

Target Audience: Emergency Medicine Residents, Medical Students

Primary Learning Objectives:

1. Recognize signs and symptoms of ethylene glycol toxicity
2. Order appropriate laboratory and radiology studies in ethylene glycol toxicity
3. Recognize and interpret blood gas, anion gap, and osmolal gap in setting of TA ingestion
4. Differentiate the symptoms and signs of ethylene glycol toxicity from those associated with other toxic alcohols e.g. ethanol, methanol, and isopropyl alcohol

Secondary Learning Objectives: detailed technical/behavioral goals, didactic points

1. Perform a mental status evaluation of the altered patient
2. Formulate independent differential diagnosis in setting of leading information from RN
3. Describe the role of bicarbonate for severe acidosis

Critical actions checklist:

1. Obtain appropriate diagnostics
2. Protect the patient's airway
3. Start intravenous fluid resuscitation
4. Initiate serum alkalinization
5. Initiate alcohol dehydrogenase blockade
6. Consult Poison Center/Toxicology
7. Get Nephrology Consultation for hemodialysis

Environment:

1. Room Set Up – ED acute care area
 - a. Manikin Set Up – Mid or high fidelity simulator, simulated sweat if available
 - b. Airway equipment, Sodium Bicarbonate, Nasogastric tube, Activated charcoal, IV fluid, norepinephrine, Simulated naloxone, Simulate RSI medications (etomidate, succinylcholine)
2. Distractors – ED noise

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CASE SUMMARY

SYNOPSIS OF HISTORY/ Scenario Background

The setting is an urban emergency department.

This is the case of a 2.5-year-old male toddler who presents to the ED with an accidental ingestion of ethylene glycol. The child was home as the father was watching him. The father was changing the oil on his car. The child drank antifreeze without the father knowing this. The child then appeared sleepy and ready for a nap. The child then went down for his nap but later on could not be aroused or awakened. The father noticed emesis on the child's clothes and in the bed. Concerned, he brought the child to the ED.

In the ED, the child is somnolent with poor airway protection and requires intubation. Clinical presentation and laboratory data suggest ethylene glycol poisoning. The child requires appropriate treatment with fomepizole and ICU admission

SYNOPSIS OF PHYSICAL

The patient is initially stuporous and delirious, with a diminished gag reflex, mumbling incoherently, and making random groans.

The patient's airway is not optimally protected at the time of his arrival to the ED.

The patient is tachycardic and hypotensive.

CRITICAL ACTIONS

1. Obtain appropriate diagnostics (must include the initial point-of-care glucose)

Order appropriate diagnostics. **At a minimum**, this must include: initial point-of-care glucose, BMP, pH (venous or arterial blood gas is sufficient), serum osmolality, urinalysis with microscopy, toxic alcohol levels (ethylene glycol, methanol, isopropyl alcohol, although these will be made unavailable), and serum calcium or ionized calcium (patient may be hypocalcemic because oxalate binding of calcium). The participant should demonstrate recognition of the metabolic acidosis and, ideally, the osmolal gap.

Cueing Guideline: The nurse can ask if the doctor would like to do order any diagnostic tests. The nurse can state that, if the participant asks for toxic alcohol levels, those tests will be sent, but results will not be immediately available.

2. Protect the patient's airway

Protect the patient's airway. The participant must recognize the need for definitive airway protection in the pediatric patient with a diminished gag reflex and prior emesis. The resident must demonstrate the ability to competently direct definitive airway protection for the pediatric patient, especially in the context of a toxin-associated metabolic acidosis.

Cueing Guideline: The nurse should ask if any other tests could be done in the setting of a negative ethanol level to help explain the altered mental status.

3. Start intravenous fluid resuscitation

Bilateral large bore IV access should be established as the patient is hypotensive and tachycardic. 20 mL/kg boluses of crystalloid fluid should be given.

Cueing Guideline: The nurse may say, "We have a line in place. Would you like any fluids?" Alternatively, the nurse can mention the tachycardia. If not done, the patient will become more tachycardic (140s) and BP may drop to 75/43.

4. Initiate serum alkalinization

Recognize severe metabolic acidosis. Give 1-2 mEq/kg bolus of sodium bicarbonate and start a bicarbonate infusion (must request consultation with the clinical pharmacist for further direction on this intervention, and the infusion will not be available during the remainder of this case).

