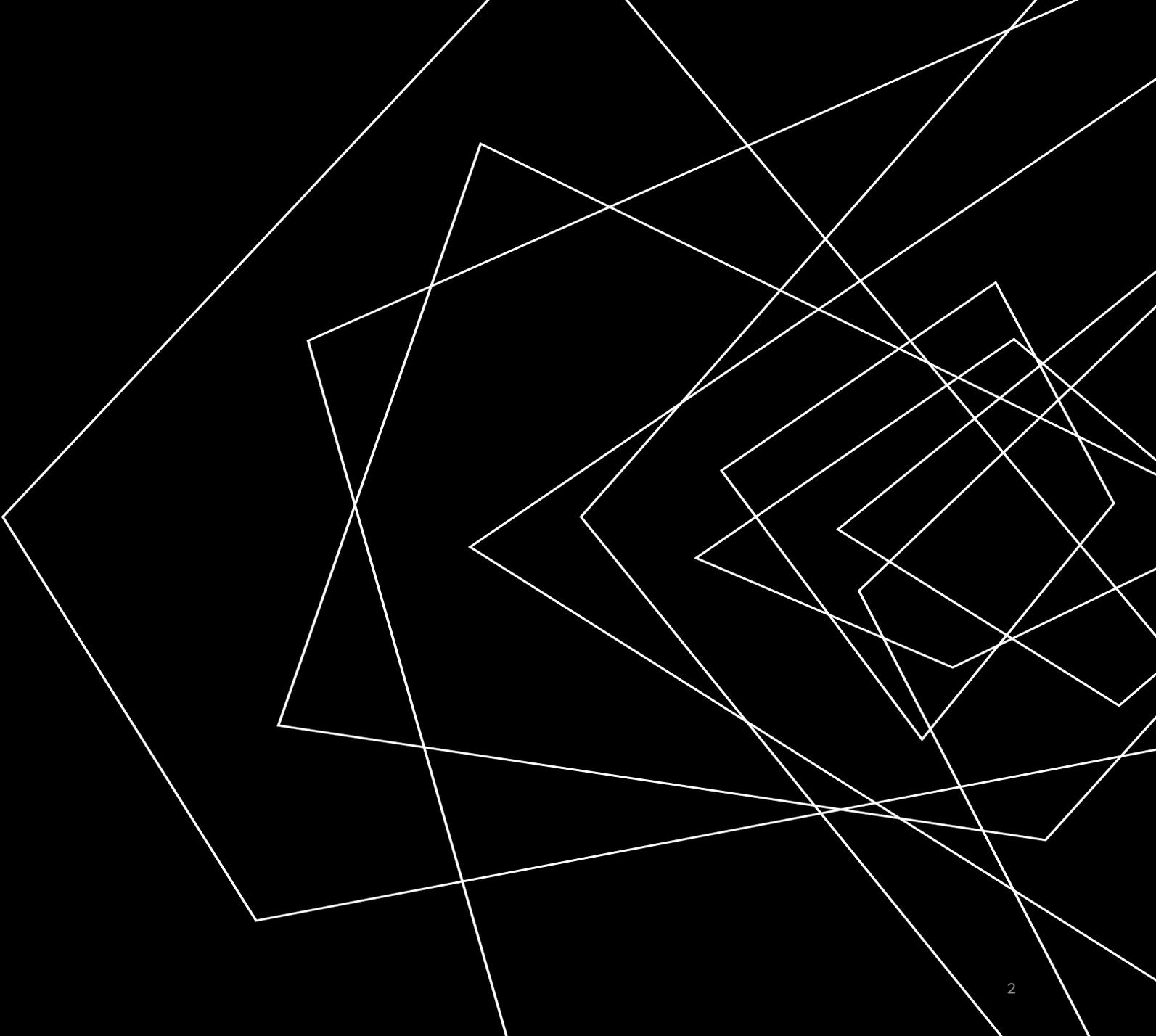
Abstract geometric lines in the top left corner, consisting of several overlapping, irregular polygons and lines that create a complex, layered pattern.

Traditions And Superstitions;
Case Presentations

Akram M Fayed, MD, ABIM

Professor, Critical Care Medicine,
Alexandria University

CASE# 1



CASE #1

A 20-year- old lady presented to the Emergency Department (ED) her family because of:

- Persistent vomiting
- Disturbed level of consciousness
- Tongue biting observed by her family members
- Body spasm

CASE #1

- Past Medical History:
 - Anemia
 - Emotional stress

- Medication History:
 - Unremarkable

- OBGYN History:
 - G3, P3

CASE #1

❖ Physical assessment on admission:

➤ Vital signs:

Temp: 37 RR:14 Pulse:70

BP: 110/70 SaO₂: 98%

➤ General appearance: sleepy,

➤ Head and neck: Bitten tongue

➤ Respiratory system: BEAE

➤ Cardiovascular system: UR

➤ Abdomen: lax and not tender

➤ Extremities: No peripheral edema

➤ GCS: EOP , localize , sounds (hang over from sedation in the ER)

➤ ABG: pH: 7.47 PaCo₂: 18.4 PaO₂: 163 HCO₃: 13.7 O₂ sat:99%

CASE #1

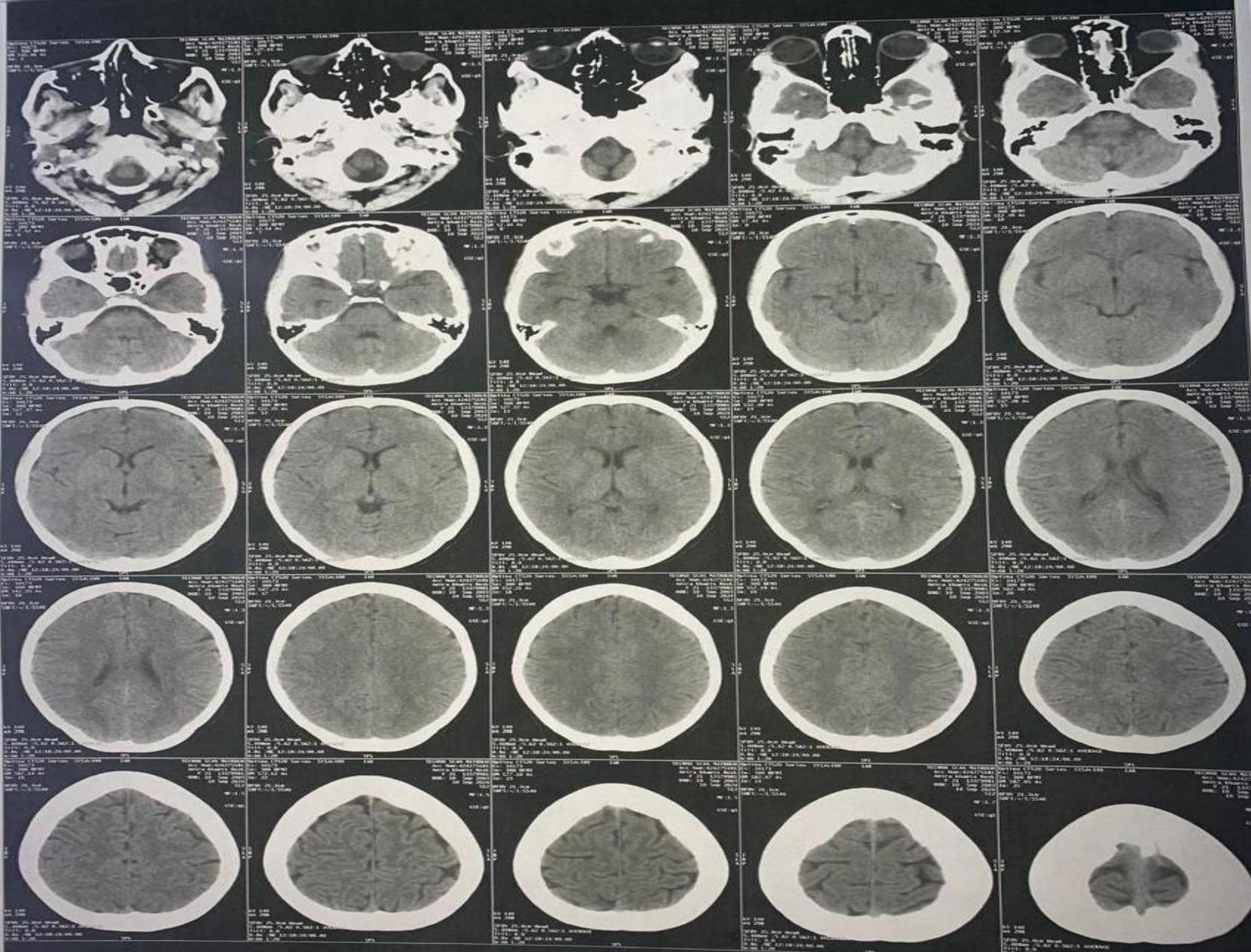
ICU Admission:

