Mind the Gap: A Look at Toxic Alcohols

https://commons.wikimedia.org/wiki/File:Mind_the_gap_2.JPG#/media/File:Mind_the_gap_2.JPG

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EmergNP 2025
AMERICAN ACADEMY OF EMERGENCY NURSE PRACTITIONERS
NATIONAL CONFERENCE

My Approach to the Possible Toxic Alcohol Ingestion

- Assessment to include:
  - Alcohol use, last drink, hx sz, rehab, other drugs
- Initial lab
- Think about thiamine
- What is my index of suspicion
  - History or witnessed ingestion
  - Elevated anion gap
  - Clinical picture that is suggestive
- Low or High Index

Labs to Consider

- CBC, Glucose, CMP
- Electrolytes- especially Mg/Ca/Phos
- Lactic acid, Beta-hydroxybutyrate, Anion gap
- Ethanol, methanol, ethylene glycol
  - May be called- volatile alcohol panel

Common Calculations

- **Anion Gap** = Na – (Cl + HCO3)

  Abnormal >12 Calculated

- **Osmolality** (using SI units) = (Na x 2) + Urea + Glucose + (Ethanol (mmol/L) x 1.25)

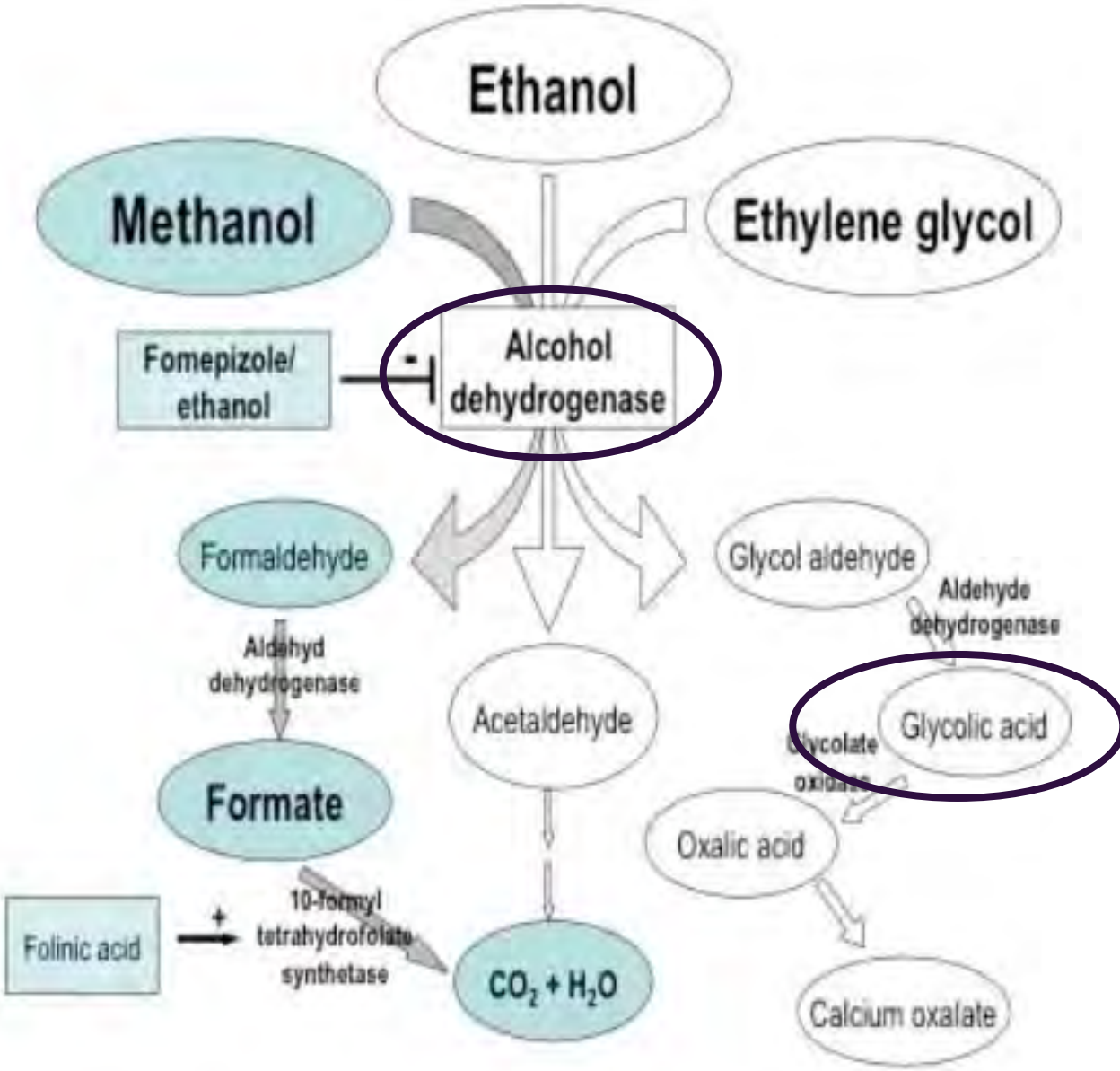
- **Osmolar Gap** = Measured Osmolality – Calculated Osmolality

  Abnormal >10

MUDPILES

- **M**: Methanol ingestion
- **U:** Uremic acidosis
- **D:** Diabetic ketoacidosis
- **P:** Propylene glycol
- **I:** Isoniazid or Iron tablets
- **L:** Lactic acidosis
- **E:** Ethylene glycol ingestion
- **S:** Salicylate ingestion

Toxic Alcohols



https://www.emdocs.net/em-cases-toxic-alcohols-minding-the-gap/

Proplylene Glycol

https://www.pfizerhospitalus.com/products/diazepam

- Propylene glycol used as a diluent in many pharmaceuticals-potential iatrogenic toxicity from continuous or large-volume medication exposures
- Metabolized to lactate through pathways similar to other toxic alcohols, resulting in metabolic acidosis
- Can increase with AKI
- Resolution occurs within 24 hours of removal of the offending containing agent
- Drugs to worry about:
  - Diazepam, lorazepam, etomidate, phenobarb, NTG, & phenytoin

Found a big bottle of antifreeze in his carry-on luggage

POCT BLOOD GASES	01/02/25	01/03/25 0555	01/03/25 0559
POC pH	7.07 ⌄⌄	19.2*	--
POC pCO2	10.2 ⌄⌄	231	--
POC pO2	158 ⌃	--	--
POC Base Excess	-24.5 ⌄	--	4.4
POC HCO3	2.9 ⌄	--	135*
POC O2 Saturation	98.8 ⌃	--	14.0
POC Carboxyhemoglobin	1.0	--	3.00*
POC Methemoglobin	0.9	--	8.5
POC Oxyhemoglobin	96.9 ⌃	--	25
Patient Temperature	37.0	--	20
Sample	ART	--	--
PS	0	--	10
POC TRIGGER EDI	0	--	--
POC BLENDER FIO2	0	--	--
POC SWEEP	NONE	--	--

