Hydrofluoric Acid Burns: Missed Diagnoses Lead to Delayed Treatment

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Hydrofluoric acid (HF) burns present a significant diagnostic challenge in emergency and primary care settings, often leading to delayed intervention. The initial clinical presentation can be deceptively mild, masking the severe underlying tissue damage and systemic toxicity that characterise these injuries. Prompt recognition and immediate initiation of calcium gluconate therapy are paramount to mitigate morbidity and prevent potentially fatal outcomes.

KEY TAKEAWAYS

- The Pivot: HF burns are frequently misdiagnosed due to a lack of immediate, severe symptoms, particularly with lower concentrations.
- The Data: Delayed diagnosis can lead to systemic toxicity, including hypocalcaemia, hyperkalaemia, and cardiac arrhythmias.
- The Action: Clinicians should maintain a high index of suspicion for HF exposure, especially in industrial or domestic settings, and initiate calcium gluconate therapy empirically if suspicion is high.

Hydrofluoric acid is a highly corrosive inorganic acid used extensively in industrial processes, such as glass etching, metal cleaning, and petroleum refining, as well as in various domestic products, including rust removers and wheel cleaners.1 Unlike other acids, HF readily penetrates the skin and soft tissues due to its small molecular size and lipophilicity.2 Once absorbed, the fluoride ion dissociates and binds to calcium and magnesium ions, leading to severe local tissue destruction, liquefaction necrosis, and systemic electrolyte disturbances.3

The clinical presentation of HF burns is often misleading. With dilute solutions (less than 20% HF), pain may be delayed for several hours, sometimes up to 24 hours, after exposure.4 This latency period contributes significantly to misdiagnosis, as the absence of immediate pain or visible tissue damage can lead clinicians to underestimate the severity of the injury.5 Initial signs may include erythema, oedema, and blanching of the skin, which can be mistaken for a minor thermal or irritant burn.6 However, the fluoride ions continue to penetrate deeper tissues, causing progressive destruction of bone and soft tissue, even in the absence of overt skin lesions.7

The insidious nature of HF burns poses a significant diagnostic challenge in emergency departments and primary care settings. Patients may present with seemingly minor skin irritation, reporting delayed onset of pain, which can lead to initial misdiagnosis as a superficial burn, contact dermatitis, or even a fungal infection. This delay in accurate diagnosis directly impacts treatment initiation, allowing fluoride ions more time to exert their cytotoxic effects locally and systemically. The lack of specific diagnostic tests for early-stage HF exposure further complicates the clinical picture, relying heavily on a thorough history and a high index of suspicion. Epidemiological data indicate that occupational exposures are common, particularly in industries involving metal processing, electronics manufacturing, and chemical production. However, domestic exposures, often involving household cleaning products, also contribute to the incidence of HF burns, affecting a broader patient population, including children. The severity of these burns can range from mild dermal irritation to life-threatening systemic toxicity, underscoring the importance of prompt and accurate identification.

Clinical Manifestations and Management

As fluoride ions are absorbed systemically, they chelate circulating calcium and magnesium, leading to hypocalcaemia and hypomagnesaemia.8 This can manifest as muscle cramps, paraesthesia, and, in severe cases, tetany, seizures, and cardiac arrhythmias, including ventricular fibrillation.9 Hyperkalaemia can also occur due to cellular damage and potassium efflux.10 The extent of systemic toxicity correlates with the concentration of HF, the duration of exposure, and the body surface area affected.11 Burns involving more than 5% total body surface area (TBSA) with dilute HF or 1% TBSA with concentrated HF are associated with a high risk of systemic toxicity.12

The cornerstone of HF burn management is immediate and aggressive calcium gluconate therapy.13 This aims to provide exogenous calcium ions to bind with the fluoride, thereby preventing further tissue damage and systemic chelation.14 Topical calcium gluconate gel (2.5% or 5%) should be applied liberally to the affected area and massaged in continuously until pain relief is achieved.15 For more severe or persistent pain, or burns involving the nail bed, digital blocks with calcium gluconate (0.5 mL of 10% calcium gluconate diluted with 9.5 mL of 1% lidocaine without epinephrine) may be administered.16 Intra-arterial calcium gluconate infusions are reserved for extensive limb burns or those unresponsive to other modalities.17 Systemic hypocalcaemia requires intravenous calcium gluconate administration, guided by serial electrolyte monitoring.18

The critical challenge for clinicians is to maintain a high index of suspicion for HF exposure, particularly when patients present with unexplained pain disproportionate to visible injury, or with a history of exposure to cleaning agents or industrial chemicals.19 A detailed occupational or recreational history is essential.20 Early recognition and prompt initiation of calcium gluconate therapy are crucial to prevent the progression of local tissue damage and the development of life-threatening systemic complications.21

Limitations in current diagnostic approaches primarily revolve around the non-specific initial presentation and the reliance on patient history, which may be incomplete or inaccurate. Furthermore, the availability of calcium gluconate gel and the expertise in administering regional or intra-arterial calcium infusions may vary across healthcare settings, particularly in rural or resource-limited areas. Future research should focus on developing rapid, point-of-care diagnostic tools to identify fluoride ion presence in tissues, which could significantly reduce diagnostic delays. Educational initiatives targeting both healthcare professionals and at-risk populations are also vital to improve awareness of HF burn symptoms and the importance of immediate first aid and medical attention.

> **CLINICAL IMPLICATIONS**
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> The insidious nature of hydrofluoric acid burns, particularly with lower concentrations, presents a significant liability for clinicians. The delay between exposure and the onset of severe pain means that patients often present with seemingly innocuous injuries, leading to misdiagnosis as a minor thermal or irritant burn.
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> This diagnostic pitfall is not merely an academic point; it directly translates into delayed calcium gluconate therapy, which is the only effective antidote. The consequences are severe, ranging from extensive tissue

necrosis requiring surgical debridement or amputation, to life-threatening cardiac arrhythmias secondary to hypocalcaemia. Emergency departments and general practitioners must adopt a lower threshold for suspicion, especially when a patient's history includes potential exposure to industrial chemicals or even common household products like rust removers. Empirical treatment with calcium gluconate gel, even in the absence of definitive diagnosis, is a prudent measure that can prevent catastrophic outcomes.

The industry, particularly manufacturers of products containing hydrofluoric acid, bears a responsibility to ensure clear and prominent labelling regarding the specific hazards and first aid measures. While safety data sheets (SDS) are legally required, their accessibility and comprehensibility for the general public, or even for all healthcare professionals, are often suboptimal. A more proactive approach to public and professional education on the unique dangers of HF, perhaps through industry-sponsored campaigns or collaborations with medical societies, could significantly improve early recognition. The current reliance on clinicians to 'think dirty' and consider rare exposures places an undue burden on an already stretched healthcare system, especially when the initial presentation offers little to guide diagnosis.

For patients, the lack of immediate, severe symptoms is a double-edged sword. It prevents immediate alarm but allows the fluoride ions to wreak havoc unchecked. This underscores the need for public awareness campaigns, particularly for individuals working with or regularly exposed to HF-containing products. Understanding the delayed onset of pain and the necessity of immediate, specific treatment could empower patients to seek appropriate medical attention sooner and to inform healthcare providers of potential exposure, even if their symptoms appear mild. The current system often fails these patients at the initial point of contact, not due to negligence, but due to a systemic underappreciation of HF's unique pathophysiology.

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