

## A Sweet Poison - An Anti-freeze Ingestion in a Tropical Setting

P Singh<sup>1\*</sup>, T Lim<sup>2</sup>, H Frieslich<sup>2</sup> and J Soderstrom<sup>3</sup>

<sup>1</sup>Children's Critical Care Unit, Gold Coast University Hospital, Queensland, Australia

<sup>2</sup>Department of Pediatric Emergency, Gold Coast University Hospital, Queensland, Australia

<sup>3</sup>Clinical Toxicologist, Department of Emergency, Royal Perth Hospital, Australia

**\*Corresponding Author:** P Singh, Children's Critical Care Unit, Gold Coast University Hospital, Queensland, Australia.

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### Abstract

**Background:** Ethylene Glycol is a colourless, odorless, viscous dihydroxy alcohol with a sweet taste. It is the most widely available glycol commercially and is used as an anti-freeze and coolant in hydraulic fluids and manufacture of low freezing dynamites and resins. It is readily absorbed from the gastrointestinal tract. Timely recognition of Ethylene Glycol ingestion is important to institute specific antidote and to be able to watch for toxin-related potential complications.

**Methods:** Our very unusual case is a 20 month old child who was brought to our emergency department with altered sensorium and ataxia of uncertain aetiology. The Child was previously healthy with no medical concerns. He developed neurological symptoms after milk formula ingestion. On systematic assessment blood tests showed increased anion gap metabolic acidosis with an elevated osmolar gap. Toxicology team was consulted regarding potential cause and management.

**Result:** Very few chemicals produce such a typical blood chemistry of increased anion gap metabolic acidosis with a high osmolar gap. Laboratory results showed raised blood Ethylene Glycol levels. Further questioning of the child carer's revealed accidental use of Ethylene Glycol instead of bottled water to prepare child's milk formula. The child received intravenous Ethyl Alcohol as an antidote, intubated for airway protection and haemodialysis given high Ethyl Glycol levels. The child made a complete recovery with appropriate and timely management.

**Conclusion:** Ethylene Glycol can produce severe central nervous system toxicity, renal toxicity, acid-base disturbance and even death. Appropriate history taking and investigations can channelize towards the right diagnosis, an institution of specific antidote and supportive therapy. Our patient showed complete neurological and renal function recovery even after an extremely toxic dose of Ethylene Glycol.

**Keywords:** Ethylene Glycol Poisoning; Ethyl Alcohol; Metabolic Acidosis; Osmolar Gap

### Abbreviations

EG: Ethylene Glycol; GCS: Glasgow Coma Scale

### Introduction

Ingestion of Ethylene Glycol (EG) is uncommon but its consequences of ingestion are very serious as it can cause considerable morbidity and mortality, if not diagnosed and treated early. Ethylene Glycol is a colorless, odorless, viscous dihydroxy alcohol with a sweet taste.

It is the most widely available glycol commercially and is used as an anti-freeze and coolant in hydraulic fluids, and in the manufacture of low freezing dynamites and resins. It is readily absorbed from the gastrointestinal tract. The maximal blood-concentration is reached within 1 - 4 hours and the half-life is 3 - 8 hours [1]. The metabolites from the breakdown of EG are cell toxins that suppress the oxidative metabolism which causes 3 stages of health effects with large quantity acute ingestion. These are initial central nervous system depression, followed by cardio-pulmonary effects and subsequently renal failure [2].

## Case Report

A 20-month-old girl, weighing 12.7 kg, was brought in by her parents to the Emergency Department with fluctuating drowsiness, irritability and ataxia. She was first noticed to be sleeping more than usual by her mother, who noted that upon waking up, she was "behaving like a drunkard". Upon further history, it was discovered that the girl was accidentally given 200 ml (200g) of 40% Ethylene Glycol that was mixed into her formula the night before by her babysitter. She had mistaken a bottle on the kitchen bench as water. The bottle concerned contained the girl's father's Fog Machine Liquid "Rave", which he unknowingly left on the bench. At the time of arrival to the emergency department, Ingestion had taken place > 4 hrs before presentation to ED. This translated into an extremely toxic exposure as the fatal dose as little as 1 ml/kg (1 g/kg) and the patient had ingested about 20 mls/kg (20 g/kg).

Initial physical examination findings were primarily neurological. These were disorientation, fluctuating sensorium and ataxic gait. Her cardiovascular and respiratory system examinations were normal. However, bradypnea with normal oxygen saturation was noted. Neurologic examination was difficult due to fluctuating sensorium however the ataxic gait was noticeable. The patient's blood chemistry data whilst in emergency department are shown in table 1.