Denke Personal

Ethylene Glycol

Etiology

Widely used as an automotive antifreeze & a precursor to polymers

Odorless, colorless, syrupy, & may be sweet-tasting

Found in: auto antifreeze, cleaners windshield wiper fluid, solvents

Causes brain, lungs liver & kidney damage

Some Statistics

- Better supportive care, ↑ awareness of tx priorities, widespread use of antidotes, & greater availability of ECTR, mortality from intentional ethylene glycol poisoning has steadily ↓
- Mortality > 80% before 1960
- Decreased to 30–40% in the 1970s & 1980s
- Trend has continued to improve during the 1990s with mortality declining to < 10 % today
- But when ECTR and antidotes are not available high mortality is still seen

Ghannoum et a., 2023

Deaths of 70 Children in Gambia

"The World Health Organization (WHO) linked this recent incident to an Indian manufacturer, saying that the cough syrups contained "unacceptable amounts of diethylene glycol and ethylene glycol"

https://www.europeanpharmaceuticalreview.com/article/177370/diethylene-glycol-ethylene-glycol-adulteration/

Pathophysiology

- Ethylene glycol metabolized into glycolate and oxalate
- See anion gap metabolic acidosis, neurotoxicity, AKI, & death
- Readily absorbed following ingestion & subsequently distributed in the body water
- Primarily undergoes metabolism in the liver & kidneys
- Metabolites are excreted primarily in the urine & small quantities of ethylene glycol may be excreted unchanged

Kinetics & Metabolism

Absorption

- Ingestion or dermal & then absorbed via the GI tract

Distribution

- Rapid, with peak concentration 1-4 hours after ingestion

Metabolism

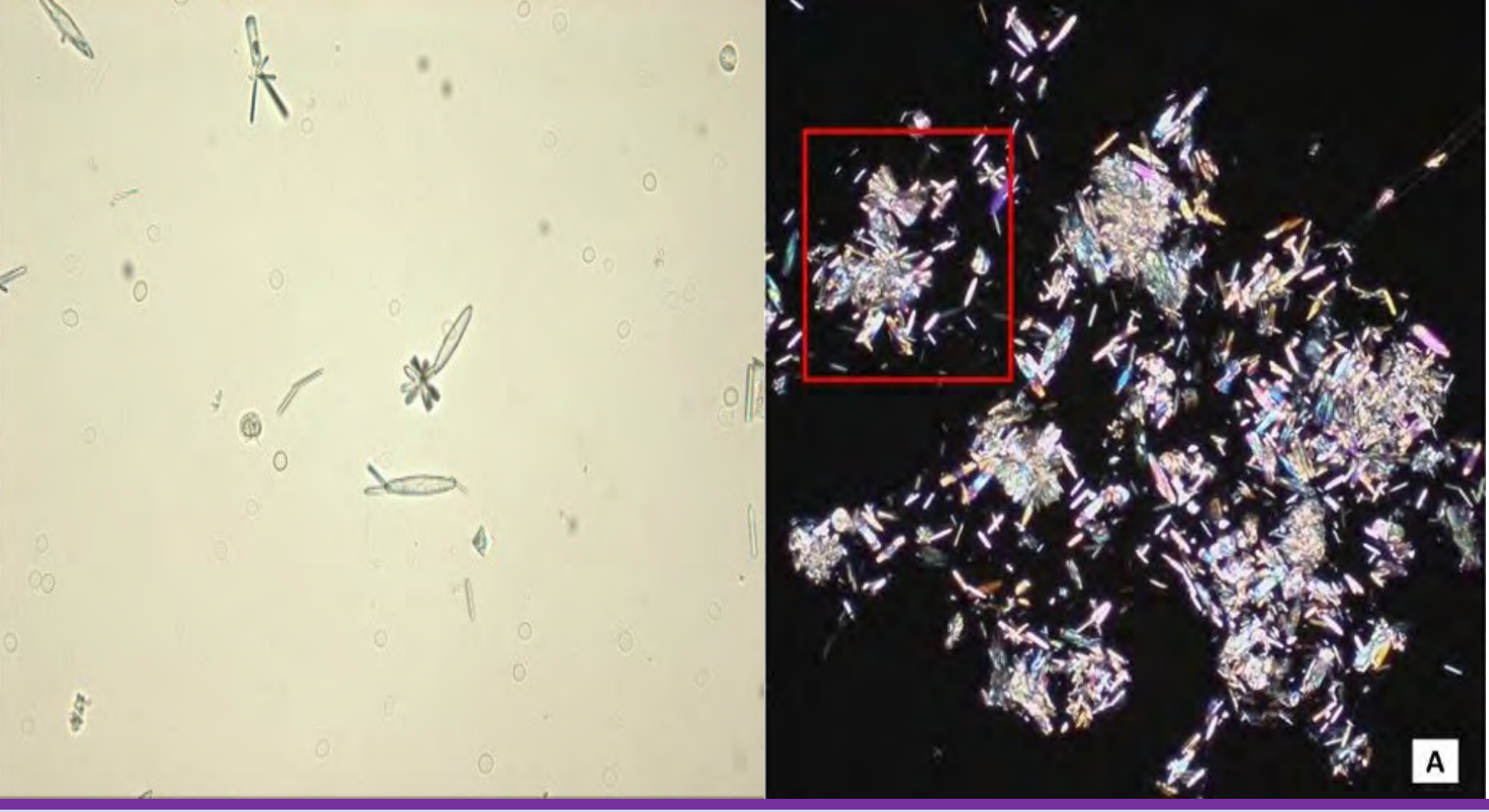
- Liver & kidneys sites of metabolism
- Affinity of alcohol dehydrogenase for ethanol > ethylene glycol

Excretion

- Urine- oxalic acid excreted in the urine
- 20% may be excreted unchanged by the kidneys
- Elimination ½ life ≈ 2.5- 8.4 hours

