

# **FATALITY DUE TO ACUTE FLUORIDE POISONING FOLLOWING DERMAL CONTACT WITH HYDROFLUORIC ACID IN A PALYNOLOGY LABORATORY**

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**Abstract**-A fatal accident involving concentrated hydrofluoric acid in a palynological laboratory is described. Similar deaths due to dermal exposure to concentrated hydrofluoric acid have been reported in the literature. It is evident that rigorous control measures including proper personal protective equipment and first aid are of utmost importance in the prevention of death and injury when handling hydrofluoric acid. Possible factors that may have contributed to the accident are reviewed. Copyright © 1996 British Occupational Hygiene Society.

## **INTRODUCTION**

Hydrofluoric acid is a corrosive and toxic liquid that is potentially toxic even following dermal exposure to small amounts (Burke *et al.*, 1973). The fatality described below highlights the potential for relatively small quantities of concentrated hydrofluoric acid to produce acute systemic toxicity and it is clear that laboratory personnel underestimated the risks associated with the acid. The purpose of this paper is to raise awareness of the inherent dangers associated with dermal contact with concentrated hydrofluoric acid, and of the importance of observing strict precautions when handling it.

## **ACCIDENT DESCRIPTION**

A palynological technique used by geologists involves the dissolving of sedimentary rock with mineral acids (hydrochloric and hydrofluoric acid) to liberate acid-insoluble microscopic fossils. The fossils are then examined by microscopy to determine the age of the rock and oil potential.

A 37-year-old male laboratory technician was performing acid digestion of oil well core and ditch samples with 70% w/w concentrated hydrofluoric acid in a fume cupboard. He was believed to be seated when he knocked over a small quantity (100-230 ml) of hydrofluoric acid onto his lap, splashing both thighs. The only personal protective equipment (PPE) worn was two pairs of wrist length rubber gloves and a pair of polyvinyl chloride (PVC) sleeve protectors. As a result of the fact that the technician was working alone, it is unclear whether the spill was from the digestion cup or the 2-l bulk acid container.

The technician sustained burns to 9% of his body surface area, despite washing his legs with water from a makeshift plumbing arrangement that supplied water at 6 l. min<sup>-1</sup>. No calcium gluconate gel was applied to the affected area and contaminated clothing was not removed during the flushing with water. Following flushing, the technician, who appeared to be in severe pain and shock, immersed himself in a chlorinated swimming pool at the rear of the workplace, where he remained for approximately 35-40 min before ambulance help arrived.

The injured man was hypothermic and hypocalcaemic on admission to an intensive care unit at a nearby hospital, and soon became unconscious. His condition continued to deteriorate despite subcutaneous injections of calcium gluconate and administration of intravenous calcium and magnesium. His right leg was amputated 7 days after the incident. He subsequently died from multi-organ failure 15 days after hydrofluoric acid spill.

## **LIKELY CONTRIBUTING FACTORS**

The most significant factor influencing acute systemic toxicity of hydrofluoric acid is the

total amount of fluoride ion absorbed. In dermal exposures this is a function of the duration of exposure, the total surface area affected and the concentration of the hydrofluoric acid (Krenzelok, 1992).

It is clear that one of the main factors that contributed to the systemic effects was the dermal exposure of 9% body area to concentrated hydrofluoric acid.

Wounds as small as 2.5% of the body surface area from concentrated hydrofluoric acid can produce hypocalcaemia of potentially lethal extent within 2 or 3 h (Greco *et al.*, 1988). Adequate personal protective equipment during the handling of concentrated hydrofluoric acid could have prevented this death. Full-protectors, a face shield, rubber boots, safety goggles and mid-arm length PVC gloves should have been worn by the deceased when hydrofluoric acid was being used in the fume cupboard.

The duration of exposure may also have contributed to the uptake of the fluoride ion. Hydrofluoric acid passes through the skin into deep tissue rapidly and the affected must be flushed with water immediately (Bracken *et al.*, 1985; Greco *et al.* 1988). The deceased did not have access to an emergency shower to remove the hydrofluoric acid, instead the skin was washed from a hose that provided water at a very low flow rate. Because of the low flow rate, the volume of water may have spread the hydrofluoric acid onto other parts of the skin, rather than washing the hydrofluoric acid off the skin. After a number of minutes, the deceased immersed himself in the swimming pool.

Although flushing is effective in removing surface hydrofluoric acid, it does not affect the fluoride ion that may have already penetrated deeper (White, 1984). In this instance, no calcium gluconate gel was applied following dermal exposure to hydrofluoric acid; calcium gluconate gel is an effective topical treatment for hydrofluoric acid burns (Trevino *et al.*, 1983).

Even though the deceased sustained burns to 9% of body surface area inhalation may also have been another route of exposure due to the relatively high vapor pressure of hydrofluoric acid. Pulmonary edema was noted at autopsy but it was unclear whether this was due to hydrofluoric acid inhalation or to other causes.

An ergonomic assessment of the work station indicated the following.

