

HYDROCARBON POISONING

AN INTRODUCTION

- Hydrocarbons are compounds that consist of hydrogen and carbon which are the chief components of petroleum and natural gases.
- Hydrocarbons can be gases (methane, propane), liquids(hexane, benzene) , Waxes (paraffin wax, naphthalene) or polymers(polyethylene, polypropylene and polystyrene).

- Hydrocarbons come in four structural classes.
- **Aromatic** - contain a benzene ring (**most toxic**) and are used in solvents and glues but also in paint and paint remover.
- **Aliphatic** - petroleum distillates found in polishes, lamp oils, and lighter fluid.
- **Halogenated** - fluorinated, chlorinated, or brominated, and are used for refrigeration (freon) and as insecticides and herbicides.
- **Terpene** - found in turpentine and pine oil. Some of these hydrocarbons may be found in various mixed forms and used as an aerosol spray propellant.

USES:

- Predominantly used in combustible fuel source
- Mixtures of volatile hydrocarbons are used as propellants in aerosol sprays
- Commonly used for lighter fluids, paints, pesticides, polishing agents and lubricants.

Hydrocarbon's ability to cause toxicity is dependent on three factors.

Route of Exposure

- Direct skin contact can happen, causing local skin irritation and, rarely, leading to systemic disease. However, prolonged exposure can lead to tissue breakdown and superficial, partial thickness chemical burns. Severe, full-thickness chemical burns can lead to absorption and acute toxic systemic manifestations.
- Ingestion and inhalation/aspiration of hydrocarbons can also occur, which may signify disease and lead to systemic toxicity and morbidity and mortality.

The Chemical Properties

- The hydrocarbon's chemical properties, including its volatility, viscosity and surface tension, affect the disease-causing potential of the hydrocarbon.
- Volatility refers to the rate at which the hydrocarbon can vaporize or exist as a gas. Chemicals with high volatility have an increased risk for pulmonary absorption and can lead to central nervous system (CNS) depression.
- Viscosity refers to the ability to resist flow. Low viscosity allows for deeper penetration into the lungs. Toxic potential mainly depends on viscosity, which is measured in Saybolt seconds universal (SSU). Hydrocarbon liquids with low viscosity (SSU<60) Ex: Gasoline, mineral oil can spread rapidly over large surface area and are more likely to cause aspiration pneumonitis than hydrocarbons with high viscosity (SSU>60) Ex: Tar.
- . The ability of the molecules to adhere along a liquid surface is surface tension. Low surface tension can allow compounds to spread easily over large areas.

Amount of Exposure

- Hydrocarbon exposure, either in a single or cumulative dose, can determine the systemic CNS effects on the patient.

EPIDEMIOLOGY

Hydrocarbon exposure in the sick patient will typically occur three ways:

- First, the unintentional ingestion of household products by children.
- Second, dermal or inhalational occupational exposure to workers.
- Lastly, the intentional inhalation of hydrocarbons by adolescents and adults as a drug of abuse.
- (In a 2016 report, the American Association of Poison Control Centers reports hydrocarbon exposure as a top 25 most frequently involved exposure, showing 29,796 exposures with 24 deaths.)
- The techniques used in intentional inhalation were sniffing, huffing, and bagging. Sniffing refers to inhaling directly from the container or an alternate container into which the substance was added. Huffing refers to soaking a towel or cloth with the substance and placing it over the mouth and nose to inhale it. Bagging is when the abuser places the substance in a plastic or paper bag and inhales, maximizing the concentration.
- Exposure can not only affect the respiratory system and CNS but also the cardiovascular system, gastrointestinal tract, and kidneys. Exposures resulting in seizure and death are usually caused by respiratory failure, arrhythmias (sudden sniffing death), or severe CNS effects.

PATHOPHYSIOLOGY

Exposure to hydrocarbons may be systemic, affecting many functions:

Pulmonary effects

- Inhalation or aspiration may lead to an asthma-like reactive airway syndrome but can lead to a chemical pneumonitis.
- Hydrocarbon has low surface tension and a low viscosity, therefore it penetrates deep into the lungs. This leads to a severe necrotizing pneumonia.
- The chemicals may also destroy surfactant, airway epithelium, alveolar septae, and pulmonary capillaries, leading to inflammation, atelectasis, and fever.

