

## FEATURE

# Hexafluorine decontamination of 70% hydrofluoric acid (HF) vapor facial exposure: Case report

Hydrofluoric acid (HF) dermal exposure at concentrations of 50% or greater results in rapid onset of intense pain and tissue destruction. Despite early water decontamination and topical calcium gluconate application, severe burns and systemic sequelae including death from cardiac dysrhythmias and cardiovascular collapse secondary to severe metabolic acidosis and electrolyte abnormalities (hypocalcemia, hypomagnesemia, hyperkalemia) cannot always be prevented. A worker in a French crystal and glass manufacturing facility had facial exposure to 70% HF vapor with immediate onset of severe pain that was relieved by washing with Hexafluorine. Only mild, painless, transient erythema occurred in the exposed area. Topical calcium gluconate was prescribed the day following the exposure for residual painless erythema. There were no sequelae and no lost work time. Hexafluorine is an amphoteric, hypertonic, *active* decontamination solution for HF splashes. It neutralizes the  $H^+$  ion and chelates the  $F^-$  ion of HF. In the case presented here, Hexafluorine decontamination was associated with the absence of expected severe local and systemic effects of 70% HF vapor facial exposure.

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## INTRODUCTION

Hydrofluoric acid (HF) is one of the strongest inorganic acids, and is widely used mainly in industry for the manufacture of fluorinated organic compounds, inorganic fluorides, fluorocarbon products, and fluoropolymers and derivatives, and for uranium

treatment.<sup>1,2</sup> It is also used in the metals industry, in oil refining, in electronics, in construction, in the chemical industry and in laboratories, in refrigeration, and in the photovoltaic industry.<sup>1,2</sup> In the glass and crystal industry, HF is used for engraving, etching, frosting, and polishing, and for quartz purification.<sup>1,2</sup> It is also used in household products, such as rust removers.<sup>3</sup>

HF is particularly dangerous because of its corrosiveness and toxicity. As it is a small molecule and only partially dissociated ( $pK_a = 3.2$ ), HF is capable of penetrating deeply into tissues. HF burns are a unique clinical entity. Dilute solutions can penetrate

deeply before dissociating, causing delayed injury and symptoms. HF causes severe tissue necrosis and systemic poisoning due to two ion delivery mechanisms: the corrosive hydrogen ion ( $H^+$ ) associated with cutaneous and mucous membrane lesions<sup>4</sup> and with ocular<sup>5</sup> and respiratory tract<sup>6-8</sup> damage; and a toxic fluoride ion ( $F^-$  with local and systemic effects (decreased myocardial contractility, tachycardia, torsades de pointes ventricular tachycardia (a specific variety of ventricular tachycardia with the electrocardiographic appearance of “twisting of the points”), and ventricu-

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lar fibrillation),<sup>9</sup> with potentially lethal toxicity.<sup>10–14</sup>

The F<sup>-</sup> ion chelates calcium and magnesium<sup>15</sup> forming the insoluble salts, CaF<sub>2</sub> and MgF<sub>2</sub>, and soluble salts, NaF and KF, which at high concentrations are direct cellular poisons. This induces metabolic disorders<sup>16,17</sup> which lead to cellular death and secondary tissue necrosis. Calcium binding is thought to increase cell membrane potassium permeability, resulting in neuronal depolarization and intense pain.<sup>18,19</sup> Fluoride ion itself is known to attack enzymes and cell membranes.<sup>20</sup>

HF's systemic toxicity is due to increased body fluoride concentrations which effect blood concentrations of calcium (hypocalcemia), magnesium (hypomagnesemia), and potassium (hyperkalemia).<sup>21–23</sup> In concentrations of 50% or greater (including 70% and anhydrous), HF causes immediate, severe, throbbing pain and whitish discoloration of the skin, usually with blistering.<sup>24</sup>

HF systemic toxicity is potentially life-threatening.<sup>25</sup> The risk of fatality is correlated with the HF concentration, the total body surface area (TBSA) exposed, and the duration of contact.<sup>25</sup>

Hexafluorine is a solution specifically developed to decontaminate splashes with HF and similar fluoride compounds. The name “Hexafluorine” is a registered trademark of the manufacturer (Laboratoire Prevor, Valmondois, France) who consider the generic chemical name and formula to be confidential proprietary information.<sup>26</sup> It is a sterile, aqueous solution containing amphoteric and chelating salts.<sup>26</sup> Hexafluorine is only supplied in one concentration and is a clear and colorless liquid with a specific gravity of 1.046 g/m<sup>3</sup> and a pH of 7.2–7.7.<sup>26</sup> Specifically, Hexafluorine *does not* contain 6 fluorine atoms; rather, it has specific binding sites for both the H<sup>+</sup> and F<sup>-</sup> ions of HF.