1. The working height of the fume cupboard was too low (by between 110 and 160 mm) for the deceased to work comfortably in a standing position. Sitting would have increased the body surface area during a spill of hydrofluoric acid.
2. Instability of the digestion cups due to lightweight construction, that is, height 75 mm, diameter of base 59 mm, diameter at top 78 mm made of 2 mm polyethylene.
3. Lack of available space in the fume cupboard, entrance of the fume cupboard was 470 mm wide and 410 mm high.
4. As a result of the design of the container, decanting from the 2-l hydrofluoric acid container was awkward, involving the pronation of the forearm.

The laboratory personnel may have minimized the likelihood of a spill through the introduction of cup supports for the digestion cups. Other ergonomic factors which may have reduced the likelihood of a spill include:

1. provision of a fume cupboard that had more working space;
2. use of smaller sized bottles or better designed 2-l. 70% w/w hydrofluoric acid containers (to minimize awkward pouring postures) or introduction of a graduated dispensing unit to negate pouring the acid;
3. provision of an appropriate bench height for the operator.

Overall, the laboratory did not comply with the requirements of Australian Standards "Safety in Laboratories" (AS 2243, 1992) Parts 1,2 and 8 in the areas of emergency procedures, safe handling and disposal of the chemical and laboratory design.

## DISCUSSION AND CONCLUSION

Mullet *et al.* (1987) described a similar fatality with 70% hydrofluoric acid where a 61-year-old-male sustained burns to 8% of his body surface area. This individual died from cardiac arrhythmia secondary to the depletion of ionized calcium, by fluoride ion. As in the case reported here, the burns were predominantly on the right leg, the injured person washing his leg with tap water for approximately 15 min. He reached the hospital 35 min after sustaining the injury. Calcium gluconate gel was not applied to the burns site until he reached the hospital and although subcutaneous and intravenous calcium therapy was given at the hospital, he died 15.5 h after the injury.

By contrast, Greco *et al.* (1988) reported the case of a 50-year-old worker who survived burns to 22% body surface area from 70% hydrofluoric acid. He showered immediately, had calcium gluconate gel applied to the wounds and was taken to a nearby hospital where he was promptly treated with subcutaneous and intravenous calcium.

It is evident that apart from location of burns, the size of the burns and concentration of the acid, washing the area affected immediately and the application of calcium gluconate gel to reduce the uptake of fluoride ion may prevent a fatality. Greco *et al.* (1988) proposed that the development of hypocalcaemia may occur in the following situations:

- (a) burns of  $\geq 1\%$  surface area from 50% (or greater concentration) hydrofluoric acid.
- (b) 5% or greater surface area with any concentration of hydrofluoric acid; and
- (c) inhalation of fumes from 60% (or greater concentration) hydrofluoric acid.

As noted by Stencil and Tobin (1987) and Mansdorf (1987), appropriate protective clothing, prompt first-aid and proper clean-up procedures are critical for workers handling hydrofluoric acid. Failure to wear appropriate PPE and failure to follow appropriate first-aid procedures, may result in severe injury and increase the likelihood of death from fluoride poisoning. Nearly 90% of hydrofluoric acid exposures result in the development of some toxic sequelae, and approximately 80% of patients require treatment in a health care facility (Krenzelok, 1992).

It is clear that the laboratory did not comply with relevant Australian Standards for Safety Commission, 1994) require that a proper risk assessment is performed and it is hoped that the enforcement of these regulations will prevent fatalities such as the one described here.

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## REFERENCES

As 2243 (1992) Safety in Laboratories, Parts 1-8. Standards Association of Australia, North Sydney.

Brackten, W. M., Cuppage, F., McLaury, R.L. Kirwan, C. and Klaasseo, C.R. (1985) Comparative effectiveness of topical treatments for hydrofluoric acid burns. *J. occup.Med.* 7, 733-739.

Burke, W.J., Hoeg, U.R. and Phillips, R.E.(1973) Systemic fluoride poisoning resulting from a fluoride skin burn. *J. occup. Med.* 15, 39-41.

Greco, R.J., Harford, C.E., Haith, L.R. and Patton, M.L. (1988) Hydrofluoric acid-induced hypocalcaemia. *J.*

*Trauma* 28, 1593-1596.

Krenzelok, B.P. (1992) Hydrofluoric acid. In Hazardous Materials Toxicology: *Clinical Principles of Environmental Health* (Edited by Sullivan, J.B. and Krieger, G.R.). Williams and Wilkins, Baltimore.

Mansdorf, S.Z. (1987) Anhydrous hydrofluoric acid. *Am. ind. Hyg. Ass. J.* 48, A452.

Mullet, T., Zoeller, T., Bingham, H., Pepine, c.J., Prida, X.E., Castenholz, R. and Kirby, R. (1987) Fatal Hydrofluoric acid cutaneous exposure with refractory ventricular fibrillation,

*JBCR* 8, 216-219.

National Occupational Health and Safety Commission (1994) Control of Workplace Hazardous Substances: National Model Regulations NOHSC: 1005 (1994). Australian Government Publishing Services, Canberra.

Stencel, J.R. and Tobin, T. (1987) Hydrofluoric acid burn: Latent period was key factor. *Am. ind. Hyg. Ass. J.* 48, A451-A452.

Trevino, M.A., Hermann, G.H. and Sprout, W.L. (1983) Treatment of severe hydrofluoric acid exposure. *J. occup. Med.* 25, 861-863.

White, J.M. (1984) Hydrofluoric acid burns. *Cutis* 34, 241-244.