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Hydrofluoric acid burns

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Abstract

Objective: The purpose of this article is to report our experience with hydrofluoric acid (HF) burns and to present our management guidelines for these burns, which include a novel way of delivering calcium combined with dimethyl sulphoxide (DMSO) for cutaneous burns. *Method:* We reviewed our institutional experience from 1977 to 1999 for patients presenting with burns caused by hydrofluoric acid and collected data on age, sex, burn size, anatomical site, method of contact, surgical procedure, and outcome. *Results:* Of a total of 2310 admissions, 42 HF burns patients were identified during the study period. The average age was 34 years. There were 35 males and 7 females. Seventy-four percent of cases received burns to the upper limb. Median burn size was 1% of the total body surface area. Seventeen percent of patients required a surgical procedure. In 24% of cases, the method of contact was work related and 40% were injured using cleaning products at home or on boats. No deaths were recorded. *Conclusion:* HF injury is uncommon but problematic burns often requiring surgery. *Recommended management:* In cases of cutaneous exposure, treatment should commence immediately with 30 min lavage followed by application dimethyl sulphoxide 50% + calcium gluconate 10% in surgical jelly. If hand or forearm is affected, regional intravenous calcium 'Bier's block' using 40 ml 10% calcium gluconate with 5000 U heparin in total final volume of 40 ml may be indicated. Subcutaneous infiltration may be indicated for elsewhere at 0.5 ml/cm^2 burn of 10% calcium gluconate. Persisting pain may require nail removal or arterial calcium infusion.

Keywords: Hydrofluoric acid; Calcium gluconate; Dimethyl sulphoxide; Burns

1. Introduction

Burns due to hydrofluoric acid (HF) are uncommon, however, when they occur they are severe and deaths are not unknown.

Hydrofluoric acid is one of the strongest inorganic acids. It has several uses in both industrial and domestic settings. It was traditionally used in polishing and etching glass [1]. More recently, it has been used as a primary component of rust removing agents, it is also found in some laundry detergents and heavy-duty cleansers. It is used in plastic and dye industries [2], and for the control of the fermentation process in breweries [3]. It is also used as a catalyst in the production of petroleum, and in the laboratory as an agent for mineral analysis and in the production of various metal products and fluoride containing compounds and semi-conductor preparation [4].

The purpose of this article is to report our experience with hydrofluoric acid burns and to present our management guidelines for these burns, which include a novel way of delivering calcium—combined with dimethyl sulphoxide (DMSO) for cutaneous burns.

2. Methods

The Burn Unit database was interrogated for patients presenting with burns caused by hydrofluoric acid between 1977 and 1999. Data collected included age, sex, burn size, anatomical site, method of contact, surgical procedure, and outcome.

3. Results

Forty-two patients were identified to have received burns from hydrofluoric acid during the study period of a total of

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Table 1
Distribution of burn size

Burn size (%TBSA)	*n*
0–l	36
2–5	4
6–10	2
Total	42

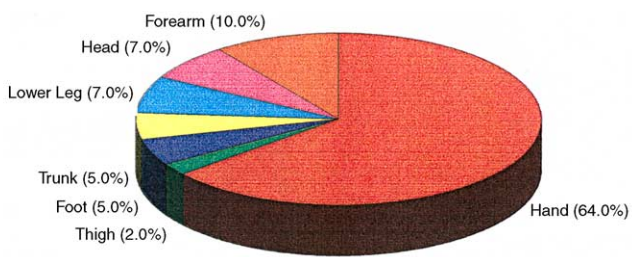

Fig. 1. Anatomic site injured by hydrofluoric acid.

2310 patients registered on the database. The average age of the patients was 34 years. There were 35 males and 7 females. Median burn size was 1% total body surface area, range 0.5–10% (Table 1). The upper limb was involved most commonly. with the fingers and hand comprising 64% of cases, and the forearm comprising 10% of cases (Fig. 1). The lower limb was involved in 14% of cases. while the head and trunk was involved in 7 and 5% of cases, respectively (Fig. 1). Seventeen percent of patients required a surgical procedure involving tangential debridement of burnt areas and skin grafting. Fourteen percent of patients required removal of fingernails. The concentration of hydrofluoric acid in individual cases varied from 1 to 98% with 38.5% of cases involving solutions with a concentration of $\leq$1% hydrofluoric acid. There were six cases in which the concentration of hydrofluoric acid was $\geq$50% and in all of these cases the method of contact was work related involving refinery workers. In 24% of cases the method of contact was work related. Forty percent of patients were injured using cleaning products at home or on boats. There were 11 recorded cases of hypocalcaemia, which were more common in those patients with larger %TBSA burns involving solutions with higher %HFA. Hypocalcaemia was treated with both oral and intravenous calcium supplementation. There were no deaths from hydrofluoric acid burns during this time.

