

# Hydrofluoric Acid Burns of the Lower Extremity

Brian B. Carpenter, DPM,<sup>1</sup> Francis G. Wolfort, MD,<sup>2,3</sup> Stephen P. Tubridy, DPM,<sup>1,4</sup> and Jeffrey H. Miller, DPM,<sup>5</sup>

*Chemical burns to the lower extremity can be disabling and of serious consequence if not managed properly. The severity and rapid onset of the burns caused by hydrofluoric acid after initial contact make this a highly dangerous substance. The potential severity of injury and the following complications make it a chemical of which all physicians should have a basic understanding. (The Journal of Foot & Ankle Surgery 38(5):366–369, 1999)*

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Hydrofluoric acid is a colorless, highly corrosive material used extensively in industry (e.g., in the manufacture of ceramics, graphite, electronics, refineries, biochemical research, pickling of metals, cleansing of brick) and in the home (1, 2). Chemical agents induce deep wounds proportional to concentration and duration of exposure (3). Unlike thermal and electrical burns, damage to the skin by chemicals will continue to cause tissue necrosis even after they are removed. In a hydrofluoric acid burn, the fluoride ion is the agent responsible for prolonged soft-tissue necrosis. In biologic tissues, hydrofluoric acid dissociates into a hydrogen ion and the free fluoride ion (4). In order to prevent this progression, the chemical must be deactivated by dilution or transformed to a nonirritant molecule through a chemical reaction. The fluoride ion is soluble and able to move through soft tissues unopposed; neutralization is the key to halting its progression. This is achieved by a chemical reaction with either magnesium or calcium into an insoluble ion that precipitates within the tissues and then is inactive.

Pain associated with hydrofluoric acid burns is thought to be initiated by the binding of calcium (5). With the binding of cellular calcium, cell death and necrosis will follow. Some theorize that the fluoride ion immobilizes calcium, resulting in stimulation of nerves by a relative excess of potassium (6). This ionic shift is thought to be

the causative factor for the tremendous pain accompanying hydrofluoric acid burns. If not fully bound in soft tissues, the acid can penetrate to the level of bone. The binding of fluoride results in a depletion of free calcium at such a rapid rate, that in some instances, the body cannot mobilize enough calcium from bone in order to avoid hypocalcemia (5). Burns sustained from hydrofluoric acid with as small as 2.5% total body surface area affected can result in hypocalcemia with serious side effects within as little as 2–3 hours (5). High concentrations of hydrofluoric acid may cause immediate burning sensations, while contact with weaker strengths may take hours to become apparent (7).

## Treatment

Initial treatment should include immediate removal of chemically saturated garments. Free chemical should be removed by prolonged copious irrigation of the area with saline. Neutralization prior to irrigation could cause an exothermic reaction leading to extensive tissue destruction. Some advocate that neutralizing agents such as sodium bicarbonate solution be applied to the burn; however, saline irrigation should not be delayed in order to search for neutralizers (8, 9).

After irrigation, the affected areas should be covered with a 2.5% calcium gluconate gel to precipitate the fluoride as calcium fluoride (7, 10). Calcium gluconate gel is not readily available in most smaller hospital pharmacies but can be formulated by mixing 3.5 g of calcium gluconate powder USP to 150 ml of a water-soluble lubricant. Since hydrofluoric acid may continue to penetrate beneath the surface until neutralized by the free calcium in bone, treatment consists of infiltration of affected tissues with a 10% solution of calcium gluconate (8, 10, 11). These injections may require local anesthesia due to the

From <sup>1</sup>The Cambridge Hospital, Cambridge, MA and <sup>2</sup>Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA. Address correspondence to: Brian B. Carpenter, DPM, P.O. Box 238, Bridgeport, TX 76426.

<sup>3</sup>Chief of Plastic and Reconstructive Surgery.

<sup>4</sup>Director of Residency Training.

<sup>5</sup>Submitted while resident.