Test	Ref Range	Time post ingestion	
		5 hrs	8 hrs
Serum Ethylene Glycol, mg/L (mmol/L)		7.1	-
<b>Venous blood gases</b>			
pH	(7.32 - 7.42)	7.18	7.04
pCO <sub>2</sub> , mmHg	(38 - 54)	33.6	49.0
HCO <sub>3</sub> <sup>-</sup> , mmol/L	(16 - 30)	12.0	13.0
Anion Gap, mmol/L	(4 - 13)	14.0	13.0
Lactate, mmol/L	(0.5 - 2.2)	28.0	14.2
Lactate Dehydrogenase U/L	(155 - 345)	437	-
Creatinine, umol/L	(< 36)	< 30	-
Osmolar Gap mmol/kg	(< 10)	17	14

**Table 1:** Venous blood gas, serum ethylene glycol and bloods results - 5 and 8 hours post ingestion.

Ethylene Glycol (EG) Poisoning was confirmed by an elevated anion gap metabolic acidosis with a serum Ethylene Glycol level of 7.1 mmol/L (Table 1). 5.5 hours after ingestion, blood gas showed metabolic acidosis had worsened. She was commenced on Intravenous 10% Ethyl alcohol starting at 8 mls/kg loading with a maintenance rate of 2 mls/kg/hr as an antidote. It's the mechanism of action is to block the metabolism of ethylene glycol as it has more affinity for alcohol dehydrogenase. The targeted ethanol level was 22 - 30 mmol/L and the osmolar gap was used as a surrogate marker for this. Hourly blood glucose level monitoring was performed due to the high risk of hypoglycemia. The patient was intubated and ventilated for the fluctuating GCS. She was subsequently transferred to the pediatric intensive care unit and was commenced on hemodialysis to prevent further renal injury also potential injuries to other organs. Ethanol infusion was continued for another 20 hours until ethylene glycol level was 0.6 mmol/L.

Electrolyte disturbances - mainly hypokalemia, hypophosphatemia, hypomagnesemia was treated with enteral and parenteral substitution. Signs of myolysis in the form of elevated creatinine kinase level and a slight increase in LFTs were also noted. She was transferred to general pediatrics ward Day 3 and discharged from the hospital Day 4. A follow-up in the outpatient clinic one month later revealed a full recovery and no signs of developmental consequences from her ingestion or treatment.

### Discussion

As stated, central nervous system depressant effects from the toxic metabolites of EG are usually the first stage of complication in acute ingestion. The subsequent metabolic acidosis and renal failures are caused by oxidative reactions that convert ethylene glycol to glycolaldehyde and then glycolic acid [3,4]. This conversion takes place slowly; hence serum glycolic acid increases with eventual by products of oxalic acid and glycine. Oxalic acid combines with calcium to form deposits of Calcium oxalate crystal in many tissues [2,5].

Prognosis of treatment is highly variable as it is dependent on the amount ingested, the timing of ingestion recognition and medical intervention. A toxic dose of EG requiring medical treatment is variable, though generally considered more than 0.1 ml (0.1g) per kg body weight (ml/kg) of pure substance. This is equivalent to 16 mL (16g) of 50% EG for an 80 kg adult and 4 mL (4g) for a 20 kg Child [6]. The lethal oral dose in humans is estimated to be about 1.4 mL/kg (1.4 g/kg) of pure EG [7].

Central nervous system depression usually manifests as confusion, ataxia, hallucination, slurred speech and reduced GCS - similar to that effect of ethanol intoxication [8]. Most of these symptoms are severe between 6 to 12 hours after ingestion where the toxic metabolites are usually at their maximum concentration. The absence of a strong odor of alcohol in a patient who appears intoxicated would then increase the likelihood of EG ingestion [2,8].

Following central nervous system symptoms, the metabolic acidosis and cardiopulmonary symptoms become more prominent. The patient may experience nausea, vomiting, hyperventilation, muscle spasm, hypocalcemia and possibly seizures. Cardiac failure with hypertension and tachycardia may also ensue. Pneumonitis and pulmonary edema are reported in some cases [9,10].

Renal complications usually evolve within 24 - 72 hours after ingestion. Calcium oxalate formation will cause depositions in the gastrointestinal mucosa, brain, liver, heart, lungs and kidneys if dialysis is not performed early. Oliguric or anuric renal failure is potential serious complication. Failure to commence early medical intervention of severe EG toxicity usually causes fatality within 24 to 36 hours [8,11-14].

### Conclusion

Our case report of accidental ingestion of EG in a child illustrates the danger with the ingestion of this common and widely available product [15]. It also highlights a possible setting in which such poisoning may occur. Subsequent evaluation of the EG containing-product labels in the market may play a part in the prevention of future accidental ingestion. Appropriate history taking, clinical examination and assessment of increased anion gap metabolic acidosis with raised osmolar gap directed towards diagnosis [16]. Confirmation of diagnosis with elevated blood EG levels. The patient involved had ingestion of up to 20 times the toxic level of EG but eventually had a great recovery due to timely diagnosis and treatment.

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