4. Discussion

HF is a highly corrosive agent. It causes tissue damage by two distinct mechanisms. Firstly, the high concentration of hydrogen ions produces a corrosive burn similar to that caused by hydrochloric and sulphuric acid. This occurs immediately. Secondly, the fluoride ions which are highly lipophilic penetrate the tissues deeply leading to painful liquefactive necrosis [5]. Necrosis due to fluoride ions occurs progressively despite surface decontamination unless it is neutralized with the formation of the salts of calcium and/or magnesium [6]. This can be by natural calcium or magnesium compounds or by those administered medically.

Death due to severe hydrofluoric acid burns have been documented [5,7]. Severe multiple organ failure due to the direct or indirect effects of local or systemic fluoride toxicity has also been reported [4]. Death may occur as a result of the consequences associated with fluoride ion poisoning such as hyperkalaemia, hypocalcaemia, and hypomagnesaemia. Sudden death and fatal cardiac arrhythmias, such as ventricular fibrillation, are well documented [5].

The severity of hydrofluoric acid burns depends on the concentration of hydrofluoric acid in the offending agent, the surface area involved, and the duration of exposure [1,3]. Massive exposure constitutes a life threatening situation. It is not widely known that massive exposure results from as little as a 1% total body surface area from a >50% hydrofluoric acid solution, or exposure of >5% total body surface area to hydrofluoric acid of any concentration.

Presentation of hydrofluoric acid burns from time of exposure varies on the basis of hydrofluoric acid concentration in the offending agent [7]. Concentrations of greater than 50% always cause immediate pain with readily apparent tissue destruction [7,8]. Concentrations of 20–50% result in the burn becoming apparent within several hours of the exposure [8]. Concentrations of less than 20% may take up to 24 h to become apparent [8]. Weak solutions (approximately 3% hydrofluoric acid) such as those found in metal cleaners can take many hours to cause symptoms, with patients often presenting in severe pain, with intense throbbing usually in the fingertips, in the middle of the night following exposure during the day. Fortunately, this final group represents the common presentation of hydrofluoric acid burns.

The aim of treatment of hydrofluoric acid burns is to neutralize the fluoride ions with calcium and magnesium ions [9]. The modalities used to achieve this goal vary widely and depends largely on the location and severity of the burn. The danger to health care workers attending to patients who have had a massive exposure cannot be underestimated. Protective apparatus should be worn. Pre-emptive calcium (and magnesium) administration is essential along with frequent estimations of serum calcium levels (Algorithm 1). Copious lavage should continue unabated for 30 min while other interventions such as intravenous access are commenced. The most usual situation, however, is that of exposure to the fingertips of a low concentration, 1–2%, of hydrofluoric acid in a metal or fibreglass cleaner. It is remarkable for the amount of pain the victim suffers and the lack of knowledge of the dangerous nature of the agent by the user.

The difficulty of delivering calcium ions to the nail folds has been solved in the past by either removing or splitting the nail. Calcium (usually 10% calcium gluconate) in a solution or a gel could then reach the effected areas. The addition of dimethyl sulphoxide to the solution can alleviate the need for nail removal. Although there is no level one evidence to support the fact that DMSO is superior to standard treatment. anecdotal evidence appears to support its use. Randomised controlled trials are currently in progress at our institution assessing the efficacy of DMSO compared to standard treatment with calcium gluconate alone.

The compound DMSO is used as a solvent to carry the calcium ions through the skin, and deeply into the tissues. The efficacy of DMSO when used in this manner relates to its rapid dermal absorption. DMSO also has a major free radical scavenge effect and so limits inflammation and ongoing injury. Its toxicity is designated as low by Laboratory Chemical Safety Summaries (LCSS) [10]. In humans it may also be used intravesically in treating refractory interstitial cystitis, it may be useful in improving ischaemia in surgical flaps, it is effective in prevention of postherpetic neuralgia when combined with idoxuridine, and it may prevent tissue necrosis after extravasation of antineoplastic agents. It is commonly used by veterinary surgeons for its local anaesthetic and anti-inflammatory properties for the treatment of sprains in animals [11].

The combined solution can be prepared by following either of the two formulae: (a) calcium chloride anhydrous 10% + dimethyl sulphoxide 50%, (b) calcium gluconate 10% + dimethyl sulphoxide 50%. The solution can also be added to a small quantity of water-based surgical lubricant to form a gel of increased viscosity for application to areas such as the face. The topical treatment can and probably should be continued while other techniques such as intra-articular calcium infusions are instituted.