Cueing Guideline: Nursing can note worsening hypotension if pH correction is not addressed. Reemphasis of blood gas or BMP results can be made.

5. Initiate alcohol dehydrogenase blockade

15 mg/kg of fomepizole should be ordered.

Cueing Guideline: If not done, patient will become increasingly tachypneic and altered. If blood gas repeated, metabolic acidosis will be more profound. Acidosis will be refractory to sodium bicarbonate. Ultimately, patient will seize and die.

6. Consult Poison Center/Toxicologist

The local Poison Center or Toxicology service should be consulted for further management recommendations (addition of pyridoxine and thiamine, dosing of fomepizole, criteria for initiation of hemodialysis, etc.)

Cueing Guideline: The nurse can ask the doctor if the Poison Center/Toxicologist have been called yet.

7. Get Nephrology Consultation for hemodialysis

Due to high osmolal gap and severe metabolic acidosis, the patient requires hemodialysis for removal of ethylene glycol and metabolites and correction of pH.

Cueing Guideline: patient will continue to decompensate with hypotension, progressive obtundation, and seizure refractory to bicarbonate or other measures.

Critical Actions Checklist¹

Resident Name								
Case Description								
Skills measured <small>Core competencies: PC Patient care, MK Medical knowledge, IC Interpersonal and communication skills P Professionalism, PB Practice-based learning and improvement SB Systems-based practice</small>	Very Unacceptable		Unacceptable		Acceptable		Very Acceptable	
Data Acquisition (D) PC MK I	1	2	3	4	5	6	7	8
Problem Solving (S) PC MK PB	1	2	3	4	5	6	7	8
Patient Management (M) PC MK IC P PB SB	1	2	3	4	5	6	7	8
Resource Utilization (R) PC PB SB	1	2	3	4	5	6	7	8
Health Care Provided (H) PC SB	1	2	3	4	5	6	7	8
Interpersonal Relations (I) IC P	1	2	3	4	5	6	7	8
Comprehension of Pathophysiology (P) MK PB	1	2	3	4	5	6	7	8
Clinical Competence (C) PC MK IC P PB SB	1	2	3	4	5	6	7	8
Critical Actions								
Yes	No				Comments:			
		Obtain appropriate diagnostics (POC glucose)						
		Protect the patient's airway						
		Start intravenous fluid resuscitation						
		Initiate serum alkalization						
		Initiate alcohol dehydrogenase blockade						
		Consult Poison Center/Toxicology						
		Get Nephrology Consultation for hemodialysis						
					Yes	No	Dangerous actions	

¹ Modified ABEM Oral Certification Examination checklist and scoresheet

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HISTORY

You are called to see a new patient: this is a 2.5 year old infant boy who presents to the ED with an accidental ingestion of ethylene glycol.

Onset of Symptoms: Hours

Background Info: Child was home with Dad watching him. Dad was changing the oil on his car. The child drank antifreeze without the Dad knowing this. The child then appeared sleep and ready for a nap. The child then went down for his nap but later could not be woken up. Father also noticed emesis on the child's clothes. No trauma, no prodromal symptoms/illness

NOTE: Father very anxious but not distracting to scenario; if asked, the father will reveal there is antifreeze in the garage.

Chief Complaint: Altered mental status, evidence of vomiting

Past Medical Hx: None

Past Surgical Hx: None

Family Med Hx: Non-contributory (no family history of seizures)

Social Hx: Child lives with parents
No siblings
No daycare
Mother away on business trip

ROS: Patient is unable to answer. Father reports that patient has previously been well and in his usual state of health.

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CASE CONTINUATION

**Vital Signs: BP: 84/60 mmHg P: 120/minute R: 24/minute T: 37.6C (99.7F)
POx: 94% (FiO₂=1 with non-rebreather mask at the beginning of the case)**

Primary Survey

- Airway – Child groans and slurs speech when stimulated; diminished gag reflex
- Breathing – Marked tachypnea, increased work of breathing, 94% SpO₂
- Circulation – Tachycardia (120's), SBP 80's, brachial and femoral pulses diminished
- Disability – Child will localize to pain, won't open eyes, won't follow commands, no verbal communication (groans)
- Exposure – No trauma or rashes