CNS Effects

- CNS effects can be both short and long-term.
- The exact mechanism by which hydrocarbons affect the CNS is not exactly known; however, some studies show that the hydrocarbons may affect NMDA, serotonin, nicotinic, glutamate receptors, voltage-gated ion channels, and the dopamine and GABA pathways in the brain. Some effects also may be due to the metabolism of the hydrocarbon into a neurotoxin. Prolonged exposures, such as seen in workplace exposures, also can result in neuropathy, reduction in brain size, and encephalopathy.

Cardiovascular Effects

- Arrhythmias may be induced following exposure.
- The hydrocarbons, mostly in halogenated form, may increase myocardium sensitivity to epinephrine, leading to a nonperfusing rhythm.
- They also may have a negative inotropic effect and dromotropic and chronotropic effects on the myocardium.
- The exact mechanism is unknown but appears to be due to altered function of calcium, potassium, and sodium channels in the myocardium. Chronic abusers may demonstrate murmurs associated with pulmonary hypertension such as a loud S2.

Gastrointestinal Effects

- Ingestion may cause GI tract irritation and breakdown of the epithelium, leading to nausea, vomiting, abdominal pain, and hematemesis.
- Some solvents may lead to hepatic toxicity.
- Vomiting hydrocarbons may lead to aspiration and pneumonitis.

Renal Effects

- Hydrocarbons (mostly due to toluene) may cause a metabolic acidosis, leading to renal tubular acidosis, urinary calculi, glomerulonephritis, hyperchloremia, and hypokalemia.
- Abuse may lead to both proximal and distal tubular injury.



Aliphatic hydrocarbons

Mode of Action

- HMW- Paraffin wax, Vaseline, Grease produces little or no toxicity
- Liquid hydrocarbons- More toxic

High aspiration potential- Gasoline, Kerosene, Mineral seal oil, Turpentine

Table 27.1: Uses of Aliphatic Hydrocarbons	
Compound	Use
I. Gases —	
Butane, propane	Fuel
II. Liquids —	
Benzine	Solvent
Diesel oil	Fuel
Gasoline (Petrol)*	Fuel
Kerosene	Fuel, curing of tobacco, lighter fluid
Mineral seal oil	Furniture polish
Turpentine (Pine oil)**	Paint thinner, paint remover
III. Semiliquids, Solids —	
Paraffin wax	Candles
Petroleum jelly (Vaseline)	Lubricant
Tar, asphalt	Road surfacing

Gasoline & Kerosene are

CLINICAL PRESENTATION

- **Respiratory System:**

Coughing usually occurs within 30 minutes of exposure but often can be delayed several hours. Many patients develop a transient cough. A prolonged cough and hypoxia however is more concerning for aspiration

Mild- Coughing, choking, tachypnea, drowsiness, rales, rhonchi.

Moderate: Grunting, lethargy, flaccidity, bronchospasm.

Severe: cyanosis, coma, seizures.

- **Nervous system:**

The additives like Aniline, heavy metals, pesticides, lead in gasoline may elevate CNS toxicity

In an acute setting, generalized depression may be seen with slurred speech, disorientation, headache, dizziness, ataxia, syncope, nausea, hallucination, agitation, violent behavior and seizure activity.

With prolonged exposure it may cause peripheral neuropathy which usually begins in the extremities and then progresses more proximally, degeneration of cerebral and cerebellar white matter, secondary parkinsons etc.

- **Cardiovascular system:**

Dyspnea or syncope may be experienced. In addition, due to sensitization of the myocardium to catecholamines, a relatively young and previously healthy patient can present in full cardiac arrest after being suddenly startled or following athletic events.

“Sudden sniffing death” may occur.

CLINICAL PRESENTATION

- **Gastrointestinal:**

Nausea,, vomiting, abdominal pain, and hematemesis. and sore throat are frequent but relatively mild.

