

CHEMICAL BURNS

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INTRODUCTION

Relative to the number of patients presenting with thermal burns, chemical burns account for only a small portion of the total burn population.¹ There are, however, over 25,000 known chemicals that are capable of causing tissue damage. Burns from caustic chemicals are unlike thermal injuries in that the destruction of tissues continues well after initial exposure. Along with the possibility of cutaneous injury, a significant amount of chemical absorption can also cause systemic toxicity.² This combined with the difficulty in determining the depth of the injury can make the management of these burns truly challenging. This paper will discuss some of the more common classes of chemicals that are known to “burn” as well as cover the treatments that should be considered when dealing with these traumatic injuries.

The number of patients requiring professional medical treatment secondary to chemical burns is estimated to be in excess of 60,000 annually in the United States. The recent literature has shown that the majority of these injuries occur in younger males at their place of employment. This gender predominance can be attributed to the higher percentage of male employees at the more labor intensive, industrial jobs. A study by Singer et al. showed 83% of these burns occurred while on the job, with 94% of these injuries occurring in men between the ages of 20-50 years old.³ The aforementioned study by Herbert et al. also tabulated the percentage of distribution involving various body sites. Around 70% of these injuries involved less than 5% of total body surface with the face and trunk accounting for the highest percentage of exposure (33%). Feet accounted for only 18% of involved burns. The author postulated that this number is low because of the protective shoe gear worn by most industrial workers

PATHOPHYSIOLOGY

Chemical agents generally do not “burn” tissues as seen with thermal injuries. The majority of them tend to coagulate protein by different reactions. The extent of damage secondary to exposure is dependent on 1) strength

or concentration of the agent, 2) quantity, 3) manner and duration of skin contact, 4) extent of penetration into tissue, and 5) mechanism of action (1). What differentiates chemical and thermal burns is the length of time that tissue damage continues. Burns from heat have a duration of insult ranging from seconds to minutes, while chemical injuries display a wide variability from one agent to the next, depending on the different factors mentioned in this section.⁵ Chemicals tend to continue causing destruction until the reaction is inactivated by a neutralizing agent or by serial dilutions with water. This obviates the prompt treatment with copious wound lavage when encountering such injuries.

GENERAL TREATMENT

Early, serial irrigation with water is the appropriate first aid therapy for almost all chemical burns. Contaminated clothing may provide a reservoir for the harmful substance and should be removed, with care being taken to not further expose those assisting the patient. Minimizing the time between contact with the chemical and irrigation should be the highest priority. Time spent searching for neutralizing agents may lead to deeper and more destructive burns. Injury sites should also be covered with wet compresses while patient is transported to the hospital.⁴ A modest number of chemical agents may cause systemic toxicity. Hypocalcemia has been reported from such chemicals as hydrofluoric and oxalic acids. These injuries usually deal with inhalation of a specific chemical or cutaneous exposure in very high quantities, and are less applicable for the podiatric physician.

Upon arrival at the emergency department, the patient should be given some form of analgesia, which should be aided by continued water lavage in relieving symptoms of pain. The name and concentration of the offending chemical should be obtained early in the history taking process. If possible, this should be confirmed by viewing the label or container used to store this product. Litmus paper is another helpful item that can assist in determining whether the substance is an acid or alkali. Mild cleansing of the burn may be

necessary. Care should be taken to not use alcohol cleansers and to avoid scrubbing the affected area. Covering of the burn should be accomplished with a non-adhesive dressing over an antibiotic ointment, with wound evaluation being performed every 2 days. Analgesia may be prescribed as warranted. Prophylaxis with antibiotics is usually not necessary. The use of aseptic technique and topical antibiotics with dressing changes should be adequate in preventing infection for the majority of these burns, unless the wound was grossly contaminated at time of initial injury. If, however, the signs and symptoms of infection become evident, the appropriate antibiotics should be administered.

During the early phases of treatment the depth of the burn is difficult to determine. In contrast to thermal injuries, chemical burns may appear deceptively superficial with only mild discoloration of the remaining skin. Over the next couple of days the depth of the damage will become evident through cracks seen in the darkened outer tissue.¹ It may become necessary to surgically debride the burn site if the eschar doesn't separate, or if an underlying infection begins. Depending on the depth of the injury, a split thickness skin graft may also be warranted for burn site coverage once the wound has a healthy granular base.