Required Actions Over Time 0-2 Minutes

- Move patient to critical care or acute care area of the Emergency Department
- Establish safety net (IV, oxygen, cardiac monitor, two large bore IVs, draw blood for labs, blood gas)
- A/B – Provide supplemental oxygen. Protect airway (aspiration precautions). Prepare for endotracheal intubation in context of diminished gag reflex, prior emesis and metabolic acidosis
- C – Cardiac monitor; 20 mL/kg IV crystalloid bolus for presumed volume depletion
- D – Finger stick glucose = 90 mg/dL; GCS = 10; labs are sent

Branch Point

- **IF NO INTERVENTIONS ARE PERFORMED**, then the patient becomes more hypotensive (78/45 mmHg), tachycardic (138/minute), and hypoxic (SaO₂ lowers to 85%).
- **IF AT ANY TIME DURING THE CASE, ENDOTRACHEAL INTUBATION IS PERFORMED AND VENTILATOR SETTINGS ARE NOT ADJUSTED TO MAINTAIN THE RESPIRATORY RATE AND PARAMETERS AT LEVELS APPROPRIATE IN THE CONTEXT OF SEVERE METABOLIC ACIDOSIS (e.g., INCREASED RATE, ADEQUATE I:E RATIO)**, the patient will suddenly develop worsening acidosis and sudden cardiac arrest will develop.

CASE CONTINUATION

General Appearance: Male toddler, appears to be sleeping, with evidence of vomiting (earlier, prior to arrival) and a diminished gag reflex

Vital Signs: BP: 85/57 mmHg P: 127/minute R: 37/minute T: 35.9C (96.6F)
POx: 94% (FiO₂=0.21) Weight = 13 kg

Head: Normocephalic and atraumatic

Eyes: PERRL, pupils 4 mm bilaterally

Ears: TM's normal.

Mouth: Dry mucous membranes, diminished gag reflex

Neck: Trachea midline, supple

Skin: Warm, dry, no rash

Chest: Notable tachypnea and hyperpnea. No trauma. Good chest rise

Lungs: Clear, equal bilaterally, tachypneic and hyperpneic (if the patient has been intubated by this time, mechanical breath sounds)

Heart: Tachycardic, S1 S2, no murmurs

Back: Normal

Abdomen: Soft, non-tender, no signs of trauma, no rebound/guarding

Extremities: No signs of trauma, no edema, radial pulses weak, central pulses strong

Rectal: Normal tone, guaiac negative

Neurological: Diminished gag reflex. If prior to sedation and intubation, groans to pain, pushes examiner away. Moves all 4 extremities though follows commands poorly. No hyperreflexia or clonus

Mental Status: Drowsy, obtunded, minimally responsive

Required Actions over Time 2-4 Minutes

- Patient should be intubated by this time
- Further resuscitation with IV NS 20 mL/kg boluses
- Obtain and interpret lab results including blood gas, BMP, and osmolality
- Consider serum alkalinization with boluses of sodium bicarbonate
- May order non-contrast head CT but cannot obtain until patient is more stable
- May place a Foley catheter (drain 30 mL of urine)

Branch Points

- **IF NO INTERVENTIONS ARE PERFORMED**, then the patient continues to worsen, becoming even more hypotensive and tachycardic.

Required Actions over Time 4-6 Minutes

- SBP remains in 90s mmHg
- Serum/urine toxicology diagnostic tests are all undetectable.
- Microscopy on UA will have calcium oxalate crystals
- If urine is fluoresced with Wood's lamp, it will fluoresce
- Participant must now recognize presence of toxic alcohol
- Fomepizole (15 mg/kg) administration should be initiated by this time

Branch Points

- **IF ALCOHOL DEHYDROGENASE BLOCKADE IS NOT INITIATED BY THIS TIME**, then the patient's acidosis will continue to worsen, mental status will deteriorate, and respirations will worsen (this may be demonstrated by "overbreathing" on the ventilator if sedation is not optimized, or by worsening acidosis if the patient is appropriately sedated while on the ventilator).