- She developed an attack of GTCs → Midazolam loaded with Levetiracetam IV
- She developed another seizure, and the patient was ETT due to Aspiration
- Another Midazolam bolus was given with IV fluid boluses
- Shortly afterwards, the patient developed focal convulsions with 2ry generalization → Loading Phenytoin then maintenance
- Seizures were controlled after adding Midazolam infusion

CASE #1

ICU Admission:

- Her vital signs were normal through the course.
- UOP in the first 4 hours was 8850 mL**
- Labs are back; only remarkable for Severe Hyponatremia; 114 meq/L**
- Hypertonic saline was infused at 20 mL/Hour**

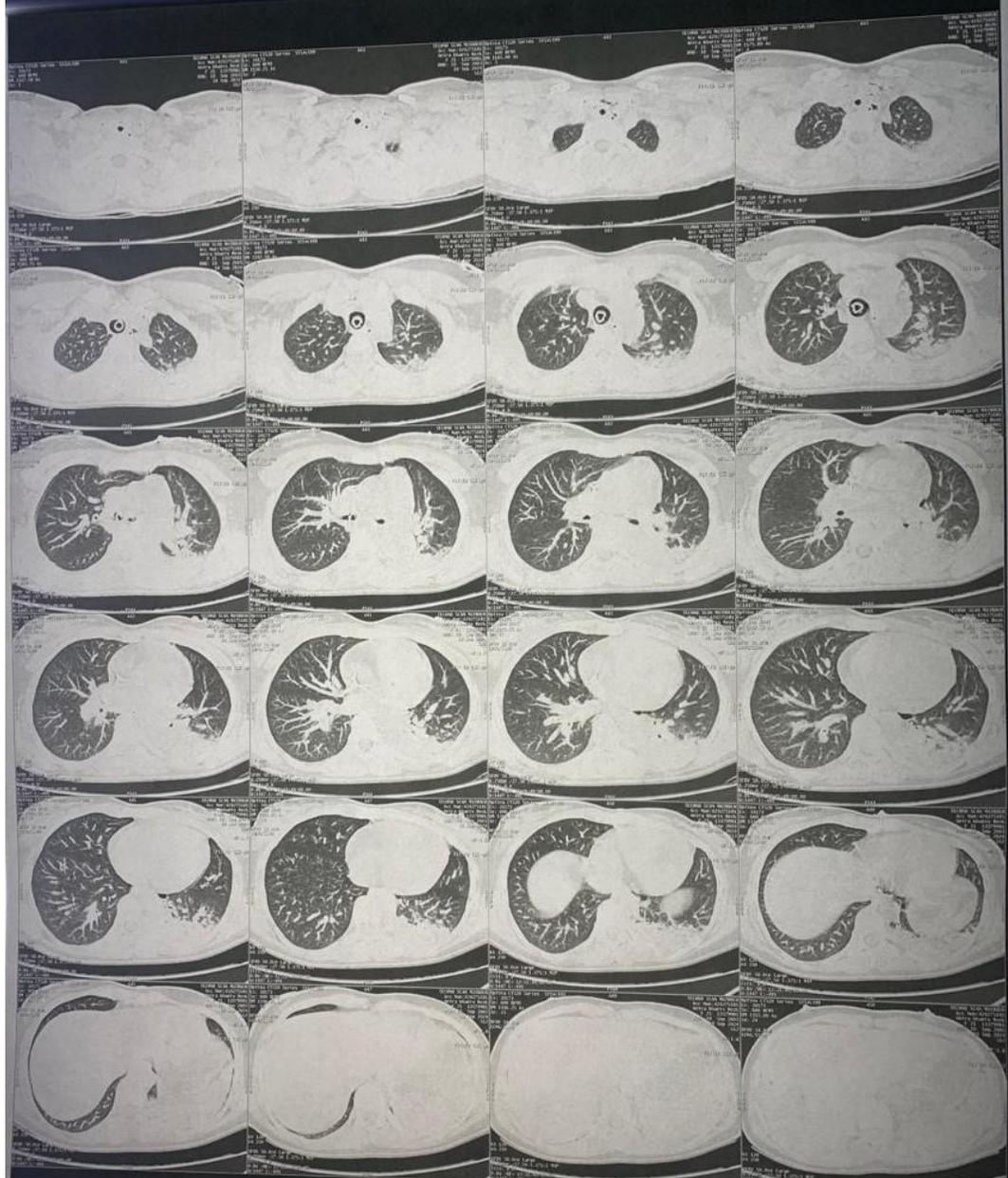


Patient ID:
Age:

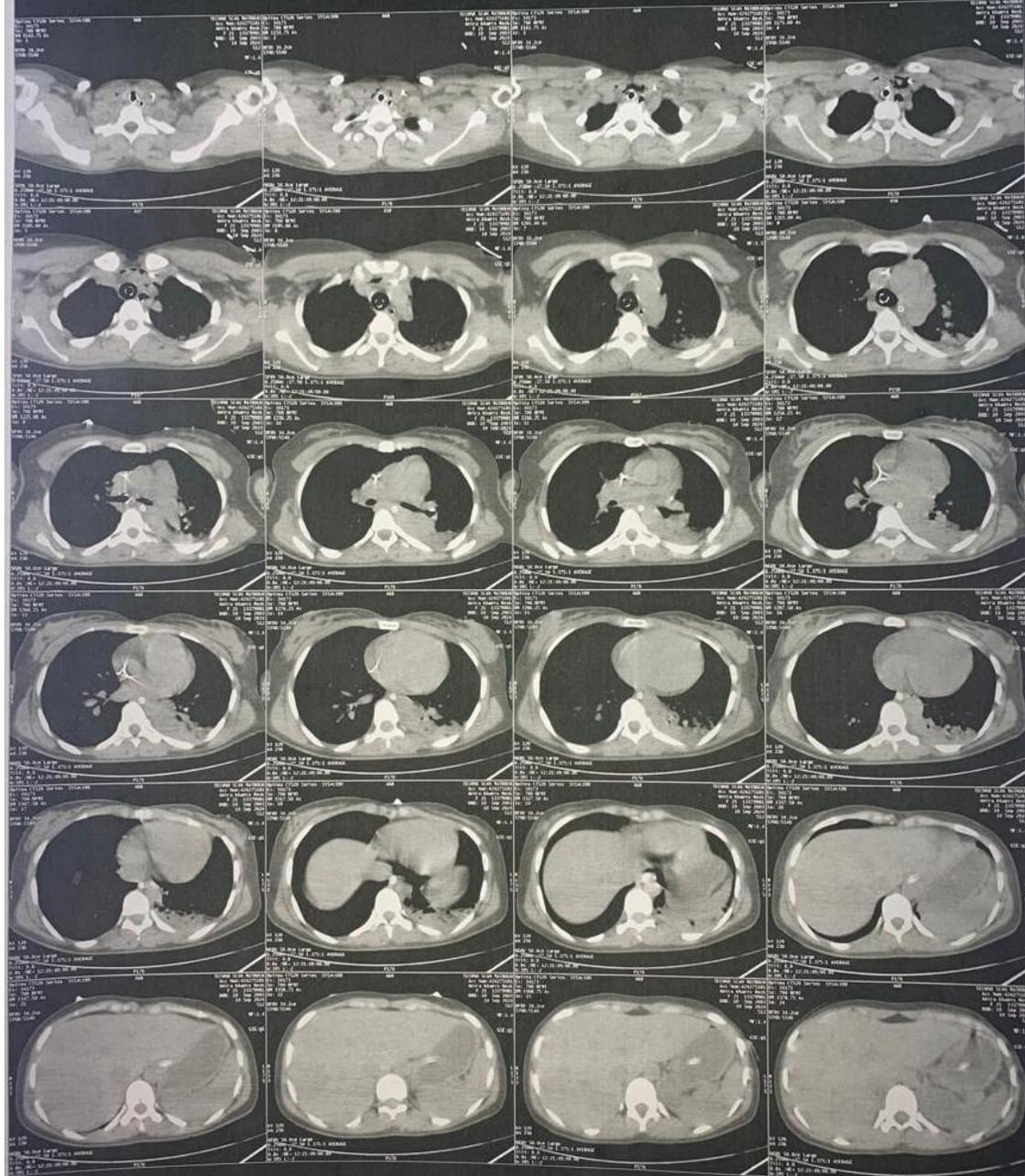
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21Y

