## G22 Ethylene Glycol Toxicity: Death Before Calcium Oxalate Crystal Deposition

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After attending this presentation, attendees will recognize that Ethylene Glycol (EG) can result in fatal toxicity in the absence of the pathologic clue of crystal deposition in the kidneys or brain.

This presentation will impact the forensic science community by raising the threshold of suspicion of EG toxicity in the absence of birefringent crystals in the kidney, which is currently the "gold standard" marker that triggers chemical analysis of EG.

EG is a key ingredient in automobile antifreeze. It is an odorless, colorless, sweet-tasting liquid that causes intoxication and death if consumed in sufficient amounts. In attempts at suicidal overdoses, its toxicity is expressed in three clinical phases of poisoning according to post-ingestion time. Much like ethanol, the first stage is characterized by direct Central Nervous System (CNS) depression, which occurs shortly after ingestion and lasts for several hours. This period involves drowsiness, disorientation, and confusion; affected individuals may appear drunk. Convulsion, stupor, and coma may develop in the next stage, about 12-24h after ingestion. In this stage, EG metabolites cause severe non-ketotic metabolic acidosis with an elevated anion gap, cardiopulmonary manifestations, and possible multisystem organ failure. In many cases of EG poisoning, the degree of acidosis can be a prognostic factor, but CNS manifestations are also potentially fatal. In the third stage, about 24-72h after ingestion, a well-known pathological feature is the formation of microscopically visible calcium oxalate crystals from the metabolism of EG into oxalic acid and calcium chelated precipitates. It takes approximately 24 hours for signs of renal impairment to occur and for calcium oxalate crystals to appear in the urine.

To date, no confirmed fatal cases of EG toxicity without tissue birefringent crystals have been reported in the literature. EG is not able to be detected by a volatile panel or traditional toxicology screens; as such, analysis typically depends on suspicion and a special request.

Presented are two cases of fatal EG ingestion in which the characteristic birefringent crystals were absent. Case 1 is a 56-year-old White male found in a field with a note on him, suggestive of a suicide note. He had prior expressed suicidal ideation with stated intent to ingest EG. Because of the history of recent suicidal ideation with specific intent, an EG analysis was requested. EG was overwhelmingly positive: 23,296mg/L in urine, 7,974mg/L in peripheral blood, 12,446mg/L in vitreous humor, and 52,175mg/L in stomach contents. No birefringent crystals were seen in the kidney or brain. The cause of death was certified as EG toxicity and the manner of death was suicide.

Case 2 is a 53-year-old White female found in her residence with a suicide note. Her past medical history included bipolar disorder and prior suicide attempts in which she ingested EG, ethanol, acetaminophen, and paroxetine. Autopsy findings were significant for severe stenotic atherosclerosis of the left anterior descending branch of the left coronary artery. Initial toxicology did not detect ethanol or acetaminophen. Paroxetine was within a therapeutic range. The kidney and brain were negative for birefringent crystals. Further analysis for EG was requested because of the history. EG was in excess of 1,113mg/L in urine and vitreous humor, and 937mg/L in heart blood. The cause and manner of death were classified as EG toxicity and suicide, respectively.

Death in massive EG ingestion may be similar to acute ethanol toxicity. Like ethanol, EG has a low volume of distribution, so it is rapidly absorbed with peak concentrations occurring 30-60 minutes after ingestion. EG also increases gamma-aminobutyric acid receptor activity in the CNS, which causes sedative effects including severe confusion, unconsciousness, and respiratory depression creating a respiratory acidosis that precedes the metabolic acidosis caused by accumulation of glycolic acid. Therefore, EG can exert profound toxicity even before its transformation into more toxic metabolites such as glycolate and the hallmark deposition of calcium oxalate crystals.

It is proposed that EG ingestion in sufficient quantities may precipitate death before crystal deposition in the tissues. This study recommends analysis for EG in all cases with a history suspicious for EG ingestion — or in cases with a history suspicious for suicide with negative initial toxicology testing — even in the absence of birefringent crystals.

**Ethylene Glycol, Postmortem, Toxicology**

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