Required Actions over the Remainder of the Case

- Arrange for PICU admission
- Contact PC/Toxicology for further recommendations
- Contact Nephrology for hemodialysis

Branch Points

- **IF NEPHROLOGY IS NOT CONSULTED**, then the patient will develop hypotension and acidosis refractory to fluid resuscitation and sodium bicarbonate administration

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STIMULUS INVENTORY

- #1 Complete blood count
- #2 Basic metabolic panel
- #3 Urinalysis
- #4 Liver function tests
- #5 Venous blood gas
- #6 Point-of-care glucose
- #7 Toxicology
- #8 Initial CXR
- #9 Repeat CXR
- #10 ECG
- #11 Lactate

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LAB DATA & IMAGING RESULTS

Stimulus #1	
Complete Blood Count (CBC)	
WBC	20,400/mm ³
Hemoglobin	15.2 g/dL
Hematocrit	46%
Platelets	79,000/mm ³
Differential	
PMNLs	45%
Lymphocytes	55%
Monocytes	2%
Eosinophils	1%

Stimulus #2 Basic Metabolic Profile (BMP)		
	INITIAL	REPEAT (no bicarb given)
Sodium	142 mEq/L	142 mEq/L
Potassium	3.4 mEq/L	3.9 mEq/L
Chloride	108 mEq/L	100 mEq/L
Bicarbonate	9 mEq/L	4 mEq/L
Glucose	92 mg/dL	90 mg/dL
BUN	28 mg/dL	12 mg/dL
Creatinine	1.7 mg/dL	1 mg/dL
Calcium	7 mg/dL	

Stimulus #3	
Urinalysis	
Color	Yellow
Specific gravity	1.030
Glucose	Negative
Protein	Negative
Ketones	1+
Leuk. Esterase	Negative
Nitrites	Negative
WBC	0/hpf
RBC	0/hpf
Microscopic	Calcium oxalate crystals

Stimulus #4	
Liver Function Tests	
AST	57 U/L
ALT	48 U/L
Alk Phos	110 U/L
Total Bilirubin	1.1 mg/dL
Direct Bilirubin	0.2 mg/dL
Albumin	2.9 mg/dL
Protein	7 g/dL

Stimulus #5	
Initial Venous Blood Gas	
pH	7.20
pCO ₂	24 mm Hg
pO ₂	52 mm Hg
HCO ₃ base deficit	9 mEq/L -17

Stimulus #6	
Troponin	
Value	< 0.01 ng/mL

Stimulus #7	
Toxicology	
Salicylate	Undetectable
Acetaminophen	Undetectable
Ethanol	Undetectable
Urine drug screen	
Amphetamines	Negative
Benzodiazepines	Negative
Cocaine	Negative
Opiates	Negative
TCA's	Negative
THC	Negative

Stimulus #8	
CXR	
	Normal

Stimulus #9	
CT head	
	Normal

Stimulus #10	
AXR	
	Normal

Stimulus #10	
ECG	
	NSR

Stimulus #11	
Venous Blood Gas (no bicarb given or HD)	
pH	7.02
pCO ₂	16 mm Hg
pO ₂	60 mm Hg
HCO ₃ base deficit	4 mEq/L -24

Stimulus #1**Complete Blood Count (CBC)**

WBC	20,400/mm ³
Hemoglobin	15.2 g/dL
Hematocrit	46%
Platelets	79,000/mm ³
Differential	
PMNLs	45%
Lymphocytes	55%
Monocytes	2%
Eosinophils	1%

Stimulus #2A**Initial Basic Metabolic Profile (BMP)**

Sodium	142 mEq/L
Potassium	3.4 mEq/L
Chloride	108 mEq/L
Bicarbonate	9 mEq/L
Glucose	92 mg/dL
BUN	28 mg/dL
Creatinine	1.7 mg/dL
Calcium	7 mg/dL

Stimulus #2B**Repeat Basic Metabolic Profile (BMP)**

Sodium	142 mEq/L
Potassium	3.9 mEq/L
Chloride	100 mEq/L
Bicarbonate	4 mEq/L
Glucose	90 mg/dL
BUN	12 mg/dL
Creatinine	1 mg/dL

Stimulus #3**Urinalysis**

Color	Yellow
Specific gravity	1.030
Glucose	Negative
Protein	Negative
Ketones	1+
Leuk. Esterase	Negative
Nitrites	Negative
WBC	0/hpf
RBC	0/hpf
Microscopic	Calcium oxalate crystals

