

Incidence of Hypocalcemia in Acidotic Ethylene Glycol Poisoned Patients

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Ethylene glycol poisoning is responsible for 0.3% of all toxin exposures reported nationally to the American Association of Poison Control Centers and is responsible for 3% of all deaths due to poisonings. For Central New York alone this represents about 150 poisoning cases per year (almost 3 per week.) We know that ethylene glycol is metabolized in the body to toxic by-products that result in an elevated anion gap metabolic acidosis and renal failure. Further, we know that oxalic acid, one of the toxic by-products, combines with calcium to form calcium oxalate and that calcium oxalate crystal deposition is responsible for the acute renal failure induced by ethylene glycol. Textbooks routinely describe the development of systemic hypocalcemia after severe ethylene glycol poisoning due to this calcium oxalate production and deposition. However, because there have been only a few suggestive case reports, it has been postulated that systemic hypocalcemia is only a theoretical concern because only calcium that would otherwise be renally excreted chelates with oxalic acid.

We theorize that in ethylene glycol poisoned patients with a severe metabolic acidosis, that serum calcium levels will be low.

The goal of our study is to document the prevalence of hypocalcemia in patients presenting to the Emergency Department with severe metabolic acidosis and a history of ethylene glycol exposure and to compare laboratory and physiological findings of those patients with patients with documented severe metabolic acidosis without a history of ethylene glycol exposure. We will perform a retrospective chart review of all patients who presented to SUNY Upstate Medical University with metabolic acidosis according to ICD-9 codes from January 1, 2000 through December 31, 2006.