Acute Clinical Features

- 1st symptom similar to being drunk
- Profound anion gap metabolic acidosis, ↑osmolar gap & ethylene glycol level
- May see CNS depression with
  - Dizziness, agitation, nystagmus, nausea/vomiting
- CV – usually see ≈12 hr post ingestion
  - Hyperventilation/tachypnea, tachycardia, cyanosis & HTN
- Renal- develops 24-72 hours
  - Oliguria, anuria, flank pain, CVA tenderness, ↑ Ca^{+}, K^{+} & ↓ Mg



https://giornaleitalianodinefrologia.it/en/2023/03/40-02-2023-05/

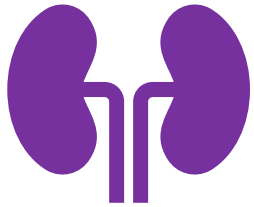
Cigar & Envelope Shaped Crystals

End Organ Toxicity

Cardiopulmonary

Deleterious effects at the cellular level- tachycardia & HTN

Basal ganglia neuron



Renal

Chronic Ingestion

- Throat irritation, mild HA, nystagmus
- Unexplained acute kidney injury
- Mildly elevated anion gap that resolves with minimal intervention
- Abdominal discomfort ranging from nausea, vomiting, & diarrhea to abdominal cramping
- May have a hx of SUD or depression

Clinical Course of Severe Ethylene Glycol Toxicity

- Level > 50 mg/dL associated with significant toxicity
- pH < 7.0 & serum bicarb < 7 mL/dL common with severe ethylene glycol intoxication
- Tx of metabolic acidosis may be difficult
  - Liberal use of sodium bicarb to correct the acidosis
- Prompt administration of ethanol or another blocking agent (i.e. fomepizole) prevents further metabolism
- Thiamine & pyridoxine
- Hemodialysis

Key

- Severe anion gap metabolic acidosis

- Osmolar gap
- Calcium oxalate crystals in urine
- Effects multiple systems, predominantly involves the central nervous, cardiopulmonary & renal systems
- Remember to ask yourself when assessing a "drunk" patient
  - “Am I sure this is ethanol poisoning?”

https://pixabay.com/photos/key-keychain-close-up-123554/

https://journal.medizzy.com/methanol-poisoning-in-49-year-old-triggered-by-alcohol/

Methanol

Deaths of backpackers poisoned by methanol-tainted alcohol in Laos highlights 'forgotten crisis'

www.abc.net.au/news/2024-11-29/methanol-poisoning-broader-impact-south-east-asia/104652326

Etiology

Methanol may be used intentionally or accidentally as an ethanol substitute (including “moonshine”)

www.abc.net.au/news/2024-11-29/methanol-poisoning-broader-impact-south-east-asia/104652326

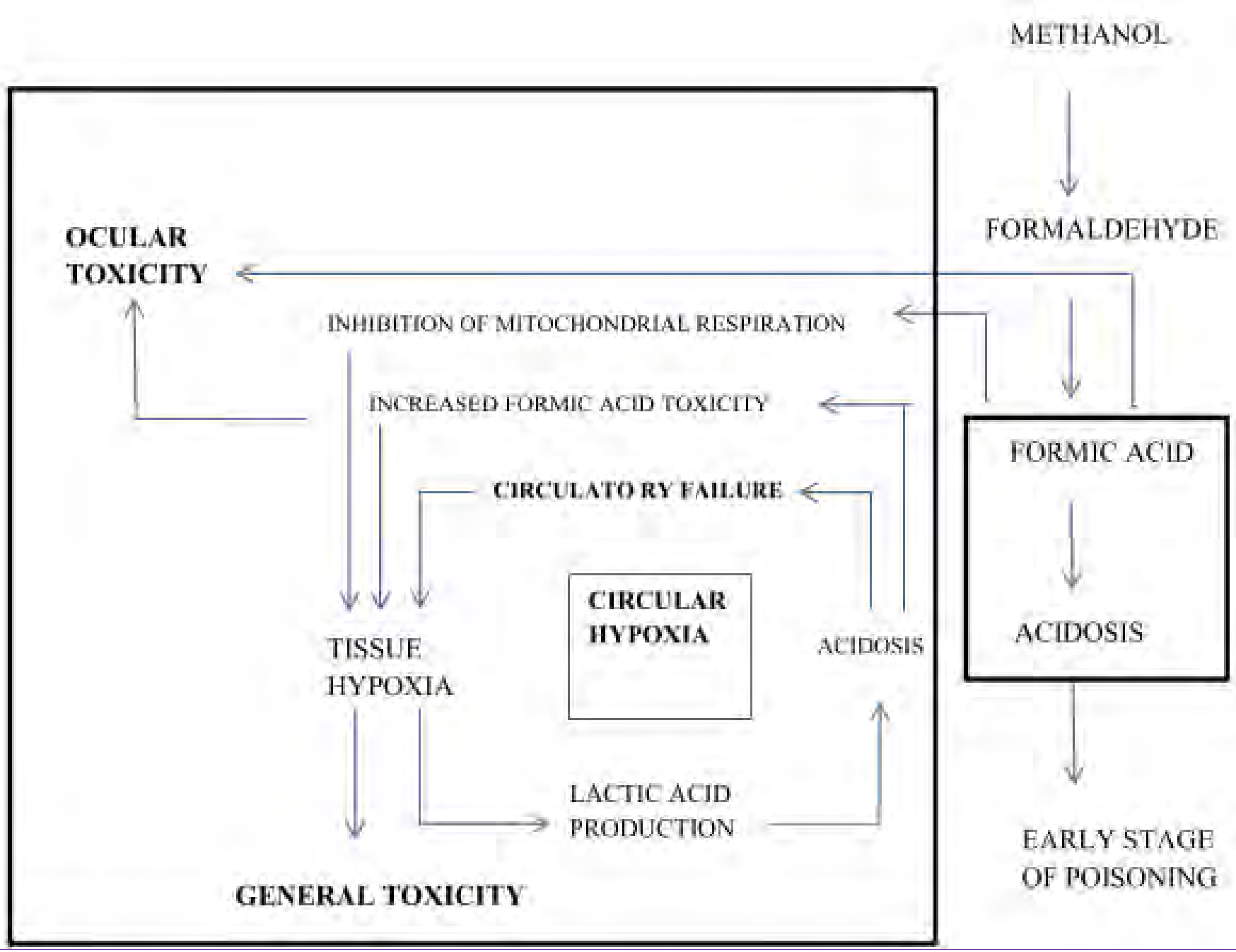
Methanol Poisoning Initiative

- Tracks global outbreaks
- According to data, over past 5 yrs, there have been almost 400 incidents, poisoning some 11,800 people & killing 4,500
- **2024**- 58 incidents worldwide affecting 1,200 people, resulting in > 400 deaths
- Indonesia has the most methanol poisoning cases worldwide- caused by bootleg liquor called “arak”
- Stigma remains a barrier in seeking treatment for methanol poisoning