Stimulus #4**Liver Function Tests**

AST	57 U/L
ALT	48 U/L
Alk Phos	110 U/L
Total Bilirubin	1.1 mg/dL
Direct Bilirubin	0.2 mg/dL
Albumin	2.9 mg/dL
Protein	7 g/dL

Stimulus #5

Initial Venous Blood Gas

pH	7.20
pCO ₂	24 mm Hg
pO ₂	52 mm Hg
HCO ₃ base deficit	9 mEq/L -17

Stimulus #6

Troponin

Value	< 0.01 ng/mL
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Stimulus #7**Toxicology**

Salicylate	Undetectable
Acetaminophen	Undetectable
Ethanol	Undetectable

Urine drug screen

Amphetamines	Negative
Benzodiazepines	Negative
Cocaine	Negative
Opiates	Negative
TCAs	Negative
THC	Negative

Stimulus #8	
CXR	Normal

Stimulus #9	
CT head	Normal

Stimulus #10	
AXR	Normal

Stimulus #11	
Venous Blood Gas	
pH	7.02
pCO ₂	16 mm Hg
pO ₂	60 mm Hg
HCO ₃ base deficit	4 mEq/L -24

Debriefing Materials – Ethylene Glycol Toxicity

Educational Goals: review the key principles of managing ethylene glycol toxicity and the other toxic alcohols.

Debriefing Approach:

- I. Decompress – “How did you feel it went?” (not asking for details; just a chance for the resident to decompress, decrease anxiety/energy level to be more open to learning/retaining knowledge)
- II. Core Medical Knowledge (instructor covers details of scenario and objectives)
- III. Advocacy/Inquiry
 - a. As an instructor “advocate” for your point of view/observations of resident actions
 - b. Inquire with an open mind to see why the resident acted the way they did – use this response as a springboard to determine what exactly to teach (e.g., was the resident’s poor performance due to missing core medical knowledge or is it a lack of communication skills with the rest of the team?)
- IV. Plus/Delta
 - a. Plus – tell the resident what went well
 - b. Delta – Tell the resident what she could change for next time

Sources of Exposure:

- Engine coolants (antifreeze), plastics industry (polymer precursor)
- Sweet in taste (“glycol”)
- Bittering agents may be added to deter exploratory or pet consumption
- Fluorescent dye may be added (to aid vehicle technician in detecting radiator leaks)
- Note: gas line antifreeze typically contains methanol, rather than ethylene glycol

Pathophysiology:

- The metabolites of ethylene glycol are thought to be the primary toxic entities
- Ethylene glycol is metabolized by alcohol and acetaldehyde dehydrogenases in stepwise fashion to glycoaldehyde and glycolic acid, respectively. Glycolic acid is similarly oxidized to oxalic acid in a two-step fashion
 - Each of these oxidation steps is coupled with the reduction of NAD^+ to NADH
- The generation of these unmeasured organic acids (glycolic and oxalic acids) results in metabolic acidosis and elevation of the anion gap
 - Unchecked metabolic acidosis results in inappropriate vasodilation, myocardial suppression, and, ultimately, combined vasoplegic and cardiogenic shock
- Primary specific end-organ toxicity of EG consumption is nephrotoxicity
 - Oxalate combines with calcium forming calcium oxalate monohydrate crystals, which precipitate in the proximal tubules and are often clearly evident on biopsy/post. Additionally, detection on microscopic UA may be of diagnostic assistance (see below).
 - Biopsy/post also may show damage to glomerular basement membranes – the etiology of this is uncertain

- Myocardial injury may occur from direct deposition of calcium oxalate, alternatively, oxalate may “sequester” free calcium resulting in hypocalcemia, QT prolongation, and cardiac dysrhythmia
- Cerebral edema, elevated ICP, and papilledema are reported, as well as delayed central nervous system effects – commonly isolated or multiple cranial nerve palsies – presumably due to direct injury from calcium oxalate
 - Intracranial hemorrhage involving globus pallidus also described

Severity of Ingestion:

- GI absorption is rapid and complete
- Serum EG levels (or, by surrogate, osmolal gap) and degree of metabolic acidosis are more indicative of toxicity than the reported amount ingested
- All possible ingestions should be evaluated in a healthcare setting
- Treatable EG levels are possible after ingestion of only a “mouthful” of antifreeze
- It is debatable about whether to empirically block alcohol dehydrogenase based on history alone in the absence of immediately available laboratory confirmation
- Patients may present as if inebriated (as in this case)