Patient Name:
Sex:

Amira Khamis Rezk
F



Patient ID: 13379981
Age: 21Y
Patient Name: Amira Khamis Rezk
Sex: F



Patient ID: 13379981
Age: 21Y

Patient Name: Amira Khamis Rezk
Sex: F



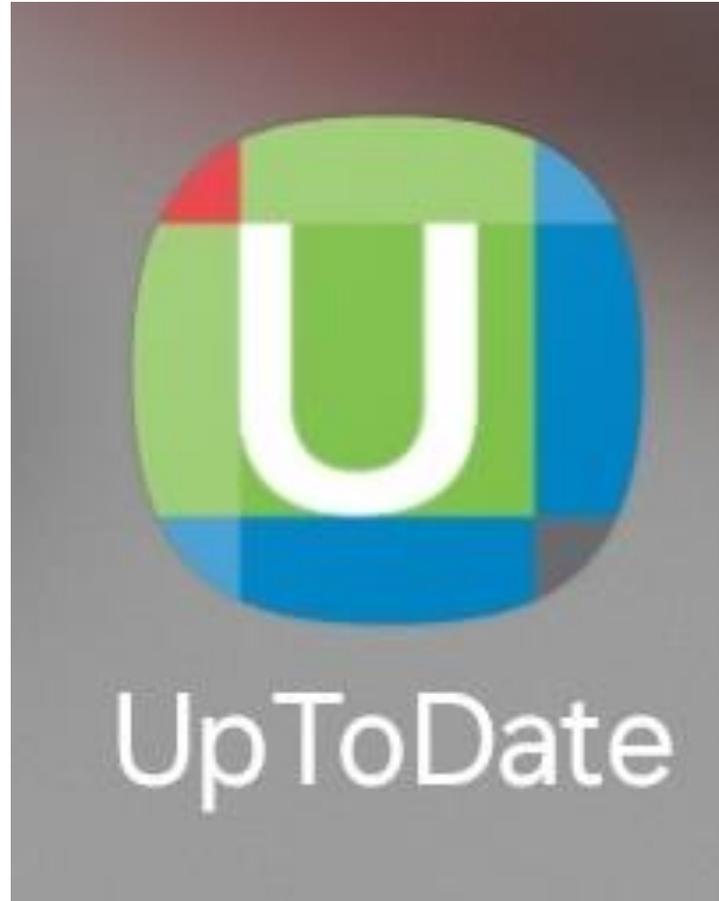
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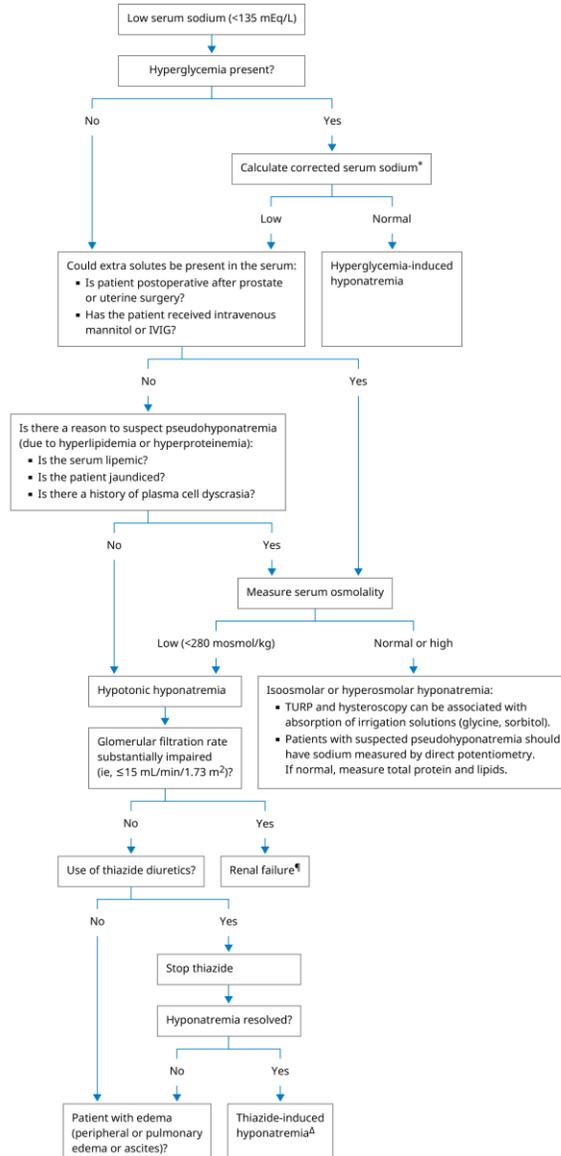
Patient Name:

