggers contain strong lye solutions. Disinfectants and bleaches are oxidizers containing high concentrations of chlorine, which coagulates proteins and causes necrosis of the skin.

LESS COMMON CHEMICALS AND THEIR INJURY PATTERN

Air Bag

Drivers and passenger-side air bags are now standard on all United States-sold passenger vehicles. During a sudden deceleration, these safety devices are designed to inflate in 1/20th of a second. Some manufacturers use a nitrogen gas mechanism to facilitate bag inflation. Burns from chemicals on the bag surface, from the heat of the venting of the gas, and from shearing contact with the bag have all been reported. Symptoms are often delayed and include a burning sensation followed by

erythema and blistering. Treatment consists of analgesics, antihistamines, and topical antimicrobial creams. These burns are usually superficial in character and easily treated on an outpatient basis without surgery.³⁵

also at risk of exposure from contact with war artifacts³² and dump sites. This yellow, oily vapor penetrates clothing and causes injury to the skin, eyes, and lungs. After contact with the skin, there is a latent period of more than 8 hours before the skin turns painful and red and begins to blister. Skin that is already moist or macerated is more sensitive to penetration by the gas. Its mechanism of action is the irreversible alkylation of DNA, RNA, and

Chemicals of War

Although lay people tend to think of mustard gas as a chemical of "war," civilians are

Critical Pathway for Chemical Burn

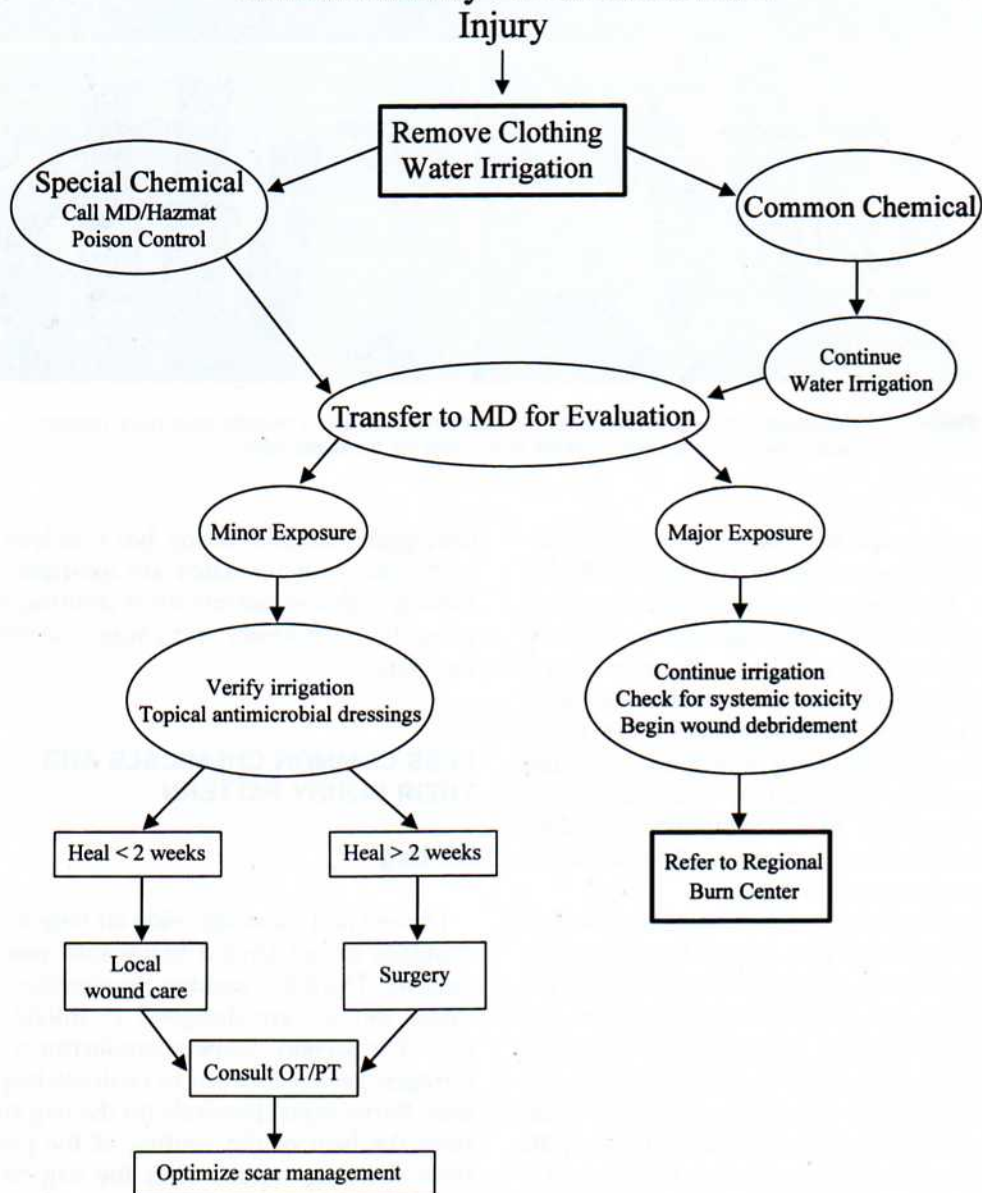


Figure 2. Critical pathway for the treatment of a chemical burn injury.

proteins, leading to cell death. An acute exposure is treated immediately with water irrigation. Where available, special chemical protective clothing is recommended for the medical team. Traditional wound care is initialized, including fluid resuscitation. The skin is split at the sub-epidermal layer (second degree) and usually heals quickly.³⁰ Chronic exposure has a slightly increased incidence of subsequent skin cancer, but a one time exposure probably has no such carcinogenic risk.

HAZARDOUS MATERIAL CONCERNS

All safety and rescue personnel attempting to help a victim of a chemical spill must take great care to protect their own personal environment. This includes early identification of the suspected chemical agent, isolation of any further "spill" by barrier methods, and the wearing of protective gloves, gowns, and goggles. Any tools or paper products used in the neutralization process should be disposed of appropriately. Most major cities provide a local hazardous material telephone number to call for specific questions if the commercial or industrial product labeling is unclear. Material Safety Data Sheets are also available from companies, detailing the specific components of chemical compounds.

SUMMARY

Chemical burns are interesting and challenging to treat. When the practitioner is comfortable with the pathophysiology of chemical injury and treatment based on these principles, most patients can be treated effectively with good outcome (Fig. 2). Early treatment with water irrigation, followed by diligent wound care, will allow our patients to benefit to the maximum from our medical skills.

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Address reprint requests to

Debra A. Reilly, MD
1450 San Pablo Street, Suite 2000
Los Angeles, CA 90033