CASE REPORT

Fatal Unintentional Occupational Poisonings by Hydrofluoric Acid in the U.S.

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Background *Case reports have identified hydrofluoric acid (HF) as causing fatal work injury, and HF has both local and systemic toxicity. Surveillance for HF-related mortality is problematic because of the lack of unique coding for this acid in hospital records and vital statistics.*

Methods *We identified HF-related fatal work injuries investigated by the Occupational Safety and Health Administration (OSHA) for 1984–94 from coding of Hazardous Substance 1460 (HF) and requested case investigation files under the Freedom of Information Act. We attempted to identify HF-related deaths in the US for the same period through literature case reports, the Consumer Product Safety Commission, and the American Association of Poison Control Centers (AAPCC).*

Results *For the 11 year period, OSHA investigated nine deaths in eight incidents which involved HF. Four deaths were from skin contact with concentrated HF, and five deaths involved both skin contact and inhalation of vapor. Unsafe work practices were factors in all of the deaths. Calcium chloride or gluconate was noted to have been administered to five of the nine victims. Calcium was administered 90 min after exposure to two victims, and more than 6 h after exposure to a third. We were able to establish that the regional poison control center had been contacted in regard to only one victim. For the period 1984–94, we were able to identify no additional deaths from CPSC reports, one additional death from AAPCC annual reports, and four other deaths from case reports in the medical literature.*

Conclusions *For the period of this study, OSHA records identified the greatest number of HF-related fatalities. The limited information in the records suggest that some victims did not receive appropriate medical care, nor was the regional poison center contacted regarding care. The full extent of health problems related to HF could be better quantified if usual surveillance sources, such as vital records, included unique coding for this acid.*
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KEY WORDS: hydrofluoric acid; poisoning; fatalities; chemical poisoning; workplace fatality; workplace poisoning

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INTRODUCTION

Hydrofluoric acid (HF) is a corrosive inorganic acid [Himes, 1989]. It is used in industry for etching glass, electronics, and semiconductor manufacturing, and in the petroleum industry to produce high octane fuels [Bertolini, 1992]. It is also a component of some consumer products,

such as rust removers [Saadi et al., 1989]. With regard to human toxicity, HF is of particular concern because not only does its contact with skin cause local burns, but the fluoride ions readily penetrate the skin to cause systemic poisoning. Skin penetration by HF is retarded by the lipid layer of intact skin and accelerated by abrasions or minor trauma [Noonan et al., 1994]. Death can occur from a splash of as little as 2.5% of the body surface with concentrated HF [Tepperman, 1980]. Inhaling HF vapors (boiling point 68°F) leads to pulmonary edema, and in high enough concentrations, death [O'Neil, 1994]. Fluoride ions that penetrate the skin into systemic circulation bind to divalent cations, primarily calcium and magnesium, leading to electrolyte imbalance, cardiac arrhythmia, and death [Sadove et al., 1990]. Fluoride also inhibits glycolysis and oxidative phosphorylation with the same results [DeLauder et al., 1994]. Contact with HF in concentrations less than 50% does not cause immediate pain, so systemic poisoning can begin before the person is aware they have had contact with the acid [U.S. Department of Energy, 1986].

There is little reported information on the epidemiology of HF poisoning. There is no unique International Classification of Diseases (ICD-9) code for HF poisoning, and fatal injuries involving HF would be included under ICD-9 983.1 (toxic effect of corrosive acids) or under coding for burns, and so information on HF is not available from vital statistics or hospital discharge data. Fatalities from HF have been reported as case reports [Menchel and Dunn, 1984; Mayer and Gross, 1985; Manoguerra and Neuman, 1986; Chan et al., 1987; Mullet et al., 1987; Chela et al., 1989; Cappell and Simon, 1993]. Several case reports detailed the circumstances surrounding the 1987 HF release in Texas City, Texas [Brender et al., 1991; Alexeef et al., 1993; Dayal et al., 1994]. For 1985–86 there were four fatalities involving HF reported to the American Association of Poison Control Centers (AAPCC); three were from ingestion and one from dermal exposure [Caravati, 1988].

Emergency management of cutaneous HF exposure includes removal of contaminated clothing, copious irrigation with water, topical application of calcium-containing gels or quaternary ammonium compounds, and intralesional or regional injection of calcium compounds [Caravati, 1988; Sheridan et al., 1995].