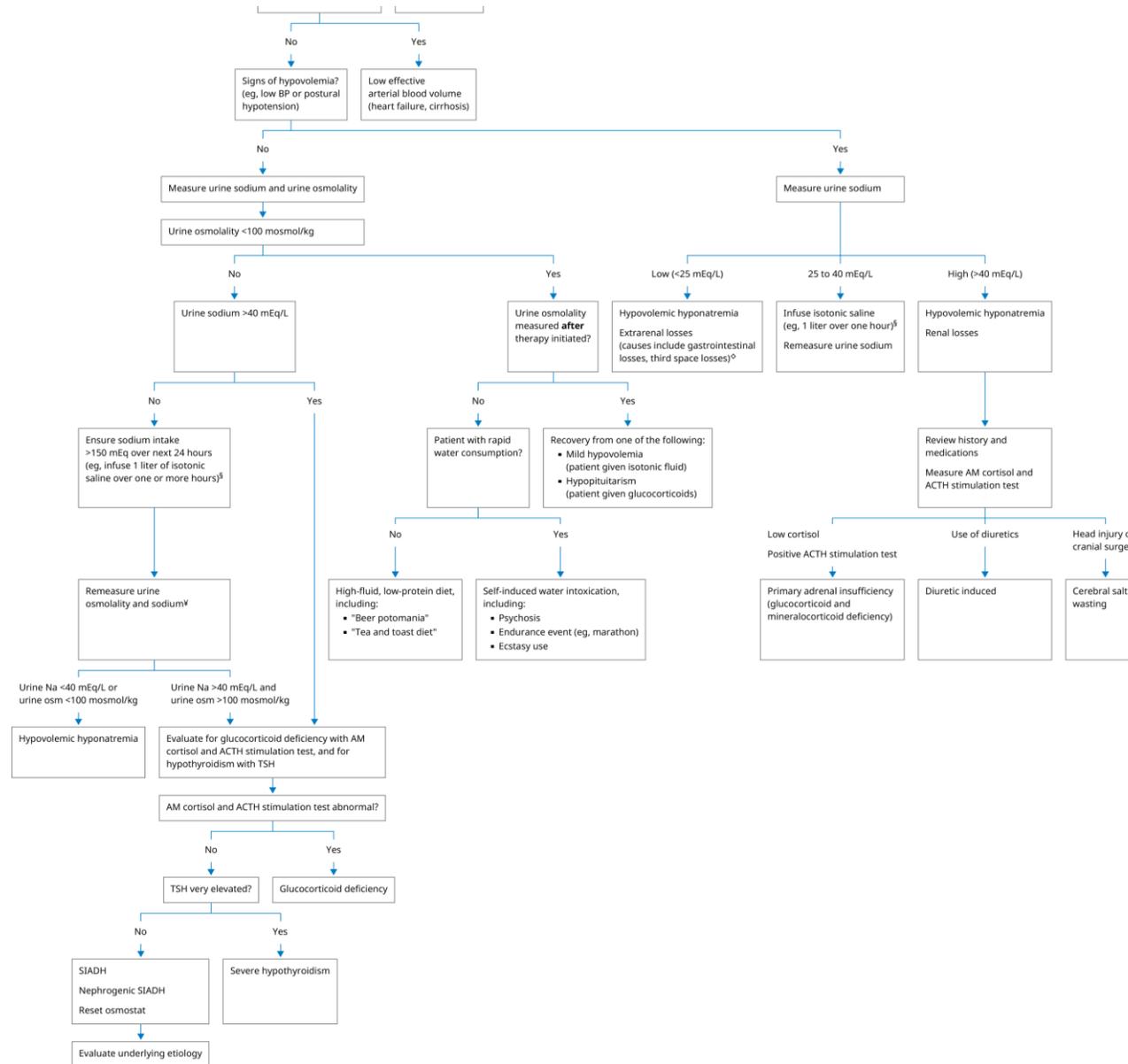
Amira Khamis Bazl

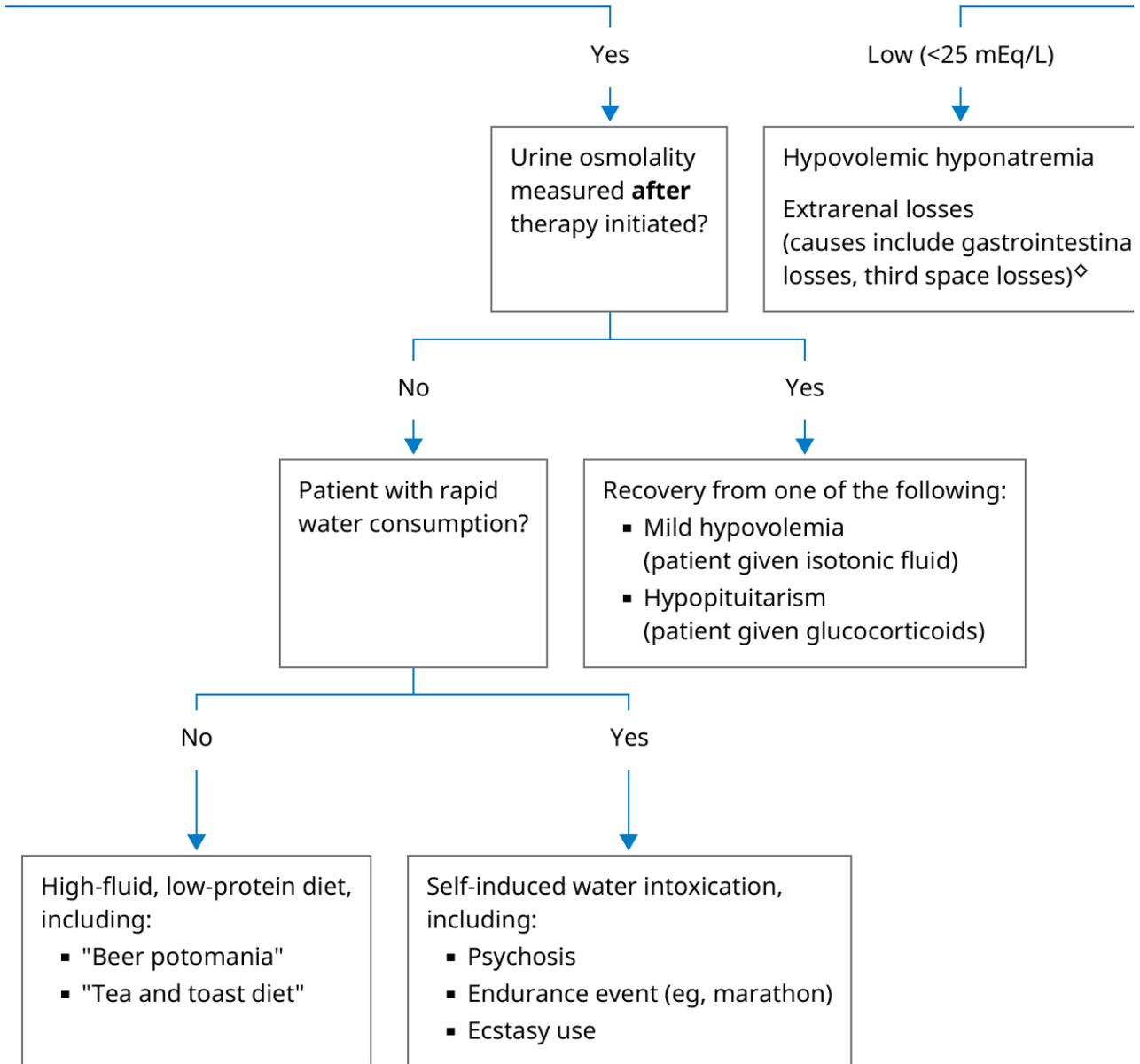
CASE #1



CASE #1







CASE #1

- ❑ Water intoxication,
- ❑ Also known as overhydration or water poisoning, occurs when there's too much water introduced into the body, leading to electrolyte imbalance
- ❑ Mild Symptoms:
 - Nausea
 - Vomiting
 - Bloating
 - Headache
 - Lethargy

CASE #1

Water intoxication

❑ Severe Symptoms:-

- Confusion
- Seizures
- Coma
- Difficulty breathing

❑ In extreme cases, water intoxication can be fatal.

CASE #1

Water intoxication

- ❑ It's important to stay hydrated, but also to avoid drinking excessive amounts of water in a short period of time, especially during heavy physical activity
- ❑ The amount of water needed to cause water intoxication can vary, but it generally happens when someone drinks more than 3 to 4 liters (about 13 to 17 cups) of water in a short period, such as one to two hours
- ❑ This is especially risky during intense physical activities

WATER INTOXICATION

❑ Treatment

- ❑ Stop Water Intake: Immediately stop drinking water to prevent further dilution of electrolytes.
- ❑ Electrolyte Replacement: Administer electrolytes, especially sodium, to correct the imbalance.
- ❑ Diuretics: In some cases, diuretics may be prescribed to excrete excess water.
- ❑ IV Fluids For severe cases, intravenous (IV) HTS may be necessary to restore electrolyte balance
- ❑ Monitoring and Support: Continuous monitoring of vital signs and electrolyte levels is essential to ensure the patient's condition stabilizes

ANCIENT EGYPTIAN TRADITIONAL MEDICINE

Water was used for various treatments due to its perceived purity and healing properties:

- ❑ Hygiene and Cleansing: Water was used for washing and cleansing wounds to prevent infection. It was believed that clean wounds would heal faster.
- ❑ Bathing: Regular bathing was considered essential for maintaining health and preventing illness. It was thought to purify the body and spirit.
- ❑ Herbal Infusions: Water was used to prepare herbal infusions and decoctions, which were then consumed or applied to the body for medicinal purposes.
- ❑ Ritual Purification: Water was used in various rituals to purify the body and soul, which was believed to aid in healing and protection against diseases.

ANCIENT EGYPTIAN TRADITIONAL MEDICINE

Why Water Was Used?

- ❑ Purification: Water was seen as a natural purifier, capable of washing away impurities and negative energies
- ❑ Balance: Ancient Egyptians believed in maintaining a balance of bodily fluids (humors) such as blood, phlegm, yellow bile, and black bile. Water was thought to help restore this balance
- ❑ Religious and Spiritual Beliefs: Water was often used in religious rituals and prayers, which were integral to the healing process. It was believed that invoking the gods and spirits through water could bring about healing

ANCIENT EGYPTIAN TRADITIONAL MEDICINE

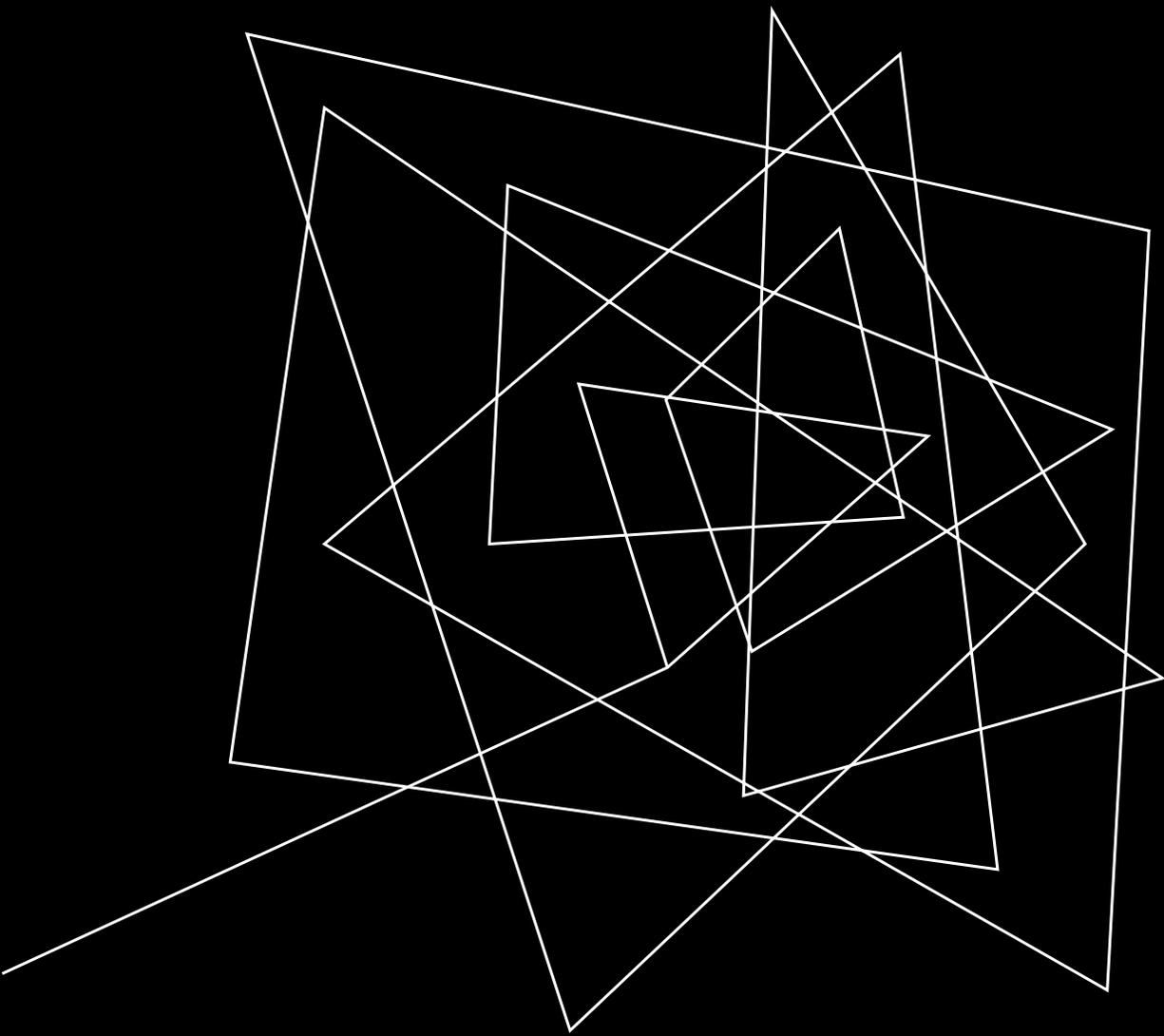
- ❑ Water's role in ancient Egyptian medicine highlights the holistic approach they took, combining practical remedies with spiritual and religious practices
- ❑ It is important how these ancient practices have influenced modern medicine!