Pathophysiology

- Clear, colorless fluid that smells & tastes like ethanol
- Ingestion of methanol, as with ethanol, results in intoxication
- Methanol becomes a problem when it is metabolizes
  - Breaks down into formaldehyde, as well as formic acid & formate, wich "basically kills cells“

It's a direct cell poison — it interferes with the energy production of the cells, and so the cells die



Nekoukar Z, Zakariaei Z, Taghizadeh F, et al. Methanol poisoning as a new world challenge: a review. *Ann Med Surg (Lond)*. 2021(66):1-11. doi:10.1016/j.amsu.2021.102445

Case

Chad

- 44 yo male presents to the ED for a recent onset of visual impairment

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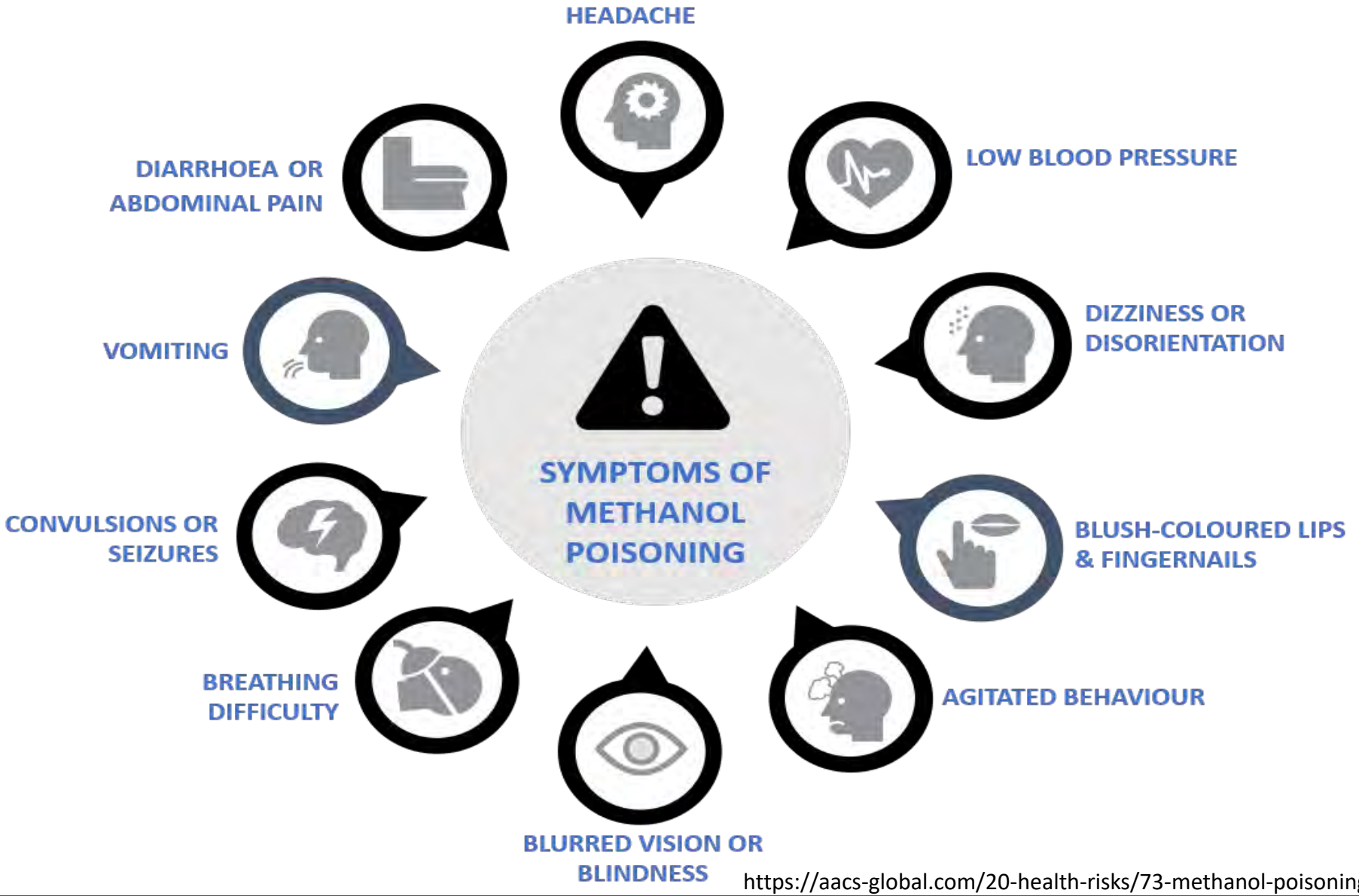
- Reported drinking an unknown quantity of hand sanitizer few days prior to presentation
- Initial laboratory
  - Methanol 97 mg/dL with metabolic acidosis, & an anion gap of 32
  - Serum bicarbonate concentration of <6 mEq/L,
  - ABG pH of 7.09
  - Clinical course complicated by seizures
  - Treated with fomepizole & hemodialysis
  - Recovered after 6-day hospitalization for acute methanol poisoning & was discharged with near-total vision loss

Clinical Features



https://aacs-global.com/20-health-risks/73-methanol-poisoning

End Organ Toxicity

Ocular toxicity

Retinal pigmented epithelial cells & optic nerve cells appear uniquely susceptible- “snowy vision”

Basal ganglia neuron

CNS lesions

Methanol Poisoning During COVID

- Onset of the COVID-19 pandemic, misinformation about the isopropyl alcohol's potential to neutralize this virus, led to significant ↑ in methanol-induced mortality
- June 30, 2020, CDC received notification from public health partners in Az & NM of cases of methanol poisoning associated with the ingestion of alcohol-based hand sanitizers
  - 15 cases found, all hospitalized, 4 died, 7 discharged (4 with no sequelae and 3 with new visual impairments

TABLE. Characteristics of patients admitted to health care facilities with methanol poisoning associated with ingestion of alcohol-based hand sanitizer products containing methanol — Arizona and New Mexico, May–June 2020