The unique toxicity of HF led us to attempt to define the epidemiology of fatal injury and to examine deaths due to HF poisoning as a result of occupational contact, using a variety of sources that allowed identification of HF. We wish to draw attention to the preventability of these deaths, and search for ways to prevent further deaths.

METHODS

Occupational Safety and Health Administration (OSHA) investigation summaries for 1984–94 which had been obtained for another study [Suruda et al., 1999] were available and provided in electronic form by OSHA from the Integrated Management Information Sytems (IMIS) database. This database contains information on OSHA fatality and catastrophe investigations in 47 states for 1984–89 and all 50 states for 1990–94. California, Washington state, and Michigan state OSHA programs had data files incompatible with the federal system until 1990 and reports from those states were excluded until that year. Included in the data were all deaths reported to OSHA for which an investigation was conducted; approximately 2,000 deaths each year. Each report included a field for a hazardous substance involved in the fatality. We first identified all fatality reports containing an entry for substance 1460 (HF). We then printed out the content of the IMIS investigation summary for each of these incidents, identified the OSHA office which conducted the investigation and the identification code for the OSHA compliance officer, and requested in writing from the applicable OSHA office a copy of the full investigation report.

Additionally, we made a written request to the Consumer Product Safety Commission (CPSC) for all fatality reports for 1984–94 involving work-related fatal injury from HF that could be identified from the CPSC Medical Examiner file and also from the National Electronic Injury Surveillance System.

AAPCC maintains the Toxic Exposure Surveillance System (TESS). TESS data include reports from the majority of US Poison Control Centers. We reviewed TESS annual reports from 1984–94 for mention of any fatal work injury involving HF.

RESULTS

For the period from 1984–94, OSHA investigated eight incidents with nine deaths due to HF poisoning. Seven of the fatalities were in manufacturing (Table I). All victims were males. Seven of the nine victims were employed at workplaces which were covered by a collective bargaining agreement with a labor union.

For the same period, the CPSC death certificate file identified three fatal injuries involving HF, all of which had also been investigated by OSHA. One work-related poisoning was identified from the 1992 AAPCC annual report and has been published previously [Litovitz et al., 1992].

A brief summary of individual cases identified from OSHA reports follows.

Case 1

Case 1 occurred on 20 August 1984. A 53-year-old worker was exposed to HF in the process of using methyl chloroform to unclog a line leading to a storage tank of HF. In the process of connecting a hose to the tank valve, the

TABLE I. Distribution of HF Deaths by Industry, 1984–1994, US

Industry group	Specific industry	Number of deaths
Agricultural services	Citrus farming	1
Manufacturing	Industrial organic chemicals	2
	Industrial inorganic chemicals	1
	Petroleum refining	2
	Glass and glassware	1
	Measuring and controlling devices	1
Services	Metals testing	1
Total		9

coupling came loose, splashing him with methyl chloroform (the cleaning agent), and HF (from the tank), resulting in burns to the arm, face, and neck covering 30% of his body. He was showered and was transported to the plant's first aid area, arriving approximately 30 min following the accident. He was transferred to a hospital where he developed seizures and then cardiac arrest, dying 7–8 min after arrival.

Case 2

Case 2 occurred on 20 September 1985. A 51-year-old worker entered an area where there had been an HF spill and cleanup efforts were underway. The worker entered the spill area with only rubber gloves, rubber boots, and a self contained breathing apparatus. A sump pump was being used to pump the spilled acid back into a tank for storage. As the employee moved the pump to a lower level to continue the process, the hose came off of the pump and the worker was sprayed with HF across 40% of his body (face and chest) and died shortly after. A review of pump specifications revealed that this pump was not recommended for use with corrosive material.