CASE #1

- ❑ After 12 hours FU Na was 125 so, hypertonic saline infusion was held
- ❑ EEG in the same night of admission → Generalized epileptiform activity.
Levetiracetam dose was upgraded to 1 gm IV / 12 hours
- ❑ Next day, sedation was weaned off and patient was drowsy , EOP , localize, ETT
- ❑ Patient started to regain consciousness gradually then she was extubated next day but remained sleepy
- ❑ On the third day patient became fully conscious and ambulatory

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- [3] What was ancient Egyptian medicine like? - Medical News Today (<https://www.medicalnewstoday.com/articles/323633>)
- [4] Egyptian medicine - World Archaeology (<https://www.world-archaeology.com/features/egyptian-medicine/>)
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CASE # 2



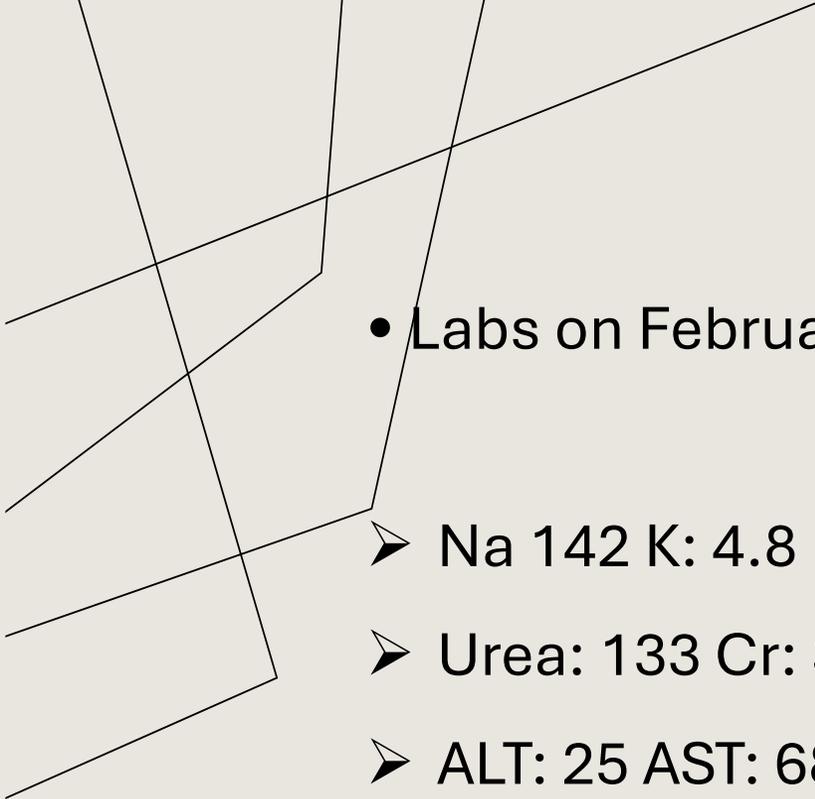
An 87- year- gentleman who presented to the ICU after a few days of

Worsening Abdominal distention

Decreased oral intake

Elevated renal function (at home)

He was treated and followed up at home



- Labs on February 1st, 2024:

- Na 142 K: 4.8

- Urea: 133 Cr: 3.03

- ALT: 25 AST: 68

- PT: 14.8 PTT: 39.6 INR: 1.3 PA: 64%

- Hb: 11.4 HCT: 34.1 WBC: 14 Platelets: 160

- T Ca: 8.4 Cal: 4.6

- Mg: 2.4 P:7.9

- CRP: 37

February 3rd:

US abdomen:

- Marked ascites,
- Moderate Cirrhotic changes; new
- Bilateral Grade I- II MN and

Abdominal pigtail was inserted by IR with 4 liters drained on the first day then 1500mL on the 2nd day

- Labs on February 8th:

- Urea: 211 Cr: 8.24

- ALT: 59 AST: 138

- Hb: 10 HCT: 27.5 WBC: 15.8 Plt: 147

- CRP:82

- Albumin: 1.8

- PSA: 31

He was admitted to the ICU

Past Medical History:

- HTN
- Prostate cancer since 2019
- Last PET scan 3/2023: prostate cancer with no Mets
- Liver cirrhosis; denied alcoholic beverages
- Appendectomy
- Previous ICU admission 19/1>>28/1 on new liver failure, tense ascites, AKI and UTI
- Renal stone
- Bladder stone extraction 28/1/2024
- Topical use of methanol for body aches
- Ex-smoker

Drug history:

- Gastrobiotic 550mg tab 1x2
- Lasix 20 mg tab 72hrs
- Tamsulin 0.4mg tab 1x1
- Zyloric 100 mg tab 1x1
- Zoladex amp /3month (last dose 2/2024)
- Cipro 500mg tab 1x2 (28/1-5/2)
- Concor 2.5mg tab 1x1(stopped on hypotension)
- Lactulose 15 CC syrup / 8 hrs (stopped last 2 days)
- Jolie 10 cc syrup / 12 hr Stopped since 1/2

Hospital course:

- CNS: Patient on admission was SEO, Obey, disoriented and agitating intubated on persistent shock state
- Improved gradually with fluctuation between fully conscious to SEO, obey, ETT
- He developed hypovolemia; upper GIT bleeding & DIC with oozing from insertion sites (SCV)
- With the worsening shock state, he became NEO, Flaccid ,ETT

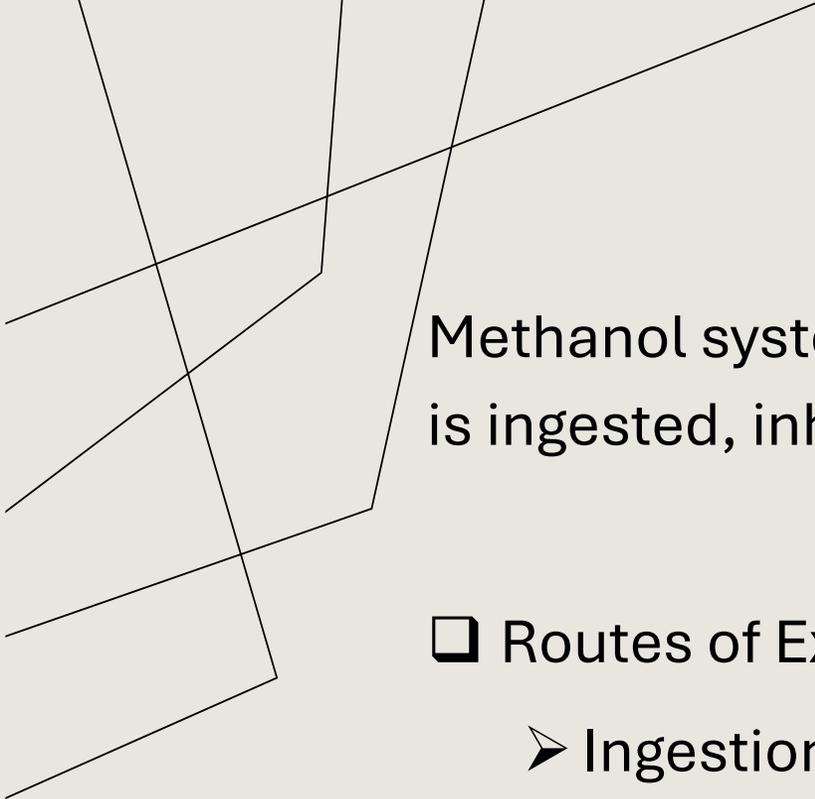
Hospital course:

- Patient was shocked
- Cardiac enzymes were positive >>NSTEMI >> Aspirin, Clopidogrel 75 and heparin were given
- ECHO : EF 39% , Mild LVH , Mild MR, AR, mild AS , bi atrial dilatation, RWMAs, mild TR and mild Pul HTN
- Current diagnosis was mixed septic and cardiogenic shock
- Cautious Fluid boluses, noradrenaline and Dobutamine
- That was worsened by upper GI bleeding; hypovolemic element of shock with failure to wean off vasopressor

Hospital course:

Abdomen :

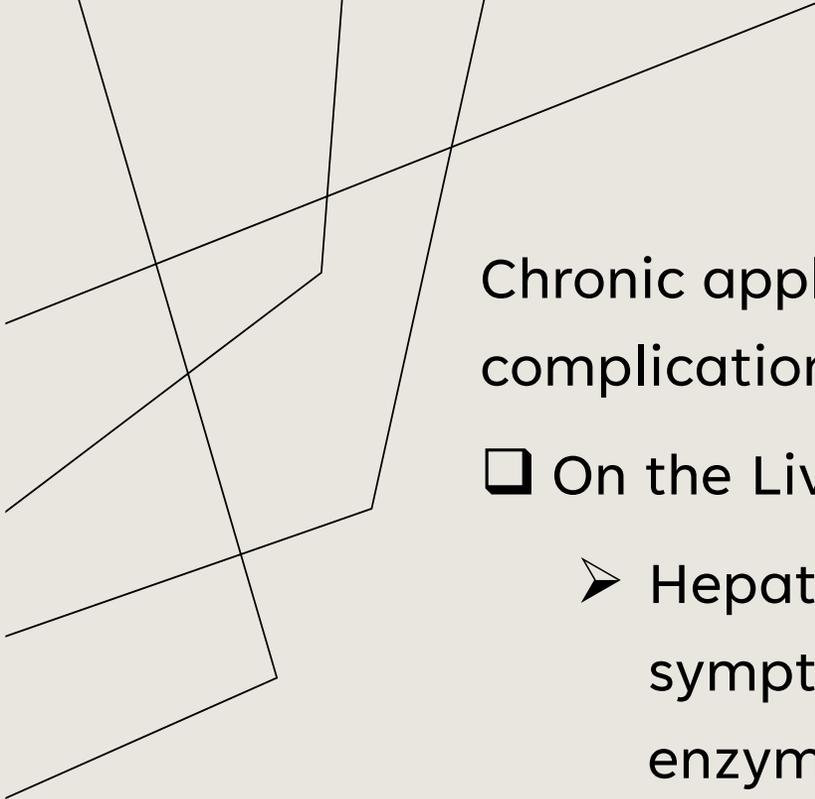
- Lax, but distended with permanent tunneled pig tail for ascites
- Thorough work up to identify the cause of his liver cirrhosis but without success
- History revealed the cause of the liver cirrhosis; rubbing methyl alcohol routinely for treatment of arthritis and generalized body aches
- Hepatology consultation: confirmed it was methanol liver toxicity and need for liver biopsy, but the patient was unfit for the procedure (shock + coagulopathy)



Methanol systemic toxicity, methanol poisoning, occurs when methanol is ingested, inhaled, or absorbed through the skin

☐ Routes of Exposure

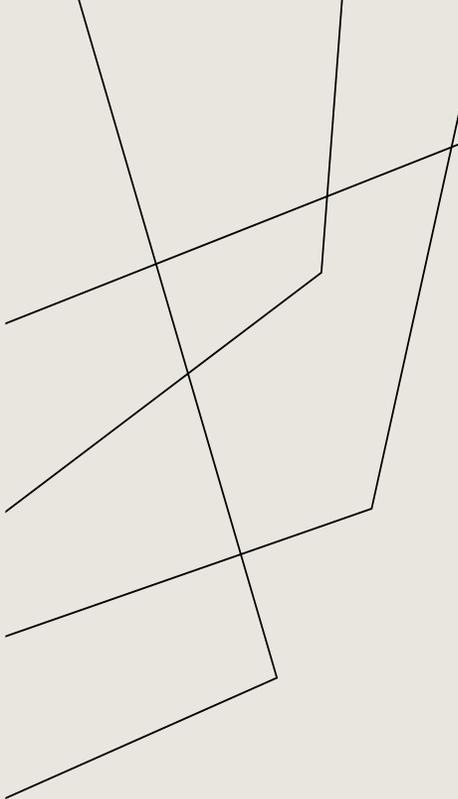
- Ingestion: Drinking methanol-containing substances
- Inhalation: Breathing in methanol vapors
- Dermal Exposure: Absorption through the skin



Chronic application of methanol on the skin can lead to systemic complications due to its absorption into the bloodstream

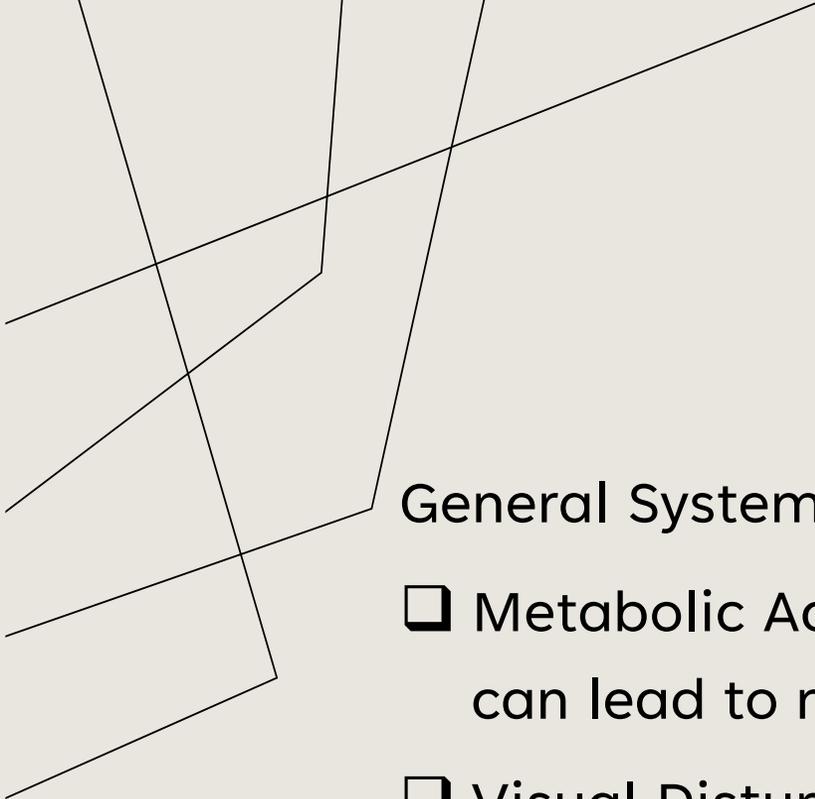
❑ On the Liver

- Hepatotoxicity: Methanol can cause liver damage, leading to symptoms such as jaundice, abdominal pain, and elevated liver enzymes
- Liver Failure: Prolonged exposure can result in severe liver dysfunction, cirrhosis and failure



On Other Body Organs

- ❑ Kidneys: Methanol can cause kidney damage, leading to symptoms like decreased urine output, blood in the urine, and elevated creatinine levels
- ❑ Central Nervous System: Methanol poisoning can affect the central nervous system, causing symptoms such as headache, dizziness, confusion, seizures, and even coma
- ❑ Respiratory System: Chronic exposure can lead to respiratory issues, including difficulty breathing and respiratory failure
- ❑ Cardiovascular System: Methanol can cause cardiovascular complications, such as arrhythmias and heart failure.