Age (yrs)	Sex	Chief complaint(s)*	Serum methanol concentration (mg/dL)	Anion gap† (mEq/L)	Serum bicarbonate§ concentration (mEq/L)	Blood pH¶	Treatment	Outcome
21	M	Gastrointestinal	44	30	6	7.15	4MP	D/C, no sequelae
30	M	Visual disturbance	35	43	11	N/A	4MP	D/C, no sequelae
35	M	Unresponsive, seizures	198	49	<5	6.87	4MP	Died
36	M	Decreased responsiveness	>500	42	7	7.23	4MP, HD	Remains hospitalized**
38	M	Gastrointestinal	131	35	<5	6.81	4MP, HD, CRRT	D/C, no sequelae
38	F	N/A	21††	N/A	N/A	N/A	4MP	Died
39	M	Seizures, unconscious	278	23	11	N/A	4MP, HD	Died
40	M	Dog bite	319	35	<5	7.00	4MP, CRRT	Remains hospitalized**
44	M	Visual disturbance, seizures	97	32	<6	7.09	4MP, HD	D/C with visual impairment
47	M	Headache, visual disturbance	43	34	8	7.25	4MP, HD	D/C with visual impairment
50	M	Visual disturbance	410	22	9	6.70	4MP, CRRT	Remains hospitalized**
51	F	Dyspnea	42	23	6.2	7.14	4MP	D/C with visual impairment
54	M	Media alert§§	56	17	13	N/A	4MP	D/C, no sequelae
63	M	Altered mental status	548	30	11	7.12	4MP, HD	Remains hospitalized**
65	M	Unresponsive, seizures, cardiac arrest	308	31	<5	N/A	4MP, HD, CRRT	Died

Abbreviations: CRRT = continuous renal replacement therapy; D/C = discharged from hospital; F = female; HD = hemodialysis; M = male; mEq = milliequivalents; 4MP = fomepizole; N/A = not available.

* Chief complaint(s) directly came from medical records. Laboratory data were earliest recorded results.

† Normal = 3–10 mEq/L; elevated levels can indicate metabolic acidosis.

§ Normal = 22–28 mEq/L.

¶ Normal = 7.35–7.45.

** As of July 8, 2020.

†† 2 days after admission.

§§ Patient saw media report on alcohol-based hand sanitizers containing methanol and wanted to be evaluated by a medical professional.

Yip et al, 2020

Key

- Need a **heightened awareness** of methanol toxicity
- Seen in **improperly distilled alcoholic beverages**
- May lead to **blindness**/death if not promptly treated
- **Anion gap metabolic acidosis** seen in large ingestions
- **As soon as the dx suspected, treat with fomepizole**
- Ethanol 2nd line for treatment
- Hemodialysis is often necessary

https://pixabay.com/photos/key-keychain-close-up-123554/

https://www.freepik.com/search?format=search&last_filter=selection&last_value=1&query=Hand+Sanitizer+Hands&selection=1

Isopropyl- Alcohol on STEROIDS!!!

Etiology

- Isopropanol, you only see an increased osmolar gap without metabolic acidosis
- This is why calculating an osmolar gap is important
- Elevated osmol gap ***without*** a metabolic acidosis

Pathophysiology

- Acetone is a ketone, not an aldehyde, it cannot be further metabolized by ALDH
- Eliminated in the urine & expired air
- Acetone does ***not*** cause eye, kidney, cardiac, or metabolic toxicity

It **Does NOT** cause an elevated anion gap or metabolic acidosis

Clinical Features

- Onset of symptoms occurs within 30-60 min
- **GI symptoms prominent**
  - Range from nausea/vomiting, abd pain, & acute pancreatitis
  - To hemorrhagic gastritis & upper GI bleeding
- Inebriation
  - Dizziness, ataxia, confusion, nystagmus
- May see CNS depression, cerebral edema, & seizure
- Cardiovascular
  - Hypotension; secondary to peripheral vasodilation

https://informationmatters.net/data-poisoning-ai/

Making the Diagnosis
A Ticking Time Bomb

Osmolar Gap Calculation

Osmolar gap =

(measured serum osmolality) — (calculated osmolarity)

A normal osmolar gap is <10

Calculated osmolarity = 2 x [Na] + [glucose] + [urea] + [EtOH]

Note that all concentrations used in the above calculations are in mmol/L.

Other Investigations

ABG

Serum osmolarity to calculate osmolar gap

Calcium and renal panel

Ethanol, Ethylene glycol or methanol level

Acetaminophen &/or salicylate level

How Can We Narrow Down the Differential?

- May present with-undifferentiated AMS to GI symptoms

- Acidemia should be present in late presenting cases of toxic alcohol ingestion, ***except for isopropyl alcohol***

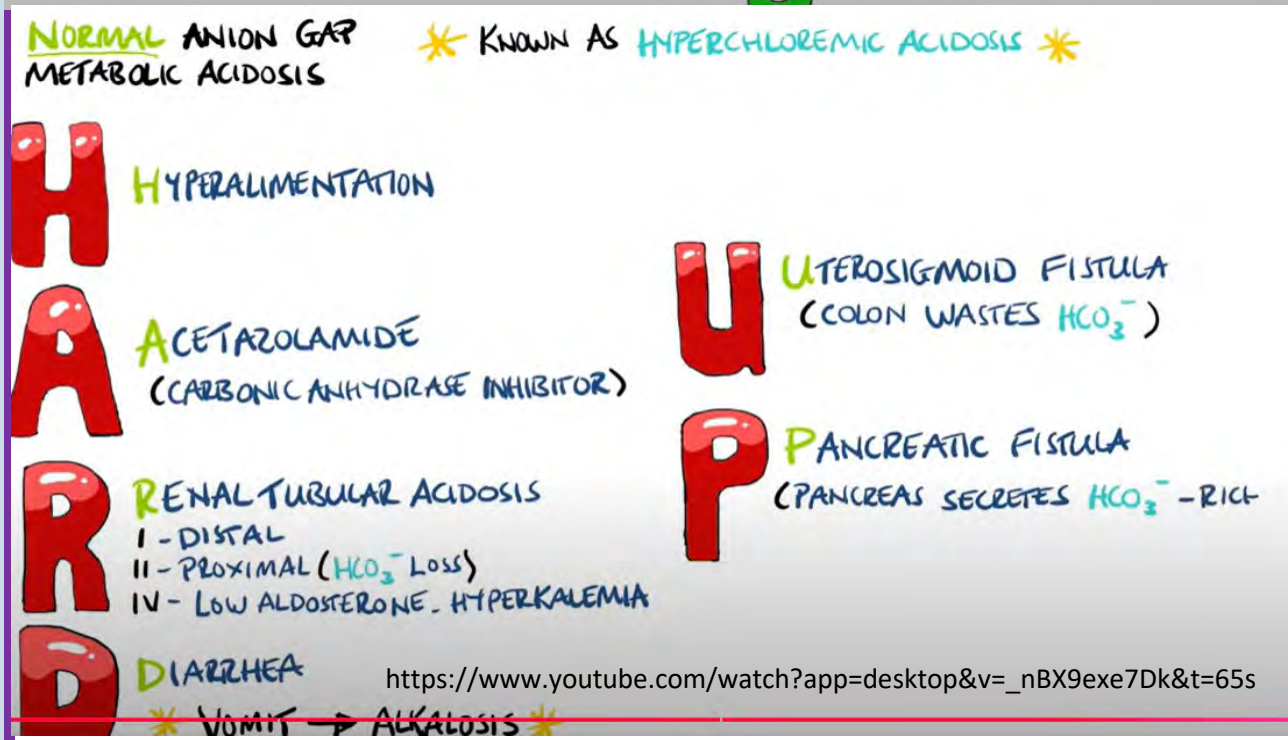
- Ethylene glycol toxicity presenting late in the disease course
  - Evidence of **myocardial depression, cerebral, metabolic acidemia, and hypocalcemia**