Organ System Effects:

- Renal:
 - Primary organ(s) of injury
 - Kidney injury may be seen early after ingestion, but creatinine can be expected to rise 2-3 days after injury
 - Early signs of kidney injury, may, in fact, be due to hypoperfusion
 - Degree of injury may vary from proteinuria/hematuria with mild BUN elevation to prolonged anuria and azotemia
 - In cases where hemodialysis is continued for renal failure, renal function is expected to recover over the course of weeks to months
- Pulmonary:
 - Direct pulmonary pathology would be unusual
 - Aspiration pneumonitis should be expected
- Cardiovascular:
 - Prolongation of the PR and QT intervals due to systemic hypocalcemia with subsequent cardiac dysrhythmia
 - Cardiogenic shock due to metabolic acidosis +/- direct oxalate myocardial injury
- Neurologic:
 - As with most alcohols, EG is inebriating leading to symptoms anywhere from euphoria, to ataxia and dysarthria, to somnolence, to coma
 - Delayed cerebral edema is reported, such patients would have coma or persistent alteration in mental status
 - Cranial nerve palsies may occur – these are typically delayed in nature, with one report of an abducens palsy at 9 days post ingestion
 - Basal ganglia injury and acquired Parkinson-like symptoms may occur
 - Globus pallidus hemorrhage may occur

- Other:
 - Multisystem organ toxicity and failure secondary to hypotension and shock
 - Shock is multifactorial and initially emphasis should be placed on volume resuscitation as patients may be multiple liters down due to nausea and vomiting, as well as diuresis due to osmotic effects

Diagnostic Testing:

- Ethylene glycol level
 - In most non-tertiary care centers serum ethylene glycol levels are not readily available
 - Ethanol, methanol, propylene glycol, and isopropyl alcohol levels should also be obtained simultaneously unless the actual ingested product is strongly verified (e.g. “antifreeze” may actually be gas line antifreeze which contains methanol)
- Osmolality
 - Available at most centers, however, the method of obtaining this lab may be critical
 - The optimal method is freezing point depression; the alternative method – boiling point elevation – risks falsely reporting below the actual osmolality due to volatility of some dissolved xenobiotics, including ethanol and methanol
- Chemistry Panel
 - Should be obtained SIMULTANEOUSLY with the osmolality
 - Repeat as needed to follow anion gap and electrolytes, particularly potassium if sodium bicarbonate being administered
- Ethanol Level
 - Should be obtained SIMULTANEOUSLY with the osmolality
- Calculation of osmolal gap:
 - $\text{Gap} = (\text{Measured [Osms]}) - (2 \times [\text{Na}^+] + [\text{BUN}]/2.8 + [\text{Glu}]/18 + [\text{EtOH}]/4.6)$
 - Assumes that [BUN], [Glu], and [EtOH] reported in mg/dL
 - Doubling of $[\text{Na}^+]$ accounts for sodium and all associated anions (Cl^- , HCO_3^- , albumin, etc.)
 - The whole point of the conversions of BUN, glucose, and ethanol are to convert from mg/dL to mmol/L = mOsm/L
 - “Normal” osmolal gap generally considered 0-10 mOsm/L
- Salicylate and acetaminophen levels in cases of intentional ingestions
- ECG
 - Watch for PR and QT prolongation (hypocalcemia)
- Blood gas + lactic acid
 - Venous blood gas will suffice unless hypoxia present
 - Repeat as needed to ensure pH correction
- Calcium/ionized calcium levels
- Urinalysis
 - Presence of calcium oxalate crystals on microscopy may suggest EG ingestion
 - Consider use of Wood’s Lamp to look for urine fluorescence
 - Absence of fluorescence does not rule out EG ingestion as not all antifreeze products contain fluorescent dye
 - Dip gauze in urine and fluoresce, do not fluoresce in Foley bag as the plastic itself may appear to fluoresce
- Computed tomography if altered mental status not otherwise explained