- **Local reactions:**

Burning sensation in the mouth, pruritis, or perioral rash are common and are usually mild. Diarrhea, melena and hematemesis are rare.

- **Dermatological:**

- Skin exposure may cause mild irritation, or with prolonged exposure, chemical burns ranging from superficial to full thickness burns. Full thickness burns may lead to systemic symptoms. Skin irritation found priorly is known as “glue sniffer's rash.” Skin lesions may present as bullae or blistering. Other skin manifestations include jaundice and/or mucous membrane irritation.

DIAGNOSIS

- **Patient history:** If patients are too obtunded to provide a history, hydrocarbon exposure may be suspected if their breath or clothing has an odor or if a container is found near them. Paint residue on the hands or around the mouth may suggest paint sniffing.
- **Aspiration pneumonitis:** Chest X ray and oximetry, which are done about 6hrs after ingestion or sooner if symptoms are severe. Double bubble sign
- **Respiratory failure:** If suspected, ABGs are measured.
- **CNS toxicity:** It is diagnosed by neurologic examination and MRI.
- **Complete blood count:** leucocytosis is common in the first 48 hours
- Hepatic transaminase levels should be done.



DIAGNOSIS

- General tests: BUN, creatinine, glucose, electrolytes, anion gap and serum glucose level.
- For asymptomatic patients, chest radiography should be performed at the end of 6 hr observation period.
- ECG should be done to assess for arrhythmias, especially in those individuals with suspected hydrocarbon abuse.

TREATMENT

PRE HOSPITAL CARE:

- Should focus on decontamination, followed by immediate transport to a medical facility capable of managing such a patient.
- GI decontamination has no role in prehospital care. Decontamination should focus on removing any remaining hydrocarbon that might be on the clothes or skin, in the correct clinical setting.
- Patients should be kept calm to prevent arrhythmia as a result of myocardial sensitization.

TREATMENT

- All patients should have their ABC managed as per routine advanced life support protocols.
- Endotracheal intubation, PEEP, HFJV.
- ECMO when other methods have failed
- Decontamination of GI tract remains controversial
- The use of Ipecac- induced emesis is contraindicated and Activated charcoal does not absorb hydrocarbons well.
- Gastric lavage should not be routinely performed, in case of very high doses cautiously do with airway intubation.

GI Decontamination in hydrocarbon poisoning

GI symptoms generally do not need treatment or decontamination. Local poison control should be consulted before starting GI decontamination. The mnemonic CHAMP is helpful in determining serious/life-threatening ingestions.

C - Camphor

H - Halogenated hydrocarbons

A - Aromatic hydrocarbons

M - Metals

P - Pesticides

- Initial treatment should be based on presentation and should focus on possible respiratory or cardiac failure.
- Providers must be prepared to protect the airway when needed, using non invasive or invasive techniques.
- Beta-agonists may be used for wheezing but may not be beneficial.
- Antibiotics may be warranted if a concomitant infection is suspected.
- Steroids have not been proven to be beneficial may cause bacterial superinfection.
- If patients are presenting agitated, or having seizures, benzodiazepines should be given.

- Those presenting with cardiovascular symptoms will need aggressive, intravenous fluid hydration in cases of hypotension.
- Ventricular dysrhythmias should be treated with beta-blockers (esmolol) to prevent catecholamine surges. Catecholamines like epinephrine should be avoided given the increased sensitivity of the conducting system associated with hydrocarbons. Electrolytes, including magnesium and potassium, should be replaced, lidocaine can be considered.

Treatment of frostbite

- Rewarm – water bath in 40 to 42 degree celcius for 15 - 30
Assure complete rewarming, refreezing thawed wound increase tissue damage.
- Wound care should be done.