General Systemic Effects

- ❑ Metabolic Acidosis: Methanol is metabolized into formic acid, which can lead to metabolic acidosis
- ❑ Visual Disturbances: Methanol poisoning can affect vision, potentially leading to blindness due to optic nerve damage

Hospital course:

- Frequent increases of intra-abdominal pressure with massive tapping was done repeatedly
- Work up confirmed that he had spontaneous bacterial peritonitis complicating permanent pigtail placement; Klebsiella Pneumoniae
- Broad spectrum antibiotics included Tigecycline and Meropenem
- He went through several complications ended with his death

USEFUL REFERENCES

[1] Methanol: toxicological overview - GOV.UK (<https://www.gov.uk/government/publications/methanol-properties-incident-management-and-toxicology/methanol-toxicological-overview>)

[2] Methanol toxicity - Wikipedia (https://en.wikipedia.org/wiki/Methanol_toxicity)

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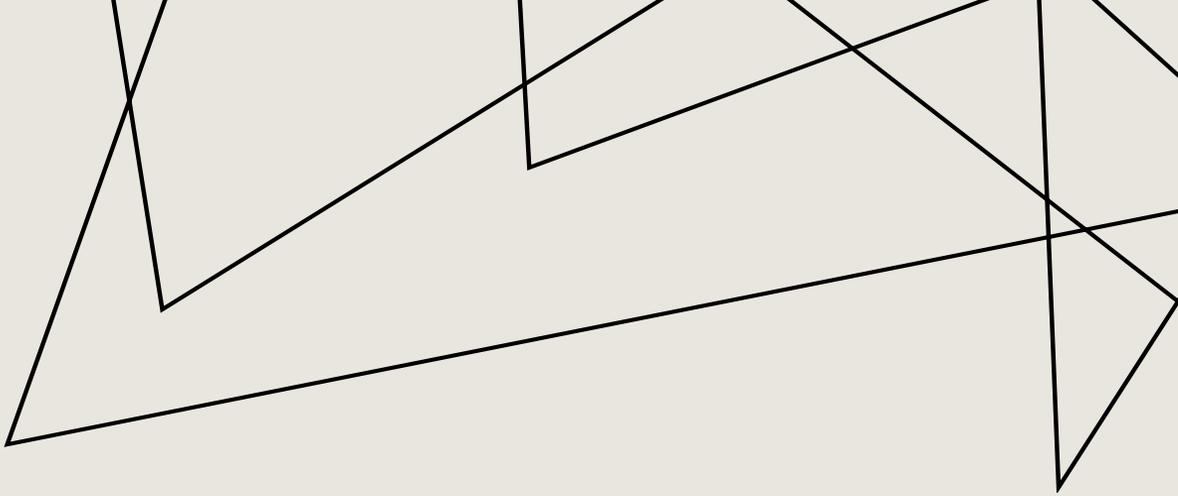
CASE # 3

COMPLAINTS AND HPI:

A 34- year- old lady was transferred from another ICU for further care. She is a physician and most of her relatives are physicians including her husband.

- ❑ The condition started 2 days ago by vomiting & diarrhea not responsive to antidiarrheal and metronidazole. Then she developed epigastric pain for which she received Pantoprazole, Ranitidine and a Spasmolytic.
- ❑ She developed bilateral Lower Extremity weakness & paresthesia>> urgent MRI spine according to her neurosurgeon; history of LDP
- ❑ The MRI technician advised her and her husband to seek hospitalization since he noticed bluish coloration of her toes

CASE# 3



COMPLAINTS AND HPI:

- ❑ Jaundice & bluish discoloration of the toes & ecchymoses began to appear , and she started to faint; became hypotensive.
- ❑ Her husband gave her NaCl and Ringer's , received Zofran 8 mg for nausea then Ranitidine & dexamethasone.
- ❑ Her BP started to become low despite of IV fluids>> Hospital

COMPLAINTS AND HPI:

At the hospital where she received fluid boluses then Nalbuphine for severe LL pain , her labs were remarkable for:

Na 125 , K 3.1

Hgb= 12.3 WBC= 14.64 Platelets= 35K

Lipase 297, Amylase 163

ALT 207 , AST 289,

CRP 128, alb, 3.2 ,

Uric acid 9, Urea 125 creatinine 2.5 , Phosphorus 5.8 ,

Bilirubin (T6.6, D4.8)

CPK 3374

ABG was 7.30, 21, 78, 10.3, 94

CASE3# 3

PAST HISTORY:

- Disc prolapse since 2010

DRUG HISTORY:

- Pregabalin 50 mg
- Paracetamol PRN
- Ketoprofen PRN
- Norflex (Orphenadrine Citrate)
- Ranitidine

CASE# 3

Physical Examination on ICU admission:

Fully conscious

BP 70/40 (on IV fluids given at home) HR= 100 Temperature- 37.8 RR= 24

Adequate AE bilaterally without wheezes or rales

Lax abdomen with mild epigastric tenderness

Bluish coloration of her toes and feet

Henna on her hands

Provisional admission diagnosis: Acute Pancreatitis, Acute Kidney Injury, Hyponatremia and Acute liver insult



CASE3# 3

- ❑ A few hours later, she started to have stridor and increased work of breathing that worsened over short period>> Intubation for airway protection and MV
- ❑ During intubation, the vocal cords looked edematous to the resident.
- ❑ Urine started to be chocolate- brown in color
- ❑ Resuscitation started with fluid boluses followed by vasopressor infusion as she was shocked
- ❑ Empiric antibiotics were started, and sepsis workup done lab investigations revealed thrombocytopenia , DIC and lactic acidosis

CASE3# 3

- ❑ Then chest condition deteriorated, and she became hypoxic and distressed on MV, developed ARDS >> lung protective strategy with high PEEP and closed suction system
- ❑ IAH was the diagnosis and pulse steroids started and received platelets transfusion daily
- ❑ Patient received pulse steroids for 5 days
- ❑ Nephrology was consulted and first session Plasma Pharesis was done
- ❑ Patient developed chest pain , ECG revealed inferior STEMI, positive cardiac enzymes.
- ❑ The Cardiologist recommended against thrombolysis because of thrombocytopenia.

CASE3# 3

- ❑ ECHO: EF 50% , ischemic Vs septic cardiomyopathy.
- ❑ Autoimmune disorders , vasculitis , leptospirosis , TSS and lupus were ruled out by lab investigations and immunological markers (all were negative)
- ❑ Virology done ---- negative
- ❑ Pancreatic enzymes were rising so CT abdomen with iv contrast done to exclude pancreatitis

CASE# 3

- Vomiting and Diarrhea
- Acute Kidney Injury; Rhabdomyolysis
- Ecchymosis, Thrombocytopenia and DIC
- Vocal Cord edema
- ARDS
- Jaundice and elevated liver enzymes
- High Amylase and Lipase without CT evidence of Pancreatitis
- NSTEMI with impairment of EF “Septic vs Cardiomyopathy”!
- Lower extremity ischemic changes
- Lactic acidosis



CASE# 3

- ❑ This is a mess!
- ❑ What is the single disease that cause of all of these organ injuries
- ❑ When you are in a MESS, Think out of the BOX



HENNA

CASE# 3

- ❑ Diagnosis of PPD (Paraphenylenediamine) poisoning
- ❑ Especially when her husband brought the pack of the henna paste she used, confirmed the diagnosis

CASE# 3

- PPD is widely present in Kala Pathar (black stone); once crushed it is typically mixed with henna (*Lawsonia Alba*) which is then used to color hair or stain soles and palms, it is marketed as a black stone dye in Egypt



Fig. 1 Black stone dye (before and after crushing)

CASE# 3

- The use of hair dyes can be traced back to 4000 B.C. when the hair on Egyptian mummies was dyed with henna
- PPD when ingested is highly toxic to the muscular, respiratory, renal, hepatic, and cardiac systems; its toxicity is mainly due to the inhibition of cellular oxidation, and its effect largely relies on the dosage consumed; the lethal dosage is estimated to be between 7 and 10 g.
- PPD generates local and systemic toxicity when administered topically and/or ingested
- The initial report of systemic PPD poisoning was in 1924 and included a hairdresser who became toxic after receiving the dye

CASE# 3

- ❑ Angioedema which can cause upper airway obstruction and difficulty breathing, rhabdomyolysis, acute kidney injury, acute hepatitis, myocarditis, convulsions, shock, and even death are all significant clinical symptoms of PPD ingestion
- ❑ Angioneurotic edema is the main presenting symptom of PPD toxicity to the Emergency department (ED) and it is the primary reason for death within the first 24 h

CASE# 3

- Acute kidney injury is the main complication occurring within the first week
- The mechanism involved in the development of rhabdomyolysis by PPD is by promoting calcium release and leakage of calcium ions from the smooth endoplasmic reticulum followed by continuous contraction and irreversible change in structure of the muscle.
- Rhabdomyolysis is the main cause of ARF
- Hypovolemia, and the direct toxic effects of PPD or its metabolites on the kidneys also contribute