Case 3

Case 3 occurred on 26 January 1986. A 62-year-old worker entered the storage area of another company to retrieve a set of tires stored in an overhead loft. He stepped on a 5 gallon plastic bucket of HF (belonging to another company) to ascend in lieu of using a ladder. Upon descending from the loft, the lid of the bucket failed. The worker was splashed with 70% HF on his right lower leg and foot, as well as his posterior thigh. The burn was later estimated to have been to 8% of the total body surface. The worker was stripped, showered for 15 min and immediately taken to the hospital, where he was seen approximately 35 min after the exposure. Morphine sulfate, meperidine, and hydroxyzine were given to control excruciating pain. Two hours later he was transferred to another hospital. Upon arrival at the second hospital some laboratory testing was done but this did not include calcium determination. Approximately 5 h after the exposure, a decision was made to operate and to debride the burned skin. However, he experienced runs of ventricular fibrillation and surgery was canceled. Serum calcium concentration was measured and was found to be low at 5.2 mg/dL. Six hours and twenty minutes after injury, calcium gluconate (60 mL, 10% calcium gluconate) and calcium chloride (2 g) were administered intravenously. His condition declined, with repeated episodes of ventricular fibrillation necessitating cardioversion. Normal calcium levels were obtained 10 h after injury and maintained thereafter. However, ventricular fibrillation eventually became refractory, and he died approximately 15 h 30 min after exposure.

Case 4

Case 4 occurred on 27 February 1986. A 40-year-old worker was replacing a valve attached to a HF line in a water condenser. The worker had closed down the two valves on the line, and opened a drain line to allow HF in the line to drain out. After replacement of the valve, the worker opened one of the valves to place it back on line without closing the drain valve. The open drain valve released HF into a catch basin, splashing the worker across the arms, chest, face, and left leg. In addition, the HF vaporized and was inhaled by the worker. The details of this worker's medical care were not in the OSHA file, other than a notation that autopsy showed pulmonary engorgement.

Case 5

Case 5 occurred on 18 October 1988. A-29-year old worker was transporting 2.6 gallons of HF in an open 15 L pail. The HF was being transported from storage bins to etching tanks where it was used to test airplane parts for flaws. The pail spilled, and the worker was splashed across the chest, neck, and face with 70% HF. This worker was wearing work clothing, long rubber gloves, and street glasses. He was immediately stripped and bathed in a water bath. Inhalation of HF fumes was inferred because he was frothing at the mouth. He died en route to the hospital.

Cases 6 and 7

Case 6 and 7 occurred on 7 March 1991. Multiple workers at a petroleum refinery were involved in the same incident, and two died. A 50-year-old worker and a 42-year, old worker were exposed to HF while replacing a pump motor. An improper lockout procedure failed to keep HF from flowing through the pipes to the pump as it was removed for repairs. This resulted in the pressurization of the pump. The pump containment failed, releasing several hundred pounds of HF onto the unprotected workers. One

worker was pronounced dead at the scene. Another was taken to a local hospital and died shortly thereafter; autopsy revealed severe lung injury.

Case 8

Case 8 occurred on 15 August 1991. A 48-year-old worker was working to modify an HF drain line for an experimental incinerator burn. The drain line was not properly locked out, and the up line valve broke when the line was pressurized. This allowed the acid to splash the worker across the head, neck, shoulders, and arms (approximately 15% of his total body surface). He also inhaled HF fumes. Immediately following the incident the worker was showered, taken to the company first aid station, and then to a hospital emergency department where calcium chloride was administered. He died shortly thereafter from cardio-respiratory arrest. An autopsy revealed pulmonary edema.

Case 9

Case 9 occurred on 10 October 1992. A 37-year-old worker was in the process of pouring HF acid from a 55 gallon drum into a 3 gallon pail for distribution. The worker and a co-worker had stacked crates on top of each other as a makeshift work platform in order to raise the work area closer to a ventilation shaft. In the process of pouring the HF, the drum struck the bucket and knocked it from the platform to the floor. One worker jumped free while the other was splashed with 70% HF to approximately 40% of body surface area. The worker who was splashed was wearing only rubber gloves, work clothes, and goggles. Following the incident calcium gluconate gel was applied within 30 min by the company nurse, and he was transferred to a local community hospital. He was latter transferred to a regional medical center. At the regional medical center, 3 h after injury, he developed ventricular fibrillation. Several doses of calcium chloride were delivered along with cardioversion, but he died approximately 4 h after exposure.

Treatment of the above nine individuals with calcium is summarized in Table II. In the five individuals who received calcium therapy in addition to decontamination by washing with water, the time of administration was noted in four cases. In two cases this time was approximately 1 h 30 min, and in one case it exceeded 6 h. We were able to establish via telephone inquiry that the regional poison control center was contacted regarding the medical care of one of the above cases that was investigated by OSHA (Case 8).