- Late presenting methanol ingestion may present with a C/O **vision loss**

- Isopropyl alcohol intoxication mainly presents as **inebriation or signs of gastric irritation** (nausea & vomiting)

Conditions Associated With ↑Anion Gap Metabolic Acidosis

- Ketoacidosis
- Lactic acidosis
- Uremic acidosis
- Salicylates
- Toxic Alcohols



https://www.youtube.com/watch?app=desktop&v=_nBX9exe7Dk&t=65s

Pearls & Pitfalls

DOES A NORMAL ANION GAP R/O TOXIC ALCOHOL INGESTION?

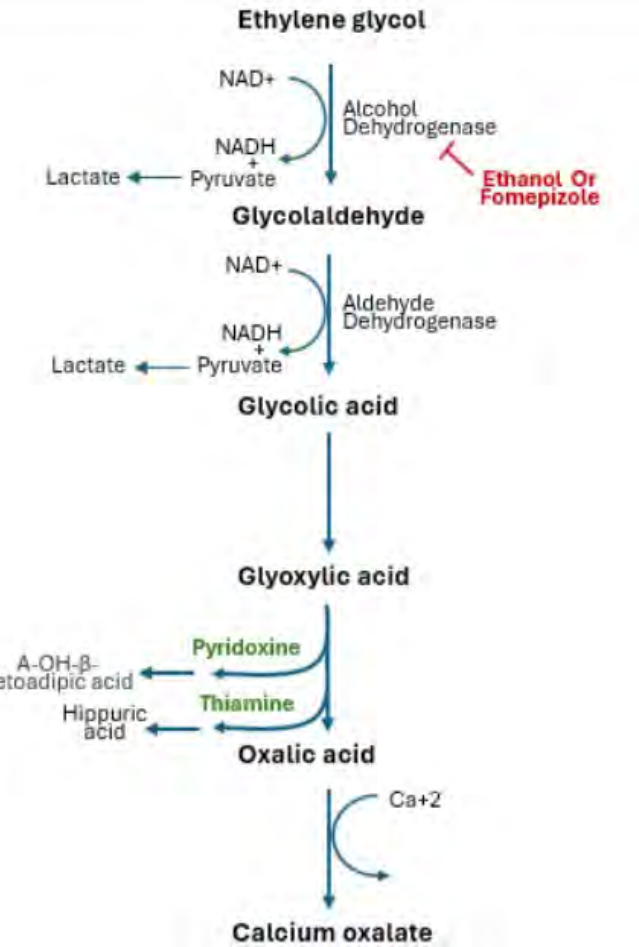
Alcohol Type	Substances Causing Toxicity	Lab Abnormalities	Clinical Findings
Methanol	Formic acid	↑ osmolar gap AGMA ↑ formate Lactic acidosis	Gastric irritation Inebriation Visual symptoms Sz, Coma, Cerebral edema
Ethylene Glycol	Glycolic acid Calcium oxalate	↑ osmolar gap AGMA Calcium oxalate crystalluria ↓Serum Ca Lactate gap	Gastric irritation & Inebriation & Seizure Myocardial dysfunction, shock Renal failure
Isopropyl	Isopropanol	↑Osmolal gap Ketonemia Ketonuria	Gastric irritation & Inebriation Hypotension (peripheral vasodilation)

Management Treatment

Block Toxic Metabolites

https://multiversus.fandom.com/wiki/PFG_Arrival

General Principles



ABC's

Fluid Resuscitation

Correct acidosis-Sodium Bicarb

Consider Dialysis

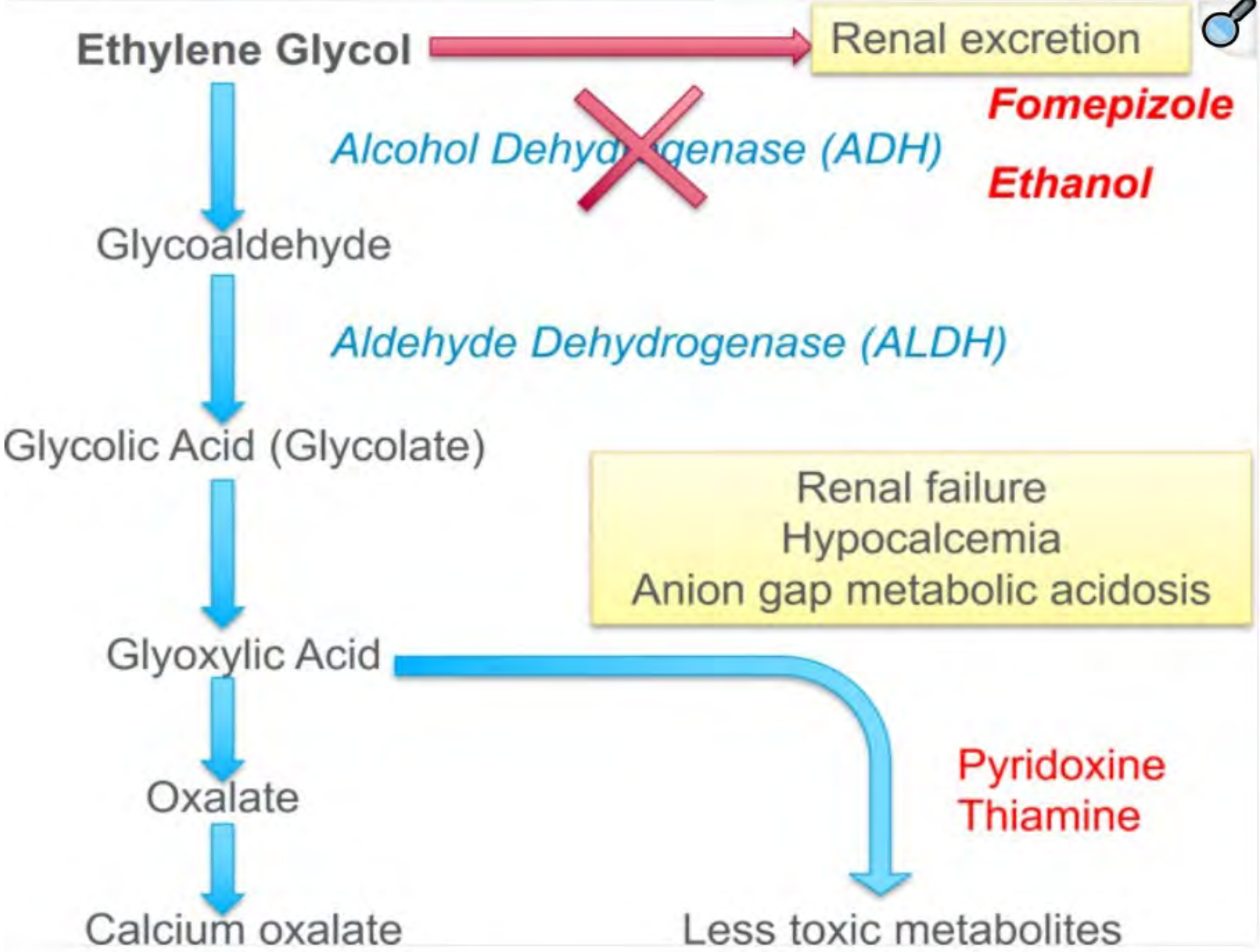
Avoid giving calcium, if possible, with ethylene glycol