CASE# 3

- ❑ Due to the lack of a particular antidote, early detection and supportive measures are the cornerstones of management

- ❑ Immediate tracheostomy is the main line of treatment followed by:
 - Forced alkaline diuresis,
 - Steroids, and
 - Antihistamines,
 - In circumstances where renal failure develops dialysis is required

CASE# 3

- CPK returned to normal and the AKI improved gradually;
- Platelets count continued to improve>>IAH improved

- She was weaned off MV
- Regained her over all previous state of health

- She had amputation of the gangrenous toes and part of the metatarsals

- She is one who vows against Henna nowadays

USEFUL REFERENCES

[1] Hair Dye Allergy: Symptoms, Treatment, and Color Alternatives
(<https://www.healthline.com/health/hair-dye-allergy>)

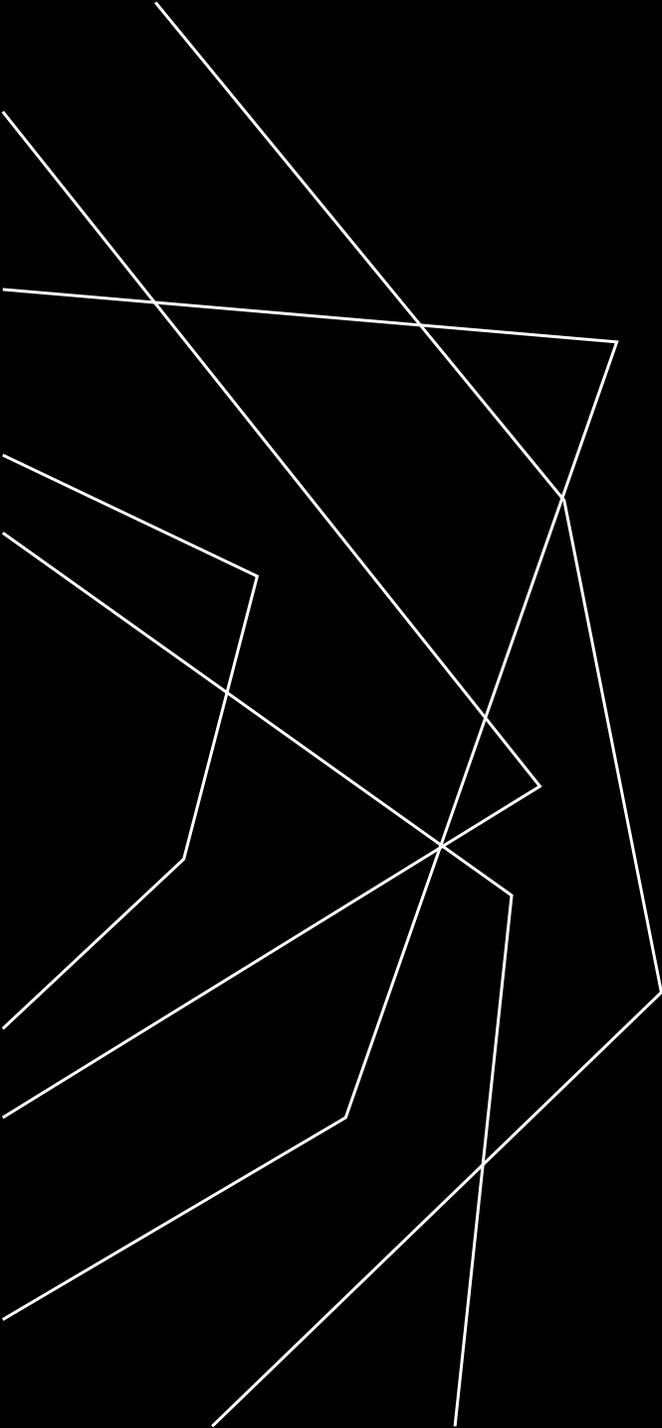
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[4] Hair Dye Allergy: Symptoms, Treatment, and Color Alternatives
(<https://www.healthline.com/health/hair-dye-allergy>)

Take Home Messages

- ❑ Importance of thorough history taking and careful examination
- ❑ Careful history is CHEAP, Unnecessary work up is EXPENSIVE.
- ❑ Not only money is wasted, time too
- ❑ Consider traditional medicine and poisons in differential diagnosis especially in multi organ failure
- ❑ Traditions and Superstitions are still there affecting people and their health
- ❑ Health awareness is important to avoid their adverse impact on people's health

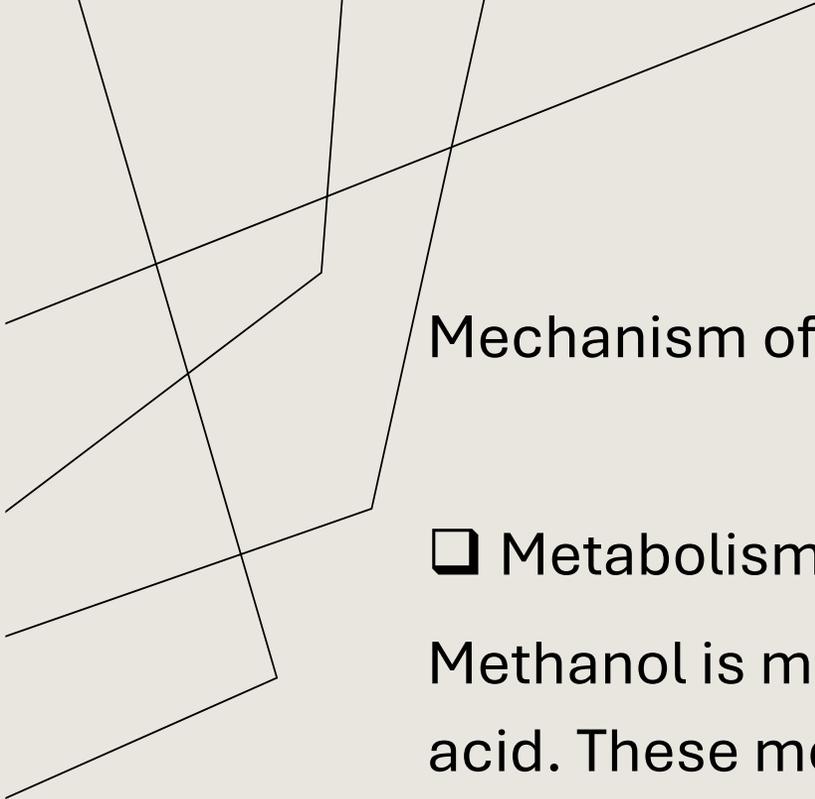


THANK YOU

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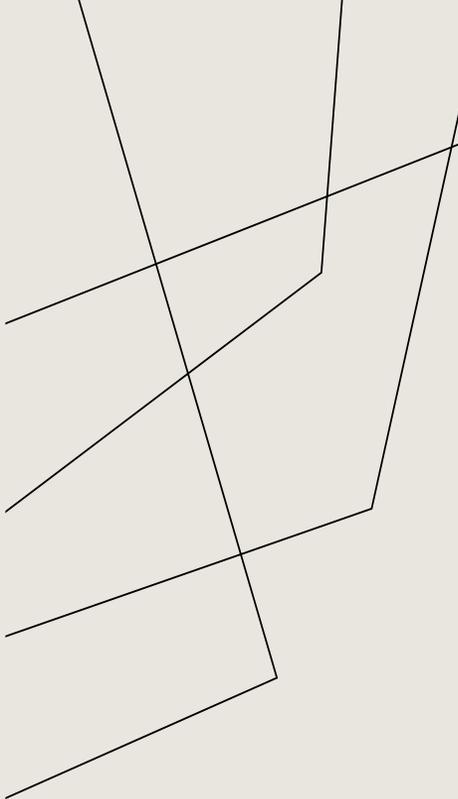
Mechanism of Toxicity:

Metabolism:

Methanol is metabolized in the liver to formaldehyde and then to formic acid. These metabolites are responsible for the toxic effects.

Ocular Toxicity:

Formic acid can cause damage to the optic nerve, leading to vision loss



Treatment

- ❑ Immediate Medical Attention: Essential for anyone suspected of methanol poisoning.
- ✓ Antidotes: Administration of ethanol or fomepizole to inhibit methanol metabolism.
- ✓ Supportive Care: Includes intravenous fluids, correction of acidosis, and in severe cases, hemodialysis to remove methanol and its metabolites from the blood.

Prevention

- Proper Handling: Use appropriate personal protective equipment (PPE) when handling methanol
- Safe Storage: Store methanol in a well-ventilated area away from food and beverages