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Selected Topics: Toxicology

RECURRENT ETHYLENE GLYCOL POISONING WITH ELEVATED LACTATE LEVELS TO OBTAIN OPIOID MEDICATIONS

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☐ **Abstract—Background: Malingering is when a patient feigns illness for secondary gain. While most patients with malingering manufacture or exaggerate symptoms, some patients may induce illness. Previous reports of malingering patients inducing illness include sepsis, kidney pain, migraine, and chest pain. However, acute poisoning as a manifestation of malingering appears to be rare. Case Report: We describe the case of a 39-year-old man who presented to the emergency department complaining of diffuse body pain. The patient reported multiple admission at outside hospitals for "lactate" and said, "it feels like it is happening again because of how my body feels." Laboratory findings were concerning for serum lactate of >20.0 mmol/L and ethylene glycol (EG) level of 19 mg/dL. A chart review found that the man had been admitted for elevated serum lactate 8 times to area hospitals in several years, often in the setting of EG poisoning. During these episodes he required intravenous fluids and frequent intravenous pain medications. When confronted about concern regarding the recurrent fallacious lactate levels in the setting of factitious EG ingestion, the patient often became combative and left against medical advice. The primary metabolite of EG, glycolic acid, can interfere with lactate assays, causing a false elevation. Our patient apparently recognized this and took advantage of it to be admitted and receive intravenous opioids. This is the only case known to us of malingering via EG ingestion. Why Should an Emergency Physician be Aware of This?: Emergency physicians should be aware that metabolites of EG may interfere with serum lactate assay. In addition, they should be aware of possible malingering-related poisoning and plausible association with requests for intravenous opioid pain medications. This represents a risk to the patient and others if undiagnosed.**

☐ **Keywords—ethylene glycol poisoning; factitious disorder; lactatemia; malingering**

INTRODUCTION

Aberrant opioid use behavior may lead patients to complain of fictitious ailments or exaggerate actual medial conditions. A significant number of these patients present complaining of dental pain, headache, and back pain (1). Recent emphasis has been placed on minimizing opioids for these complaints where data do not support their use (2). While such guidelines emphasize the appropriate duration and type of oral pain medications to prescribe upon discharge, they rarely address treatment of pain for admitted patients. A different dilemma presents itself when a patient requires treatment for a potentially life-threatening illness and insists on intravenous pain medication.

Toxic exposure has not commonly been associated with patients who are suspected of drug-seeking behavior. One notable exception is a patient found to have multiple fictitious snakebites requiring narcotics

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(3). Factitious disorder, where a patient makes themselves sick to obtain medical care, has been associated with the surreptitious use of insulin and anticoagulants. We report a case of lactic acidemia caused by recurrent, surreptitious ethylene glycol (EG) ingestion. EG is commonly used in cases of suicidal ingestion as well as accidental ingestion (4). Cases of suicidal ingestion of EG and malicious poisoning with EG have been associated with heightened media coverage following sensational poisonings (5). Its main toxicity results from conversion to glycolic acid, responsible for metabolic acidosis, and then oxalic acid, which precipitates with calcium in renal tubules, leading to acute kidney injury (Figure 1) (6). While EG can lead to elevated lactate levels up to 5 mmol/L secondary to metabolic derangement and elevated nicotinamide adenine dinucleotide (similar to alcoholic ketoacidosis), much higher levels have been reported. These cases generally reflect the glycolic acid metabolite causing a false-positive lactate assay on specific models of blood gas analyzer (7,8).

CASE REPORT

A 39-year-old man with history of major depressive disorder, anxiety disorder, migraines, and chronic back pain presented to our emergency department with a complaint of "this is how it feels when my lactate is acting up." He reported 2 days of diffuse myalgia and fatigue. Other symptoms included persistent fevers in the evening and dyspnea on exertion for 4 months. In the past 2 years, he had been admitted to multiple hospitals for similar symptoms in the setting of lactatemia that typically resolved with intravenous fluids. The patient stated, "nobody has figured out why I have recurrent lactic acidosis." An attempt was made to get previous medical records to access previous diagnostic workups; however, this was complicated by multiple visits at multiple hospitals extending over the course of years.

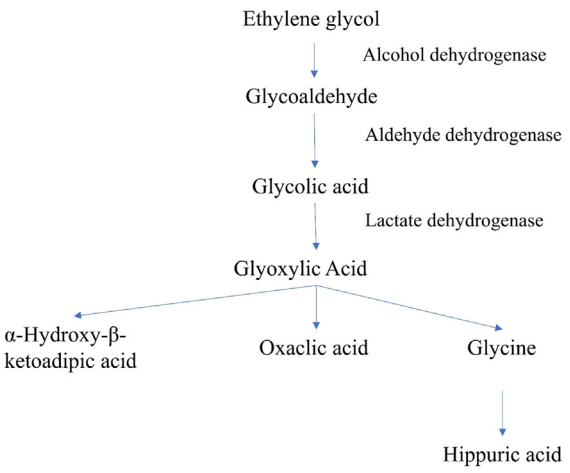

Figure 1. Metabolism of ethylene glycol.

During his first presentation to our hospital, his physical examination was notable for a heart rate of 121 beats/min and mild right lower quadrant tenderness without rebound or guarding. Laboratory evaluation revealed a whole blood lactate of >20.0 mmol/L and a venous blood gas showing pH of 7.36, pCO_2 of 20, pO_2 of 87, and bicarbonate of 11.4. The lactate level (and all other whole blood lactate levels) was measured on a Radiometer ABL 835 FLEX blood gas analyzer (Radiometer America, Brea, California). Creatinine kinase, ethanol, acetaminophen, salicylate, carboxyhemoglobin, and urine drug screen were within normal ranges. Liver function tests and serum creatinine were within normal limits.

His anion gap acidosis led to the measurement of toxic alcohols, including an EG level that was found to be elevated at 19 mg/dL. Fomepizole was initiated but was rapidly discontinued in the setting of minimal acidosis with a down-trending EG level. The patient repeatedly denied intentional ingestion and was unable to explain his previous hospitalizations. A social history revealed that his mother prepared many of his meals, but when confronted about this, he assured us that she was "definitely not poisoning me" and he refused police involvement.

The patient was admitted to the intensive care unit (ICU) where he received multiple doses of intravenous hydromorphone and oral oxycodone. Ultimately the patient left against medical advice, refusing a psychiatric examination and insisting that he knew the risks of leaving (kidney stones, renal failure, and possible death, although still denying ingestion).

He presented to the same hospital 1 month later, with encephalopathy, slurred speech, and an unsteady gait. His laboratory values were notable for an EG level of 133 mg/dL and a whole blood lactate level of 19 mmol/L. Fomepizole was initiated and he was admitted to the ICU.

Further review of the patient's records from 3 hospitals demonstrated 8 visits for elevated whole blood lactate (ranging from 7.5–24 mmol/L on presentation), and that his first visit to our hospital represented his sixth hospital admission for the same symptoms (Table 1). All tests were conducted on whole blood. Tests from our hospital on visits 6 and 7 were done on the Radiometer ABL 835 FLEX blood gas analyzer, and serum lactate levels on other machines were not performed.

Serum EG was positive on 5 of 8 of these visits. During most episodes, he was admitted to the ICU and received multiple doses of intravenous opioid pain medications. As his EG was not always detectable on presentation, he had undergone extensive testing for the source of his elevated lactatemia. This included evaluations by nephrology, genetics, and surgery for the purposes of