Absolute contraindications

- Emetics
- Olive oil/ Mineral oil



Aromatic hydrocarbons (benzene & Naphthalene)

Benzene

- Colourless, odourless, inflammable liquid with strong pleasant odour

Sources

- Natural- Volcanoes, forest fires, crude oil
- Recovered from coal tar
- Petrochemical refineries
- Catalytic reformat
- Cigarette smoke

Uses

- Solvent in labs
- Extensively used in industries- drugs, paint, glue, varnish, polish, rubber tyre. Explosives, batteries shoes etc
- Printing, photography, dry cleaning
- Present in petrol as booster

Toxicokinetics

- Absorbed through all routes, as it is highly lipophilic crosses the BBB & placenta.
- metabolised extensively in liver majorly to phenol and excreted in urine

Exposure & Clinical features:

Acute:

- Begin to smell benzene in air: 1.5- 4.7 ppm, water; 2 ppm
- Brief exposure (5 to 10 min) of high benzene air conc (10k to 20k ppm) is fatal
- Inhalation: vertigo, tinnitus, vomiting, dyspnea, convulsion, coma & death. Cardiac arrhythmias may occur.
- Ingestion: burning pain of mouth pharynx, epigastric pain. Vomiting, vertigo, tachycardia, hypotension, dyspnea, convulsion, coma.
- Aspiration : Similar to aliphatic hydrocarbons
- Skin: irritation, erythema, burning. edema and blistering

Chronic

- **Bone marrow suppression & Cancer**
- Carcinogenic effect : induces leukemia (translocation of chromosomes 8 & 21)
- Aplastic anemia, haemolytic anemia, pancytopenia.
- Headache, dizziness, irritability, nervousness, fatigue, anorexia, epistaxis.
- Paroxysmal nocturnal haemoglobinuria (occupational exposure) associated with aplastic anemia, rarely cause acute leukemia.
- Type 2 DM
- Low birth weight
- Crosses placenta – featal abnormalities similar to alcohol

Diagnosis

- Urine phenol & trans muconic acid levels
- Gas chromatography
- Baseline CBC
- ECG (arrhythmias)
- The anion gap will mostly be normal, but in acute toluene intoxication, an elevated anion gap can be present. The presence of an anion gap, especially if associated with a profound acidosis in a patient appearing intoxicated, however should prompt an evaluation for other etiologies (methanol, ethylene glycol, salicylates).
- Serum creatinine kinase(CK) level should be obtained, as acute rhabdomyolysis has been reported in aromatic hydrocarbon intoxication.

TREATMENT

- Supportive care
- Ipecac induced vomiting contraindicated because of CNS depression and seizures.
- Consider lavage with large-bore orogastric tube in potentially life threatening ingestion within 60mins.
- Remove contaminated cloth and wash thoroughly with soap and water
- Administer 100% humidified oxygen, endotracheal intubation and Beta 2 agonists if bronchospasm occurs.
- Treat convulsions.

- Psychiatry consultation should be performed if deemed clinically relevant.
- Patients need long term follow up as there is evidence that these chemicals can cause bone marrow depression and cancer.

Naphthalene

- White scaly powder which volatilises at room temperature

Sources

- Essential oil of the roots of Radix and Herba ononidis and crude oil
- Boiling coal tar oils, crystallisation and distillation.
- Pyrolysis of cigarette smoke.
- Catalytic processing of petroleum.

Uses

- Mouth repellent
- Deodorant cakes
- Scintillation counters

Toxicokinetics and mode of action

- Absorption: Oral, inhalation & dermal routes
- Metabolism:
- First, Hepatic mixed function oxidases to epoxide, naphthalene 1,2 oxide.
- Epoxide is enzymatically converted into the dihydrodiol, 1,2 dihydroxy- 1,2 dihydroxynaphthalene or conjugated with glutathione to form naphthoquinone.
- Excreted in urine and bile.

MOA: Naphthalene by itself is not responsible for any toxic effects, its metabolites alpha and beta naphthols, naphthoquinones are powerful hemolytics

Individuals with G6PD deficiencies are highly vulnerable.

