IS – 37
AACT Symposium lecture Hydrofluoric Acid

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Hydrofluoric acid (HF) and related fluoride compounds are unique among acids producing both local and systemic toxicity. Because fluoride is strongly electronegative, free dissociation is restricted leaving a large amount of HF in its non-dissociated form.

Mineral acids (such as hydrochloric acid) are almost completely dissociated into hydrogen ions and an anion. By comparison, HF with a pKa of 3.17 is a weak acid. HF is able to penetrate the skin far more easily than other mineral acids because uncharged molecules pass across biological membranes more easily than charged molecules.

Once in and beyond the skin, fluoride avidly binds to available cations (such as calcium and magnesium) interfering with critical cellular pathways. Additionally, the hydrogen ion liberated is now available for direct tissue injury. While small body surface area burns with dilute hydrofluoric acid typically produce painful direct tissue injury, larger burns and those with more concentrated HF also produce life-threatening systemic toxicity.

Cutaneous injury from HF is rarely responsible for toxicity. Systemic toxicity is manifested by hypocalcemia, hypomagnesemia and hyperkalemia. While it is assumed that systemic toxicity is a direct result of these electrolyte abnormalities, patient fatalities are reported even when these electrolytes are corrected, suggesting additional toxic pathways such as ion channel dysregulation. An example is seen with cardiac toxicity that still occurs with normalization of electrolytes but seems to be mitigated with quinidine or amiodarone. [1,2]

Treatment requires a two-tiered approach addressing both local toxicity and potential systemic toxicity. Local treatment begins with copious irrigation of the skin to remove any remaining HF. This is followed by a graded approach that begins with application of calcium gel for mild injury and progresses to direct tissue infiltration with calcium gluconate or intra-arterial perfusion with dilute calcium gluconate solutions. When intra-arterial perfusion is unavailable or technically not feasible, a bier-block technique is sometimes suggested. [3] Unfortunately no trials exist to compare any of these techniques.

When the body surface area of the burn exceeds 2-3% and the HF solution is concentrated (> 10-20%) systemic toxicity should be anticipated. Patients should be observed in a high acuity setting with continuous cardiac monitoring. Aggressive treatment of hypocalcemia and hypomagnesemia may be required and require rapid laboratory turnaround time and ECG monitoring. When patients exhibit refractory cardiac toxicity with dysrhythmias, treatment with a potassium channel blocking antidysrhythmic such as quinidine or amiodarone seem reasonable, albeit with limited supporting evidence. Although rarely feasible in a clinically meaningful time-frame fluoride is amenable to hemodialysis. [4,5]