Fortunately, cases of exposure to concentrated HF are not common. However, when they do occur, traditional initial water decontamination may not always prevent or mitigate severe local skin injury and systemic toxicity. Hexafluorine represents an alternate method for more efficacious

initial HF decontamination. An illustrative case is described below.

## CASE REPORT

A 70% HF facial vapor exposure occurred in a French industrial facility that manufactures glass and crystal. Since 1972, this facility has used about 240 tons of HF per year. Personnel who work on the HF line are trained about the hazards and risk of HF and HF splash decontamination. Until 1993, this consisted of copious water washing followed by topical application of 3% calcium gluconate gel, which did not always result in satisfactory clinical outcomes. After 1993, Hexafluorine (Laboratoire Prevor, Valmondois, France) was used instead of water as the initial decontamination solution and calcium gluconate was only used when necessary to treat actual HF burns.

A 35-year-old male technician was exposed to 70% HF vapor on the right cheek (approximately 1–2% total body surface area) when opening a valve in the hydrofluoric acid circuit. He immediately felt severe pain in the exposed area. Safety goggles were worn, so no eye exposure occurred. The worker immediately decontaminated himself with a Hexafluorine 5-Liter high volume/low pressure portable shower, after which he rapidly became pain-free.

On medical examination in the facility infirmary, there were no clinical findings other than mild, painless erythema of the exposed area. The following day, erythema had mostly resolved and the worker had no pain sensation. Topical treatment with 3% calcium gluconate was initiated the day following the exposure as some residual painless erythema was still present. During the following week and at 1 month post-exposure, the worker was re-examined in the facility infirmary and no sequelae were noted. There was no lost work time.

## DISCUSSION

Numerous experimental studies have been published in which various decontamination solutions were tested for the

ability to decrease the severity of HF burns following dermal exposure.<sup>27–30</sup> The most widely recommended decontamination method is initial water washing followed by topical inunction of 2.5–3% calcium gluconate gel,<sup>19,31</sup> although iced benzalkonium chloride is frequently used in industrial settings.<sup>15</sup> Water washes unabsorbed HF off the exposed skin surface while the calcium ion (Ca<sup>2+</sup>) from calcium gluconate binds the fluoride ion (F<sup>-</sup>) as calcium fluoride (CaF<sub>2</sub>), mitigating its local and systemic effects.

However, published cases of the use of water washing plus initial topical calcium gluconate application for concentrated HF exposure do not provide convincing evidence of efficacy in preventing severe dermal burns or systemic toxicity, including death from severe metabolic acidosis and electrolyte abnormalities (hypocalcemia, hypomagnesemia, hyperkalemia) resulting in dysrhythmias, cardiovascular collapse, and cardiac arrest.<sup>14,32–42</sup>

Burn healing time may be quite long in non-fatal concentrated HF exposure cases, and lost work time has ranged from 24 to 50 days up to 1 year when standard water decontamination and treatment with calcium salts has been done.<sup>25,41–43</sup> Concentrated (≥50%) HF dermal facial exposures are particularly dangerous, and deaths have been reported at these concentrations when only about 2.5% or less of the TBSA has been involved. A further concern is that sometimes in emergency situations, hydrofluoric acid exposure may be confused with hydrochloric acid exposure because of the *similar sounding chemical names*, resulting in specific treatment delays and potentially serious or even fatal outcomes.<sup>3,42</sup> While hydrochloric acid exposure may cause severe coagulation necrosis skin injury, systemic toxicity does not commonly occur and fatalities are unusual. With hydrofluoric acid exposure, severe local tissue injury is common, and with concentrated HF, severe or even fatal systemic toxicity commonly occurs, as described above. Specific HF treatments such as calcium salts have no place in the management of hydrochloric acid injuries.