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amount of volume of fluid necessary and multiple injection sites necessary to infiltrate the entire area. If a local anesthetic is used, it should be the shortest acting available. This is due to the need for patient input on pain symptoms as an indicator for complete neutralization. Infiltration of calcium gluconate beneath the affected areas should extend to a margin of approximately 0.5 cm around the injured tissues (8). Early intervention with calcium gluconate has been shown to significantly shorten healing time and arrest the progressive tissue destruction (12).

In no instance should hydrofluoric acid burns be treated with analgesics since this would mask the need for neutralization treatment (8). In those patients with continued pain, the injection of calcium gluconate into the tissues should be repeated (8, 13). In addition, supplemental calcium could be delivered to the burn site by intra-arterial infusion (14–16). However, intra-arterial infusion of calcium is an invasive procedure that may have complications. One must be aware of thrombosis, arterial spasm, and monitor serum calcium. If continued severe pain occurs after several hours, a repeat application of calcium gluconate gel, calcium gluconate tissue perfusion, or intra-arterial calcium infusion should be repeated.

Following irrigation and neutralization, the burn should be cleansed and dried. A topical antibiotic is then applied. The one most commonly used in the emergency room is silver sulfadiazine. Other topical applications, such as bacitracin, sulfamylon, 5% silver nitrate solution, gentamycin cream, mupirocin ointment, and povidone-iodine ointment, are acceptable (17–19). The burn should be covered with a nonadherent dressing followed by a bulky dry sterile dressing and splinting. If the burn is on an extremity, the affected extremity should be elevated to decrease edema. Prophylactic antibiotics are not usually required, but an antibiotic such as penicillin or first-generation cephalosporin should be used to cover normal skin flora if prophylaxis is desired. Initially, the most common infecting organisms are *Staphylococcus aureus* and beta-hemolytic *Streptococcus*; Gram-bacilli (*Pseudomonas aeruginosa* and *P. vulgaris*) are detectable 3–5 days later (17). The recommended systemic antibiotics for surgical wound manipulation prophylaxis, after initial injury, are intravenous vancomycin and amikacin (20). As with all burns, tetanus immunization status should be evaluated and appropriate prophylaxis administered (21).

Full-thickness chemical burns should be treated as previously described. After a 24–48-hour demarcation period, an escharectomy and debridement can be performed. If infection is not present, the wound is covered with a skin graft, local flap, or free flap (9, 22). In order to determine the best treatment regimen, the surgeon must estimate the depth of the burn and the time required for healing. Burns involving the superficial dermis heal within 3 weeks, generally without hypertrophic scar formation

(23). Second-degree burns should heal within several weeks. However, if there are no signs of healing in 2 weeks, consider debridement and split-thickness skin grafts (23, 24). Prolonged healing by secondary intention results in a hypertrophic scar covered by unstable epithelium and/or contracture of the burn wound, especially in chemical burns (23, 24). This process ultimately affects satisfactory coverage. In full-thickness burns, proceed directly to wound coverage with skin grafts (24). A full-thickness burn is characterized by skin that is white, leathery, and without normal epicritic sensation. Remove all nonviable tissue, control infection, and establish a recipient bed with sufficient vascularity to support the graft. Debride the burn wound mechanically or enzymatically, avoiding exposure of bare tendons or bones, as this may produce a wound that will not support a skin graft. The debridement of all necrotic tissue is essential in providing an optimal bed (25). Quantitative bacterial cultures of less than  $10^5$  bacteria/ml are usually ideal for grafting. Grafts are viable on the paratenon, perineurium, or periosteum. An intermediate-thickness split-thickness skin graft of 0.014"–0.016" is preferred. It possesses some of the qualities of a thin skin graft (excellent host tissue incorporation) and some of a thick skin graft (good durability and less contraction). Skin grafting permits earlier ambulation and hospital discharge for many patients with hydrofluoric acid burns of the lower extremity. The advent of free and fascial flaps offers new hope to those patients with exposed bone, since it is a composite pedicle flap and more trauma resistant.