TREATMENT ACCORDING TO CHEMICAL CLASS

Treatment for burns of individual chemicals can be organized according to their class (acid, alkalis, etc) or by their specific chemical activity.⁶ This section will highlight the treatments for the more common chemical burns according to their class. (See Table 1)

Alkalis are found frequently in washing powders, drain-type cleaners, wet cement, and some paint removers. They include sodium, potassium, calcium, ammonium, barium, and lithium hydroxides, and their burns have similar clinical presentations to sodium hypochlorite (bleach) and caustic soda. These chemicals will tend to penetrate deeper than acids becoming more destructive and causing a "liquification necrosis" of the involved tissues.⁶ Along with copious water irrigation, a weak acid such as vinegar or orange juice may be used to neutralize the alkalis. One of the more common chemical burns encountered by podiatric physicians is caused by exposure to the caustic component of cement known as calcium oxide (lime). When mixed with water it forms calcium hydroxide. While not initially painful with exposure, over time this chemical penetrates deeper into tissues and becomes quite painful and difficult to dilute.

Table 1

EARLY TREATMENTS FOR CHEMICAL BURNS

- I. Water Lavage
 - Lyes
 - NaOH
 - KOH
 - LiOH
 - NH₄OH
 - Sodium hypochlorite (bleach)
 - Calcium hydroxide (wet cement)
 - Phenol
 - Acids
 - Chromic
 - Hydrofluoric
 - Formic
 - Acetic
 - Tannic
 - Picric
 - Sulfosalicylic
 - Cresylic
 - Tungstic
 - Trichloroacetic
 - DMSO
 - Cantharides
 - Dichromate Salts
 - Potassium permanganate
- II. Avoid Water
 - Sodium and Potassium metals
 - Hydrochloric acid
 - Sulfuric acid
- III. Cover with Oil
 - Phenol
 - Sodium and Potassium metals
 - White phosphorus
- IV. Avoid Oils
 - Cantharides
- V. Administration of Calcium Salts
 - Hydrofluoric acid
 - Oxalic acid
- VI. Chemical Specific Treatments
 - Lyes - weak acid lavage (vinegar, orange juice, etc)
 - Phenol - avoid alcohol, dilute with polyethylene glycol, cover with oil
 - HCl and Sulfuric acids - soap, soda lime, and magnesium hydroxide washes
 - Na or K Metals - Excision of particles followed with oil covering
 - Hydrofluoric acid - Local infiltration of calcium gluconate after lavage
 - Alkyl mercury agents - Debride blister and remove fluid

* References^{1,2,4,6}

Metallic sodium and potassium are probably the only chemicals where water irrigation is completely contraindicated. Water mixed with these substances may cause them to ignite leaving a highly caustic residue.⁴ Oil should be used instead of water following removal of particles from the burn sites.

Acids, with the exceptions of hydrofluoric, chromic, and formic acids, have a tendency to not penetrate and burn as deep as the alkalis.⁴ This diminishes the need for the extensive water lavage that is used for some of the alkalis. Hydrofluoric acid is a highly toxic, deeply penetrating chemical that tends to require more extensive treatment. Following initial water lavage, calcium gluconate is either applied topically or injected subcutaneously around the site of the burn. This prevents further damage caused by fluoride ions in the deep tissues. Hydrochloric and sulfuric acids should be treated with direct neutralization using soda lime, soap, or magnesium hydroxide. Water can produce heat and further damage through ionization of the acid and should be avoided.