Clinical features

- **Non hemolytic:** Vomiting, abdominal pain, diarrhea, headache, diaphoresis, optic neuritis, restlessness, lethargy, fever, convulsions, hepatomegaly, splenomegaly,
- Hyperbilirubinemia & fatal kernicterus may occur in new borns with hemolysis
- Coma and lung injury in severe toxicity
- Skin exposure: hypersensitivity dermatitis
- Repeated exposure: Corneal ulcers, lenticular opacities, cataracts, malaise, headache, malaise, vomiting

- **Hemolytic:** Pallor, weakness, jaundice, cyanosis, hemolytic anemia, methaemoglobinemia, hyperkalemia, dysuria, haematuria, albuminuria, oliguria & acute renal failure.
- CV shock in severe hemolytic anemia
- Metabolic acidosis
- **Hematological findings:** Increase WBC, fragmented RBC, anisocytosis, Heinz bodies & poikilocytosis.
- **G6PD deficiency, sickle cell anemia, sickle cell trait are at high risk.**
- **Chronic exposure:** Aplastic anemia, hepatic necrosis, jaundice may be associated with laryngeal & intestinal carcinoma.

Diagnosis

- CBC, LFT, RFT, Electrolytes, G6PD levels.
- Urine dipstick test for hemoglobinuria.
- Urinary metabolites (1- naphthol or mercapturic acid) to confirm diagnosis.
- X- ray: To differentiate between mothballs or other products.

Treatment

- Ingestion of one mothball may produce toxicity, more than this amount should be referred to a healthcare facility for gastric decontamination and observation
- If lab findings are negative and if patient is asymptomatic for 4-6 hour observation period, patient maybe discharged.
- Follow up CBC uranalysis for up to 5 days. Instructed to return in case of symptoms.

Decontamination:

- Induce emesis (avoid in lethargy and CNS depression)
- Gastric lavage (poor solubility of naphthlene)
- Since mothballs dissolve slowly, consider it even in late presentation.
- Dermal decontamination

- Avoid oral administration of lipids
- Control seizures
- **Alkaline diuresis-** Prevent renal deposition of Rbcs and breakdown products and RF.
- 1-2 mEq/kg NaHCO_3 - i.v bolus
- Add 132mEq NaHCO_3 20-40 mEq KCl to 5% dextrose in water and infuse at approximately 1.5 times the maintenance fluid rate.
- If patient is dehydrated add isotonic NS to maintain the urine output.
- Manipulate bicarbonate infusion to maintain urine pH of 7.5 (obtain hourly input output & urine pH)
- **Heamolysis-** Blood transfusion, packed RBC.
- **Methemoglobin > 30%** - methylene blue 1-2mg/kg/dose iv over 5mins as needed for q4hrs. Contraindicated in G6PD deficiency.
- Nitrates may also be used
- Hemodialysis may enhance elimination but not routinely performed.

Polycyclic Aromatic Hydrocarbons

- Benzoanthracene, benzopyrene, benzo[a]fluoranthene, phenanthrene etc

Source

- Forest fire
- Sea food agricultural products
- Charring
- Smoke
- Coal tar pitch
- Coke production
- Engine exhaust etc

Clinical features

- Acute poisoning is rare
- Chronic exposure through inhalation or dermal route can predispose to skin or lung cancer.
- Increased skin, bladder, lung. GIT cancers have been described in PAH exposed workers.
- Eye irritation, photosensitivity, skin erythema, cough, bronchitis, hematuria
- Respiratory: Cough, bronchitis
- Mouth: Leukoplakia
- Dermal: coal tar warts, erythema burns, acne
- Mild hepatotoxicity and renal toxicity
- Hematuria
- Routine monitoring required even in the absence of symptoms.

Halogenated hydrocarbons

- Clear, colourless, non inflammable, s
- sweetish, chloroform like odour.