As burns due to hydrofluoric acid are not commonly seen by medical staff, their management can be problematic. Our experience with hydrofluoric acid burns of differing concentrations and anatomical sites led us to develop a series of algorithms for the management of these burns, incorporating new techniques that include dimethyl sulphoxide, and also calcium topical application, and regional, ischaemic, and intravenous calcium injection. Listed below are our guidelines for the management of massive burns (Algorithm 1), cutaneous burns (Algorithm 2), inhalation burns (Algorithm 3), enteral burns (Algorithm 4), and ophthalmic burns (Algorithm 5) due to hydrofluoric acid.

Algorithm 1. Inhalation, ingestion or massive cutaneous exposure to hydrofluoric acid.

>1% surface area of > 50% hydrofluoric acid
> 5% surface area any concentration hydrofluoric acid

↓

Systemic therapy

Pre hospital- consider oral therapy (6 calcium gluconate tablets every two hours)

Hospital- immediate 20 mL 10% gluconate IV (before levels known)

↓

Monitor calcium levels, ECG, clinical signs: paraesthesia, muscle twitching, muscle cramps, tetany

↓

Calcium level not maintained

↓

Further 10 mL 10% calcium gluconate IV,
then 40 mLs 10% calcium gluconate IV over one hour if needed

↓

Calcium Level Not Maintained

Give more calcium
Consider immediate wound excision
Consider Haemodialysis

Algorithm 2. Hydrofluoric acid cutaneous injury.

Lavage 30 minutes ↘ | Institute Systemic Therapy for Major Cutaneous Injury ↙

Initial Topical Treatment with
DMSO 50% + Calcium Gluconate 10%

↓

Pain Persists

↙ Forearm / Hand

↓

Consider Nail Splitting / Removal

↓

Pain Persists

↓

IV Regional Calcium Gluconate
(10ml 10% Calcium Gluconate + Heparin 5000 Units, total volume 40mls) using Bier's Block technique

↓

Pain Persists

↓

Intra-Arterial Calcium Gluconate
(10mLs 10% gluconate in 50mL saline over 4 hours, repeat up to two times)

↓

Pain Persists

↓

Surgical Excision

↘ Elsewhere on Body

↓

Subcutaneous Infiltration of 10%Calcium Gluconate 0.5ml/cm2 of burn

↓

Pain Persists

↓

Surgical Excision

Algorithm 3. Hydrofluoric acid inhalation.

Assume if: 1 Exposure to hydrofluoric acid >50%
2 Clothes Soaked
3 Head or Neck Burn
4 Exposure in Confined Space

↓

Risk Exists!!
Prevent Exposure to Staff
(Mask, Gloves, Goggles, Gown)

↓

Airway First Aid,
Commence Systemic Therapy

↓

100% Humidified Oxygen Therapy
Consider Nebulised Calcium Gluconate 2.5%
(Add 1ml 10% Calcium Gluconate to 4mls Normal Saline)

↓

Monitor: Airway Patency
Conscious State
PO2 / Blood Gases
Consider Intubation, Fibreoptic Laryngoscopy or Bronchoscopy, +/- Lavage

Algorithm 4. Hydrofluoric acid ingestion.

Pre-Hospital: Encourage Oral Fluids (water, milk)
Do Not Induce Emesis

↓

Systemic Therapy and Airway Management as Needed

↙ ↘

Spontaneous Emesis ↓ Calcium Gluconate 10%, 250mls oral

No Spontaneous Emesis ↓ Gastric Lavage if < 2hours since ingestion,with10% calcium Gluconate (use 250ml initially)

↘ ↙

Observe for Signs Upper GI Bleed / Perforation

↓

Consider Endoscopy

Algorithm 5. Hydrofluoric acid eye injury.

Saline Lavage 30 mins

↓

1% Calcium Gluconate Eye drops
one to two drops every two hours to affected eyes
(Add 1ml of 10% Calcium Gluconate IV solution to 9mls Sterile Water)

↓

Immediate referral to Ophthalmologist

5. Summary

Hydrofluoric acid inflicts serious, potentially fatal injuries. Although uncommon in our practice, they often require surgery and are always fatal. Copious irrigation and administration of ionic calcium or magnesium to the site of injury are the two elements of successful treatment. Topical application of DMSO/calcium, Biers block and arterial infusion can be combined to produce affective treatment. Legislative measures to limit the concentration of HF available to the public and ensure appropriate labelling and packaging should be pursued.

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