Organic compounds such as gasoline, paint thinner, triethylene glycol, and phenol should all be treated with copious water lavage. Phenol has a high lipid solubility making it more advantageous to dilute in polyethylene glycol. Water mixed with polyethylene glycol at a 1:1 ratio is the ideal solution for dilution of this chemical. Alcohols should be avoided for use in treatment of these burns. Since phenol is an alcohol itself, it would merely spread the substance over a greater surface area.⁶

COMPLICATIONS

The complication most frequently seen with chemical burns is contracture of joints with resultant loss of function. Other sequelae can include infection, adhesions, hypertrophic scars, pigment changes, chronic ulcerations, and squamous cell carcinoma.^{1,4,7}

CASE PRESENTATION (FIGURE 1)

A 59 year-old male presented to the Emergency Department with a chemical burn on his left foot. The patient related spilling a chemical used for high-pressure washing of stones on his left shoe. This substance soaked through the top of his shoe and was left in contact with the dorsum of his foot for around two hours. At this time the patient started noticing a burning and irritation where the chemical had soaked through his shoe and sock. He then went to the ER where he was treated with water lavage, silvadene cream, and a dry sterile dressing.

The substance was noted to have both potassium and sodium hydroxide as part of its chemical mixture. The patient was seen 2 days later and was placed on an oral antibiotic for prophylaxis. Throughout the following week the extremity had increased pain, warmth, and redness with the patient also relating increasing fever, chills and a low-grade fever in this same time frame. At his next visit he was diagnosed with ascending cellulites of the left leg and was taken to the OR for debridement of the eschar to rule out any frank infection at the site of the burn. Debridement revealed partial thickness burn to the level of the extensor tendons. The majority of the neurovascular structures were left intact as well all tendinous structures. There was significant serous fluid build up below the eschar, but no purulence was appreciated. The wound was copiously irrigated with normal saline solution following intra-operative cultures and sensitivities being taken of the serous fluid. A non-adhesive, compressive dressing was then applied to the foot. Patient was started on IV antibiotics and admitted to the hospital. Multiple debridements were done over the next week in preparation for a split thickness skin graft. Cellulitis responded to antibiotics and receded. A vacuum-assisted wound device was utilized to build a healthy granular base for the skin graft. Plastic surgery was consulted and completed a split thickness skin graft approximately 2 weeks after initial injury. The patient went on to a full recovery with the only sequelae being decreased dorsiflexion of all 5 digits of the affected foot.

CONCLUSION

Relative to the number of patients presenting with thermal burns, chemical burns account for only a small portion of the total burn population. There are, however, over twenty-five thousand known chemicals that are capable of causing tissue damage. This combined with the difficulty in determining the depth of the burn can make the management of these burns truly challenging. Initial care for all chemical burns should include removal of all affected clothing and copious water lavage (with the exception of the sodium metals, hydrochloric and sulfuric acids). Alkali injuries usually warrant prolonged water lavage. Phenol, hydrofluoric, chromic, and oxalic acids all require more extensive treatment as reviewed above. Continued treatment of chemical burns is identical to that of the more common thermal injuries. This includes debridement of necrotic tissue, non-adhesive dressings, and split thickness skin grafts if deemed necessary.



Figure 1A. Sodium hydroxide chemical burn 4 days after initial injury.

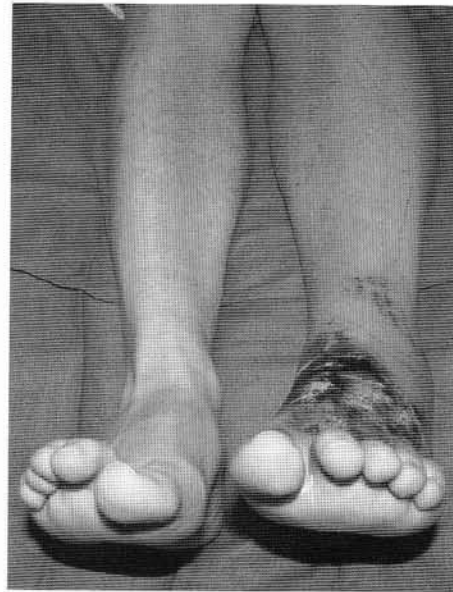


Figure 1B. Edema & erythema of left leg secondary to ascending cellulitis.