- Relationship between osmolal gap and anion gap
 - Early after ingestion, osmolal gap will be elevated, while anion gap will be nil
 - Over time (hours), osmolal gap will fall as anion gap rises – as EG is metabolized to acid metabolites, their presence in serum becomes accounted for by sodium in the osmolal gap calculation (while bicarb and chloride fall)
 - Late ingestions of ethylene glycol may have not have a significant osmolal gap at all

Treatment:

- Decontamination
 - NG aspiration can be considered if presentation is immediate after ingestion
 - Due to rapid absorption, no role for AC, NGL, WBI
- Volume resuscitation with crystalloid
 - If hypotensive, 4-6 L over 1-2 hours
- Correction of pH
 - 100-200 mEq sodium bicarbonate boluses to restore pH to at least 7.2-7.3
 - Start sodium bicarbonate infusion at 200-250 cc/hr (5% dextrose in water + 150 mEq sodium bicarbonate, consider addition of 40 mEq KCl)
 - pH refractory to correction is an absolute indication for hemodialysis
- Alcohol dehydrogenase blockade
 - Indications:
 - Documented EG ingestion and EG level >20 mg/dL or osmolal gap >10 mOsm/L
 - OR
 - Suspected ingestion and 3 of the following:
 - pH < 7.3
 - Serum bicarbonate <20 mmol/L
 - Osmolal gap >10 mOsm/L
 - Oxalate crystalluria
 - Fomepizole (4-methylpyrazole): 15 mg/kg (maintenance is 10 mg/kg q12hrs x4 doses, then 15 mg/kg q12hrs)
 - Ideally, ensure an ethanol level is available prior to initiation of 4-MP (see below)
 - If no 4-MP available, ethanol can be administered
 - PO may be best due to ease of availability and dosing
 - IV ethanol increasingly rare and approximately now cost-equivalent to 4-MP
 - 4-5 shots of hard liquor via NG tube for the average adult.
 - IV ethanol is not recommended due to difficulty of dosing, need for central access
 - Goal serum ethanol is 100 mg/dL which should effectively block metabolism of ethylene glycol
- Hemodialysis
 - Indications:
 - Metabolic acidosis (pH <7.2-7.3) that is refractory to standard measures
 - Hemodynamic instability refractory to standard measures
 - Acute kidney injury or renal failure

- If initiated, HD should be continued until acidosis corrected and osmolal gap closed OR [EG] < 20 mg/dL
- IHD is preferred over CRRT
- Patients without acidosis and normal kidney function can be managed with 4-MP alone
- Vitamin repletion
 - Thiamine and pyridoxine may promote metabolism of toxic metabolites to nontoxic entities, and administration is considered benign
 - Thiamine 100 mg IV q8hrs, pyridoxine 50 mg IV q6hrs
- Normalize magnesium
- Supplement calcium only for symptomatic hypocalcemia or seizures
- Vasopressors: titrate to improving markers of perfusion: mean arterial pressure, capillary refill, lactate, urine output
 - Norepinephrine 1st line: start at 0.1 mcg/kg/min
 - Epinephrine 2nd line: start at 0.1 mcg/kg/min
 - Vasopressin 3rd line: 0.4 units/min

Consultations:

- Regional poison center or a local medical toxicologist for additional information and patient care recommendations
- Nephrology if meeting or potentially meeting hemodialysis criteria

Disposition:

- All potential ingestions should be evaluated in an emergency care setting
- Admit patients with major signs and symptoms or requiring HD to an ICU
- **Note: concurrent ingestion of ethanol may delay the development of toxicity**
- Consult psychiatric service personnel for stabilized patients with intentional overdose.

Take-Home Points:

- Obtain SIMULTANEOUS chemistry panel, ethanol level, and osmolality
- EG level or osmolality and metabolic acidosis are better predictors of toxicity than the reported amount ingested
- Aggressive crystalloid resuscitation is a must – patients may be SEVERELY volume depleted secondary to N/V as well as osmotic diuresis
- Antidote is fomepizole (4-methylpyrazole)
 - PO ethanol can be substituted while awaiting 4-MP or transferring to center with 4-MP
- Hemodialysis is for removal of toxic metabolites.
 - Indications: metabolic acidosis, kidney injury, signs of end organ injury (hypotension, shock, etc.)
 - Continue HD until acidosis corrected AND osmolal gap closed or [EG] < 20 mg/dL
- Assess serial blood gases and chemistries to titrate therapy

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