Antiemetics, Benzos

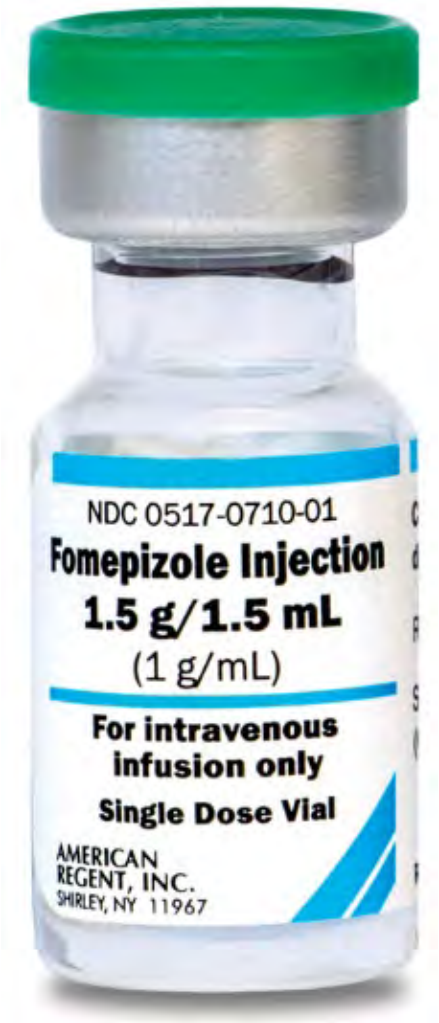
https://www.path.pitt.edu/diagnosis-and-discussion-case-1133

GI Decon

https://commons.wikimedia.org/wiki/File:No_not.png



Sagar, et al., 2018



The Antidote

https://www.americanregent.com/our-products/fomepizole-injection

Fomepizole

LOADING DOSE

15 mg/kg in 100 mL D5W OR NS IV over 30 minutes

Followed by

10 mg/kg Q 12 h in 100 mL D5W OR NS IV over 30 min for 4 doses

Followed by

15 mg/kg every 12 hours until ethylene glycol or methanol levels have decreased to < 20 mg/dL

Patients undergoing dialysis, fomepizole should be re-administered every 4 hours

What About an Ethanol Drip ?

- Ethanol inhibits the formation of toxic ethylene glycol metabolites
- Optimal blood ethanol level is 100 -150 mg/dL;
  - Attain quickly with 10% ethanol IV over 30- 60 min
  - Adjust if going to dialysis

Alcohol Dehydrogenase Blockade	General Dosing
Fomepizole	• 15 mg/kg IV loading dose • Followed by 10 mg/kg Q 12 hrs for 4 doses • Followed by 15 mg/kg Q 12 hrs until ethylene glycol/methanol levels < 20 mg/dL
Ethanol IV	• 10 mL/kg 10% ethanol IV then • Maintenance infusion 1 mL/kg/hr for **target serum concentration of 100 mg/dL** until ethylene glycol or methanol levels < 20 mg/dL
Ethanol PO	• Loading dose **3-4 standard drinks**, followed by a maintenance dose of **1-2 standard drinks/hr** with a goal concentration of 100mg/dL

What Else Can I Do?

- **Thiamine** 500 mg IV Q8 for 6 doses – a theoretical benefit to increase elimination, then 100 mg daily
- **Pyridoxine** (vitamin B6) 50-100 mg IV Q12 – theoretical benefit to increase elimination
- **Folic acid** 50-100 mg IV Q 4 hr for methanol
- **Don’t replace Ca+ unless low enough to cause manifestations**

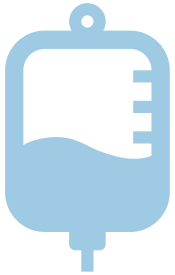
Sodium Bicarb

- **Should be** considered for severely acidotic pts, especially for methanol poisoning while awaiting hemodialysis
- Normalizing pH could keep toxic metabolites in an ionized state (e.g. formate, glycolate)
  - Makes molecules less likely to penetrate tissue (brain, retina)
  - Molecules in the ionized state pass into the renal tubules, get "trapped" & excreted in urine

Not a substitute for hemodialysis,

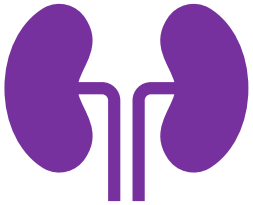
but a temporizing measure

Dosing of Bicarb



Bolus

1-2 mEq/kg of hypertonic $NaHCO_3$ IVP to rapidly improve the pH



Maintenance Infusion

3 amps of $NaHCO_3$ 8.4% (150 mEq) in 1 L of D5W @ 150 mL/hr

Extracorporeal treatment for ethylene glycol poisoning: systematic review and recommendations from the EXTRIP workgroup

Marc Ghannoum[1,2,3], Sophie Gosselin[4,5,6], Robert S. Hoffman[7], Valery Lavergne[1], Bruno Mégarbane[8], Hossein Hassanian-Moghaddam[9,10], Maria Rif[11], Siba Kallab[12], Steven Bird[13], David M. Wood[14], Darren M. Roberts[15,16*] and for the EXTRIP Workgroup