From reported cases, two basic principles may be determined: (1) a

necessity to minimize local tissue injury and destruction by reducing the duration of HF skin contact, and; (2) limiting or preventing systemic effects of the exposure through an *active* decontamination with a solution having specific HF binding activity. Such an *active* decontamination solution for HF dermal splashes should act simultaneously against the *corrosive* potential of the H<sup>+</sup> ion and the *toxicity* of the F<sup>-</sup> ion. Water decontamination provides only mechanical rinsing and dilutional actions and may have a “wash-in effect” increasing chemical permeation into the skin.<sup>44,45</sup> Water thus has only *passive* actions.

Hexafluorine is an *active* decontamination solution.<sup>41,46-48</sup> It is not irritating to the eyes or skin, is non-sensitizing, and nontoxic.<sup>48</sup> Hexafluorine is water soluble, is supplied as a sterile solution in water,<sup>48</sup> and would be expected to have the same mechanical washing and dilution actions as a similar volume of water.

For HF decontamination *in vitro*, in equal volumes, water had little effect on either the pH or pF (negative logarithm of the fluoride ion concentration) and calcium gluconate did not return the pH or pF to physiologically tolerable levels, while Hexafluorine did return the pH and pF to such tolerable levels.<sup>48</sup>

In a study of 120 New Zealand albino rabbits with dermal exposure to 70% HF, water washing alone resulted in the appearance of skin injury by 10 minutes after exposure, which became severe by 2 hours through 6 days of observation.<sup>46</sup> Water washing followed by topical 2.5% calcium gluconate delayed the onset of injury appearance and decreased its severity, while no dermal injury occurred after Hexafluorine washing.<sup>46</sup>

In a recent study of 70% HF exposure using living human skin explants *ex vivo*, concentrated HF penetrated to and damaged all layers of the skin within 5 minutes.<sup>49</sup> Washing after exposure to 70% HF with water followed by topical application of calcium gluconate gel delayed the onset of dermal injury and decreased its severity, while washing with Hexafluorine completely prevented any skin injury.<sup>50</sup>

Hexafluorine has been reported to be effective in 32 workers with eye or skin

HF chemical splashes with concentrations ranging from 6% (as “pickling acid”); 6% HF/15% nitric acid) to 70%.<sup>44,47,51</sup> A recent case report of 70% HF dermal exposure showed efficacy in arresting burn progression, even with delayed Hexafluorine washing.<sup>52</sup> Pain relief as noted in the case reported here most likely results from HF inactivation, as Hexafluorine has no analgesic or anti-inflammatory properties.

In contrast, a study in rats in which 50% HF was left in contact with the skin for 3 minutes did not find superior efficacy of Hexafluorine washing as compared to water washing or water washing followed by topical calcium gluconate.<sup>53</sup> Because the 50% HF was left in contact with the rat skin for 3 minutes before washing with either water or Hexafluorine was begun, it is likely that the “window of opportunity” for the efficacy of decontamination had passed and that irreversible tissue injury had already occurred.<sup>54</sup> What these authors have actually shown is further proof of the known efficacy of topical calcium gluconate as a *treatment* for already established HF burns. As these authors did not utilize a Hexafluorine washing + topical calcium gluconate group, it is not possible to know whether or not the same results might have been found as in the water washing + topical calcium gluconate group. This rat study is inconsistent with the results described above for *in vitro*, rabbits *in vivo*, and human skin explants *ex vivo* studies and with the above-described human clinical case reports, all of which demonstrate better efficacy of initial Hexafluorine decontamination.

## CONCLUSION

In the case reported here, emergent use of Hexafluorine self-decontamination and delayed topical application of calcium gluconate gel was associated with the absence of expected serious burns and potential systemic toxicity after dermal facial 70% HF vapor exposure.

## CONFLICT OF INTEREST

At the time this case occurred, Dr. Siéwé was an employee of Laboratoire

Prevor, Valmondois, France, and the manufacturer of Hexafluorine. Dr. Siéwé's views as expressed in this paper *do not in any way* express those of his current employer. Dr. Mathieu and M. Blomet are employees of Laboratoire Prevor. Dr. Hall is a consultant to Laboratoire Prevor.

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