In addition to the fatalities mentioned above, we identified four deaths reported in the medical literature that did not appear to match the cases reported by either OSHA, CPSC, or AAPCC. Two defense workers unloaded a truck containing 70% HF acid, were splashed when a hose disconnected, and died of HF poisoning [Mullet et al., 1987]. A worker ingested rust remover containing HF, mistaking it for water, and died of cardiac arrest [Manoguerra and Neuman, 1986]. There was a case report of a worker splashed across the thighs with HF who died of cardiac arrhythmia approximately 17 h following exposure [Menchel and Dunn, 1984].

DISCUSSION

These data show the difficulty in determining the exact number of work-related poisonings from HF in the US during a particular time period. We were able to identify several cases, but we did not identify all such deaths in 1984–94 using information from AAPCC, CPSC, and OSHA.

In contrast to HF, poisoning by carbon monoxide is identifiable from hospital records and death certificates by the ICD-9 code 986. If there were a unique ICD code for HF, it would facilitate surveillance of poisoning from this acid.

TABLE II. Use of Calcium Treatment

Case no.	Approximate time from exposure to death	Calcium gluconate administered? (Y/N)	Time from exposure to administration
1	30 min	N	
2	3 h 30 min	N	
3	15 h	Y	6 h
4	4 h	Y	1 h 30 min
5	30 min	N	
6	30 min	N	
7	2 h 30 min	Y	Unknown
8	4 h	Y	1.5 h
9	4 h	Y	30 min

Some of the OSHA reports suggest that appropriate treatment for HF poisoning was not given in a timely manner. A common recommended treatment for HF burns for relieving pain from local injury is application of calcium gluconate after surface decontamination is done by removing clothing and washing the affected skin areas with water [Blunt 1964; Velart, 1983; Pegg et al., 1985; Dunn et al., 1996]. Intravenous administration of calcium gluconate is recommended if systemic poisoning is suspected. Calcium chloride is not recommended because it stimulates sensory fibers, causing pain at the site of injection [Pegg et al., 1985]

Regional poison control centers are generally recognized as community resources which can provide information concerning proper treatment of poisoning, including poisoning with HF. It would be useful for research purposes to be able to link OSHA reports of workplace poisoning with AAPCC records, for the purpose of determining whether a regional poison center was consulted regarding care of the injured worker.

Prevention of HF poisoning involves engineering controls to reduce or eliminate exposure, proper work practices, personal protective equipment, and appropriate first aid and emergency care. The danger of work on or in the vicinity of pressurized lines containing HF should be recognized and appropriate lockout/tagout procedures followed. When such work is performed, personal protective equipment effective in reducing dermal and respiratory exposure in case of a spill should be provided. One of the authors provided emergency care to a worker who was splashed by the rupture of a high pressure HF line during servicing of a valve. This worker sustained only a small second degree burn on the chin despite a direct splash to the head, and the burn responded well to topical calcium gluconate therapy. At the time of the splash the worker was wearing a suit, including a helmet, that was impermeable to corrosive acids, and was also equipped with an SCBA. At the time of the incident, there was a standby worker similarly equipped who was ready to assist with evacuation and decontamination.

At work sites where HF or solutions containing HF are in use, decontamination showers should be readily available. Personnel should be trained in decontamination and in application of topical calcium gluconate if on-site medical care is not immediately available. Appropriate topical therapy would be calcium gluconate gel 2.5–10%. Commercially available calcium gluconate gels are not approved by the Food and Drug Administration for sale or use in the US but are available from Canadian and European vendors. We are aware of one major US manufacturer which stocks tubes of gel from European sources at US work sites and trains worker in its use. Calcium gluconate gel can be compounded readily for emergency use by mixing equal amounts of lubricating jelly (K-Y) and 10% calcium gluconate solution and applying it to a wound. For HF burns associated with severe pain out of proportion to physical evidence such as swelling or blistering, local injection of calcium gluconate into the burn area can relieve pain and prevent or delay systemic poisoning [Blunt, 1964; Velart, 1983; Pegg et al. 1985].

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