### Case 1

A construction worker, age 29, sustained burns to his left lower leg and ankle as a result of accidental exposure to 20% hydrofluoric acid while pressure washing a building under renovation. The burned area was immediately washed with water at the work site and he returned to work. Upon completion of the work day, he proceeded home, cleansed the leg, and applied a topical antibiotic ointment. The next morning, upon arising, he noticed the area of the burn was red and was more painful than the previous day with a blister on the outside of his ankle. He proceeded to work where he completed the day's duties even though the pain in his leg was becoming more intense. After work, he again inspected the burn site and determined that it looked worse. He sought medical attention.

The patient arrived at the Emergency Department approximately 30 hours after exposure to the acid. The pertinent findings on admission were limited to the skin, which showed epidermal slough of a 3-cm-diameter area surrounded by erythema and multiple small vesicles on the leg. In some areas, the vesicles were ruptured, revealing a necrotic appearing base. A 3-cm-diameter vesicle on



**FIGURE 1** A 3-cm vesicle on the anterior lateral aspect of the ankle approximately 30 hours after acid exposure (case 1).

the anterior lateral aspect of the ankle also was present (Fig. 1). Findings from all routine laboratory tests were within normal limits with the exception of a WBC count of 11,500/mm<sup>3</sup>.

The burned area was irrigated with copious amounts of sterile saline. Without anesthesia, 20 cc of a 10% solution of calcium gluconate was injected via a 30-gauge needle into the subcutaneous tissues at the burn site. This was followed by debridement of nonviable tissue and a topical application of calcium gluconate in a water-soluble gel covered by a nonadherent dressing. Within 10 minutes of treatment, the patient related cessation of the burning pain. The patient was admitted for intravenous antibiotic therapy, elevation of the extremity, and dressing changes for 24 hours. Upon discharge, the patient performed dressing changes twice a day with silver sulfadiazine and continued with an oral antibiotic. The burned area healed satisfactorily in the course of the next 14 days without complication.

## Case 2

A worker at a micro brewery, age 36, sustained burns to hands and to his left anterior and lateral ankle as a result of a spill of 50% hydrofluoric acid while cleansing a holding tank. The patient was unsure, but believed that some of the acid might have come into contact with his eyes. The exposed areas were immediately washed with water and wrapped in gauze before he went to the Emergency Department.

The pertinent findings on physical examination were mild erythematous conjunctivitis, mild erythema of the palmar aspect of both hands without vesicle formation but with a sensation of burning, and a 6 cm × 7 cm burn to left lateral and anterior ankle (Fig. 2). The affected area of the left ankle was accompanied by intense pain and tough



**FIGURE 2** Photograph of the left lateral and anterior ankle of case 2 demonstrating the coagulated skin.

coagulated skin. Findings from all routine laboratory tests were within normal limits with the exception of serum Mg at 1.6 mg/dl.

All exposed areas were irrigated with copious amounts of sterile saline. A topical application of calcium gluconate gel with dressings was applied to the exposed areas of the hands which alleviated the burning sensation. A subeschar injection of 25 cc of 10% calcium gluconate to the burn site of the left ankle was performed. Fifteen minutes following injection the severe pain was relieved. All necrotic tissue was debrided and a topical application of calcium gluconate gel and a nonadherent dressing was applied. The patient was admitted for monitoring of his electrolytes, which subsequently resolved without intervention and an ophthalmology consult. There was no tissue loss on either hand and the burned area of the left ankle healed without complication, with silver sulfadiazine dressing changes twice daily.

## Conclusion

Careful attention to detail and recognition of the nature of hydrofluoric acid burns and their treatment will decrease the morbidity and mortality associated with this chemical. With the advent of free flaps and fascial flaps, even those patients with severe full-thickness burns have a good prognosis. If appropriate and early treatment is provided, it is very effective.

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