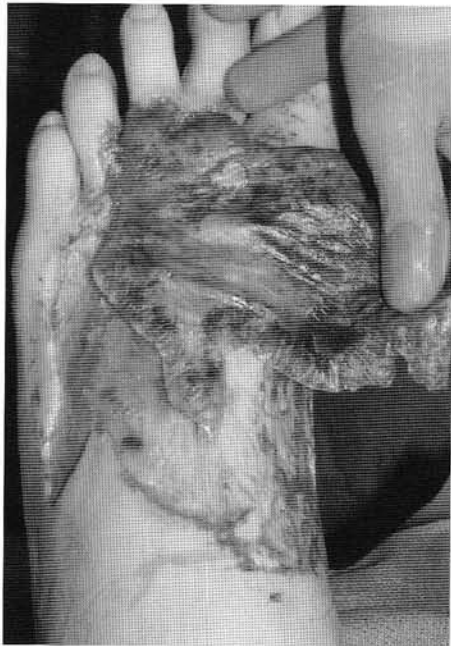


Figure 1C. Intra-operative debridement.



Figure 1D. Removal of eschar. Extensor tendons and majority of neurovascular structures are left intact.



Figure 1E. Copious lavage with normal saline solution.



Figure 1F. Wound following initial debridement.



Figure 1G. Non-adhesive compressive dressing applied.



Figure 1H. Post-operative day 1 dressing change.



Figure 1I. Vacuum-assisted wound device applied to stimulate granulation tissue formation.

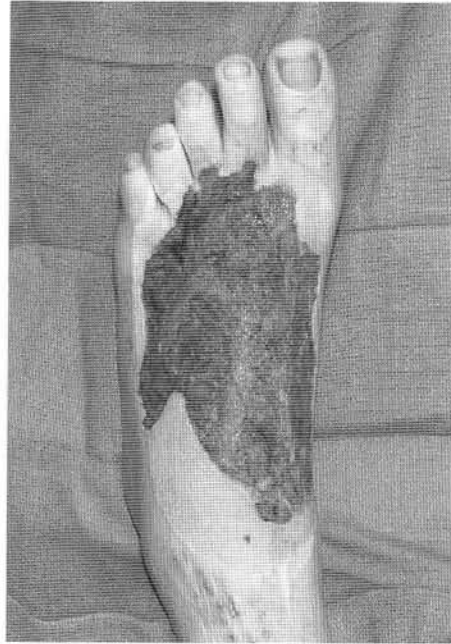


Figure 1J. Wound has healthy granular base in preparation for split thickness skin graft approximately 2 weeks following initial injury.



Figure 1K. Split thickness skin graft is placed on recipient bed.



Figure 1L. Post-operative day 5 with healthy appearance of skin graft.



Figure 1M. 3 weeks following skin graft with continued healing.



Figure 1N. 3 months following initial injury. Graft has healed.

REFERENCES

1. Curreri WP, et al. The treatment of chemical burns: specialized diagnostic, therapeutic, and prognostic considerations. *J of Trauma* 1970 10,8: 634-42.
2. Mozingo DW, et al. Chemical Burns. *J of Trauma* 1988 28, 5: 642-7.
3. Singer A et al. Chemical burns: our 10-year experience. *Burns* 1992. 18, 3: 250-2.
4. Herbert K, Lawrence JC. Chemical Burns. *Burns* 1989 15, 6: 381-4.
5. Leonard LG et al. Chemical burns: effect of prompt first aid. *J of Trauma* 1982 22, 5: 420-3.
6. Jelenko C. Chemicals that "burn". *J of Trauma* 1974 14, 1: 65-72
7. Wallace GF. Nonosseous Injuries. *McGlamry's comprehensive textbook of foot and ankle surgery*. In Banks A et al. 3rd edition vol 2. 2001. 1645-6.
8. Henry JA et al. Intravenous regional calcium gluconate perfusion for hydrofluoric acid burns. *Clinical Toxicology* 1992 30, 2: 203-7.
9. Huisman LC et al. An atypical chemical burn. *Lancet* 2001 358: 1510.
10. Sawhney CP, Kaushish R. Acid and alkali burns: considerations in management. *Burns* 1989 15,2: 132-4.
11. Donoghue JM, et al. Caustic soda burns to the extremities: Difficulties in management. *BJCP* 1996 50,2: 108-10.
12. Hodgkinson DJ et al. Chemical burns and skin preparation solutions. *Surgery, Gyn, and OB Oct* 1978, 147: 534-6.