### EG is present in some antifreeze, coolant, brake fluid, and solvent products. Ingestion is potentially lethal. CONSULT A CLINICAL TOXICOLOGIST EARLY

## **Toxicity / Risk Assessment**

Ingestion of >1 mL / kg 100% EG is potentially lethal Manage all deliberate ingestions as potentially lethal Accidental "less than a mouthful" exposures are usually benign Dermal and inhalation exposure does not cause toxicity

# Clinical features:

- Rapidly absorbed: peak concentration 1-4 hours post ingestion
- Metabolized to acids responsible for clinical toxicity
- During the 1-2 hours post EG exposure, the osmol gap (OG) may be high (a normal OG does not exclude exposure), and the anion-gap (AG) and pH will be normal
- As EG is metabolized, the OG $\downarrow$ , pH $\downarrow$  and the AG $\uparrow$
- Co-ingestion of ethanol delays onset of toxicity
- **STAGE 1 (1-12 hours)**: ataxia, slurred speech, drowsiness (similar to ethanol intoxication)

**STAGE 2 (6-24 hours)**: AG<sup>↑</sup>/acidosis,  $\uparrow$ RR  $\uparrow$ HR  $\uparrow$ BP  $\downarrow$ GCS **STAGE 3 (24-72 hours)**: progressive acidosis, ARF,  $\downarrow$ Ca<sup>2+</sup>, seizures, coma, death

Calcium oxalate crystalluria is diagnostic, but present in <50% of cases

Management *More acidosis = worse outcome. Early treatment = good prognosis* Any delay in commencing treatment with an antidote results in more severe toxicity. **Decontamination:** Activated charcoal does not adsorb EG and is not indicated. Laboratory: Obtain U&E/VBG/ethanol/glucose/AG/measured osmolality at the same time. **Calculated osmolarity** = 2[Na<sup>+</sup>] + urea + glucose + 1.25[ethanol] (concentrations in mmol/L) **Osmol Gap (OG) = Measured osmolality - Calculated osmolarity EG concentrations** are generally not readily available; use surrogate markers (pH/AG/OG) **Antidote**: Alcohol dehydrogenase blocker such as **Ethanol** or **Fomepizole (4-MP)** See separate *Ethanol* or *Fomepizole* guideline Indications for discussion with clinical toxicologist for consideration of Rx with an antidote: documented history of ingestion & OG>10 **OR** suspicion of ingestion AND at least 2 of the following: pH <7.30, HCO3 <20, OG >10, urinary oxalate crystals OR EG concentration of > 20 mg/dL **<u>8.4% Sodium Bicarbonate</u>**: correct acidaemia if pH <7.30 (bolus of 1-2 mL/kg 8.4% solution) **Enhanced elimination Intermittent haemodialysis** is the preferred modality. (Discuss with clinical toxicologist) *Indications*: acidosis / ARF / haemodynamic instability (continue until acidosis resolves) - Increase ethanol / 4-MP infusion rate during haemodialysis

<u>**Cofactors</u>**: IV pyridoxine 50 mg q6h & thiamine may help in metabolism to non-toxic metabolites. <u>**Disposition**</u> - Discharge pending mental health assessment if well + normal pH + HCO3 >20 + OG <10 + ethanol is undetectable at least 4-hours post ingestion</u>

#### **POISONS INFORMATION CENTRE: 13 11 26**

Version 4: Published 4/2023. Review 4/2026

## AUSTIN CLINICAL TOXICOLOGY SERVICE GUIDELINE