Ghannoum et al. (2023) *Critical Care, 27*:56

Dialysis

- **Indications**
  - Ethylene glycol level > 6-8 mmol/L (50 mg/dL)
  - Methanol > 15 mmol/L
  - Acidosis < pH 7.15- 7.25
  - Anion gap > 24
  - AKI
  - Osmolar gap >10 and hx of lg ingestion- if toxic alcohol level not available
  - End organ damage (visual field deficits, sz, or coma)
- **Endpoints**
  - Ethylene glycol level < 3.2 mmol/L (20 mg/dL)
  - Normal osmolar gap <10
  - Correction of acidosis

Other Consideration

- Thiamine concentration ↓ between 5-40% during hemodialysis
- ECTR may shorten LOS & associated morbidity
- May reduce overall healthcare costs, especially when fomepizole is used

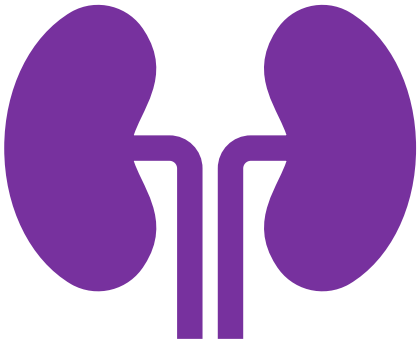


Table 8 Final EXTRIP recommendations on the use of ECTR in ethylene glycol poisoning

INDICATIONS* 1

EG Dose

a. In patients presenting with EG poisoning, **we recommend against** ECTR based solely on the reported EG dose

Plasma EG concentration

a. Fomepizole is used
   i. In patients presenting with EG poisoning, **we suggest** ECTR if EG concentration is > 50 mmol/L (> 310 mg/dL)
b. Ethanol is used
   i. In patients presenting with EG poisoning, **we recommend** ECTR if EG concentration is > 50 mmol/L (> 310 mg/dL)
   ii. In patients presenting with EG poisoning, **we suggest** ECTR if EG concentration is 20–50 mmol/L (124–310 mg/dL)
c. No antidote is available
   i. In patients presenting with EG poisoning, **we recommend** ECTR if EG concentration is > 10 mmol/L (> 62 mg/dL)

Osmol gap (calculated as measured osmolality − calculated osmolarity, in SI units and adjusted for ethanol) when there is evidence of EG exposure

a. Fomepizole is used
   i. In patients presenting with EG poisoning, **we suggest** ECTR if the osmol gap is > 50
b. Ethanol is used
   i. In patients presenting with EG poisoning, **we recommend** ECTR if the osmol gap is > 50
   ii. In patients presenting with EG poisoning, **we suggest** ECTR if the osmol gap is 20–50
c. No antidote is available
   i. In patients presenting with EG poisoning, **we recommend** ECTR if the osmol gap is > 10

Plasma glycolate concentration

a. In patients presenting with EG poisoning, **we recommend** ECTR if the glycolate concentration is > 12 mmol/L
b. In patients presenting with EG poisoning, **we suggest** ECTR if the glycolate concentration is 8–12 mmol/L

Anion gap (calculated as $Na^+ + K^+ - Cl^- - HCO_3^-$) when there is evidence of EG exposure

a. In patients presenting with EG poisoning, **we recommend** ECTR if the anion gap is > 27 mmol/L
b. In patients presenting with EG poisoning, **we suggest** ECTR if the anion gap is 23–27 mmol/L

Clinical indications

a. Coma
   i. In patients presenting with coma due to EG poisoning, **we recommend** ECTR
b. Seizures
   i. In patients presenting with EG poisoning and seizures, **we recommend** ECTR
c. Kidney Impairment
   i. In patients presenting with EG poisoning and CKD (eGFR < 45 mL/min/1.73m^2), **we suggest** ECTR
   ii. In patients presenting with EG poisoning and AKI (KDIGO stage 2 or 3), **we recommend** ECTR

Ghannoum et al (2023)

MODALITY

a. In patients presenting with EG poisoning requiring ECTR, when all ECTR modalities are available, **we recommend** using intermittent hemodialysis rather than any other type of ECTR
b. In patients presenting with EG poisoning requiring ECTR, **we recommend** using continuous kidney replacement therapy over other types of ECTR if intermittent hemodialysis is not available

CESSATION

a. **We recommend** stopping ECTR when the anion gap (calculated as $Na^+ + K^+ - Cl^- - HCO_3^-$) is < 18 mmol/L
b. **We suggest** stopping ECTR when the EG concentration is < 4 mmol/L (25 mg/dL)
c. **We suggest** stopping ECTR when acid–base abnormalities are corrected

https://www.emdocs.net/em3am-methanol-toxicity/

ALCOHOLS IN THE ED

@hansonsanatomy
www.hansonsanatomy.com

ETHANOL

BOOZE

→ acetate

ALCOHOLIC KETOSIS
STARVATION

ASPIRATION
INTOXICATION
RESPIRATORY DEPRESSION

X LACTATE: NML
X ANION GAP: NML
X OSMOLAR GAP:

TX: FLUID RESUSCITATION + TIME

ISOPROPYL ALCOHOL

RUBBING ALCOHOL

→ acetone

KETOSIS W. OUT ACIDOSIS

X LACTATE: NML
X ANION GAP: NML
X OSMOLAR GAP:

REFRACTORY HYPOTENSION
HEMORRHAGIC GASTRITIS
TRACHEOBRONCHITIS
SIGNIFICANT INEBRIATION

TX: FLUID RESUSCITATION + TIME

ETHYLENE GLYCOL

ANTI-FREEZE

→ oxalate & glycolic acid

UA:
+ CALCIUM OXALATE CRYSTALS

X LACTATE: (ON ABG / NML SERUM)
X ANION GAP:
X OSMOLAR GAP:

COMA/SEIZURES
MENINGISMUS
MUSCLE SPASMS
★ HYPOCALCEMIA
RENAL FAILURE
ARDS

TX: FOMEPIZOLE (OR ETOH OR HEMO-DIALYSIS)

METHANOL

WOOD ALCOHOL

→ formic acid

X LACTATE:
X ANION GAP:
X OSMOLAR GAP:

BLURRY VISION
PANCREATITIS
TRANSVERSE MYELITIS
MILD INEBRIATION
BLINDNESS

+ FOLATE

TX: FOMEPIZOLE (OR ETOH OR HEMO-DIALYSIS)

Which Lab Findings Suggests Ethylene Glycol Ingestion?

A. Anion gap 16 and pH 7.32 associated with vision changes

B. Lactate 0.9 mmol/L, pH 7.12, and creatinine 2.5 mg/dL

C. Osmol gap 20, anion gap 8, and ketonemia

D. pH 7.10, lactic acid 5.5 mmol/L, and anion gap 21

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Thank You
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