Table 27.2: Halogenated Hydrocarbons

<i>Compound</i>	<i>Use</i>
Acetylene tetrabromide (Tetrabromoethane)	Gauge fluid, solvent, refractive index liquid in microscopy
Carbon tetrachloride* (Tetrachloromethane)	Manufacture of fluorocarbon propellants (Freon), solvent, cleansing and degreasing agent, grain fumigant, dry cleaning, fire extinguisher
Chloroform	Anaesthetic agent
Dichloroethylene (1,2-Dichloroethane)	Degreaser, solvent, fumigant, manufacture of nylon, rayon, etc
Ethylene dibromide (1,2-Dibromoethane)	Soil fumigant
Ethylene dichloride (1,1-Dichloroethane)	Cleansing and degreasing agent, solvent, grain fumigant
Fluorocarbon, Freon	Solvent for cleaning electronic equipment, degreaser, refrigerant, fire extinguisher, dry cleaning
Methyl bromide	Fire extinguisher, fumigant insecticide, refrigerant
Methyl chloride	Refrigerant (now obsolete)
Methylene chloride (Dichloromethane)	Solvent, paint remover, degreaser, manufacture of aerosol propellants and urethane foam
Propylene dichloride (Dichloropropane)	Degreaser, dry cleaning, stain remover, manufacture of cellulose plastics
Tetrachloroethane	Feed stock, cleanser, degreaser
Tetrachloroethylene	Solvent, dry cleaning, pesticide, metal cleaner
Trichloroethane	Solvent, degreaser, pesticide
Trichloroethylene	Solvent, degreaser, refrigerant, typewriter cleaning fluid, paint remover, adhesive, anaesthetic

*Banned from most commercial uses in Western countries

Toxicokinetics

- Usual route of exposure is through inhalation or ingestion. Can be absorbed through dermal routes too but slowly
- Distributed mainly in blood, brain and adipose tissue
- Metabolised in liver by CYP 450 oxidation and partly by glutathione conjugation
- Excreted majorly in urine and partially in bile.

Mode of Action

- Powerful hepatorenal toxins.
- CCl₄ : Hepatic mixed function oxidase metabolises it to CCl₃. trichloro methyl radical
- This initiates lipid peroxidation, protein lipid cross links, adduct with DNA, protein and lipids.
- Poison Cyt, P450 either released or converted by reduction to chloroform. HCl and CO maybe formed.
- React with oxygen to form peroxy free radical which may react to form phosgene which is hepatotoxic
- Recent studies focus Calcium homeostasis, it disrupts ATP dependant Ca⁺⁺ pumps.
- Methyl bromide and others act like alkylating agents and disrupt SH-enzymes.
- Hexokinase and pyruvate oxidase inactivated by methylation of SH group in CNS.

Clinical features:

Acute poisoning:

- Vomiting, diarrhea, abd. pain, headache, lethargy, vertigo, stupor
- Headache, fatigue, confusion, altered mental status, delirium, amnesia, incoherent speech, ataxia, positive Romberg sign may occur.
- Methyl bromide: psychosis like symptoms
- Liver damage- hepatitis, jaundice & hepatic encephalopathy
- Renal damage- oliguria, anuria, hematuria & renal failure
- Acidosis, hypertension, convulsions, respiratory failure. Hypotension, VF, slow pulse may occur
- Methyl bromide: characterised by myoclonic convulsions and permanent brain damage.
- Alcohol along with halogenated hydrocarbons especially carbon tetrachloride worsens the symptoms
- Dermal exposure : second degree burns, erythema, blisters, dermatitis etc

Chronic poisoning:

- Degreaser's flush
- Painter's syndrome
- Renal and liver cancer
- Extra pyramidal syndrome
- Myasthenic reaction

Fatal Dose

4-5ml for most compounds

20 – 25ml for a few

Diagnosis

- Characteristic odour in breath
- Fehling's test
- Isonitrile test: foul skunk-like odour because of phenyl isonitrile
- Gas chromatography
- CCl₄- 2-5mmg/ dl are toxic
- Methyl bromide- sr. inorganic bromide 5mg/100ml toxic
- Hepatic and renal toxicity tests (HPLC)
- Chest radiograph
- Abdominal radiograph for CCl₄

Treatment

Decontamination:

- Dermal and ocular decontamination
- Potentially lethal ingestions- Gastric lavage
- Administer oxygen if the mental status and respiratory failure
- Treat arrhythmia, aspirational pneumonia, hepatorenal failure
- CCL4 induced cirrhosis occurs because of the failure to detoxify bile acids. Rat studies show administration of cholestyramine reduced cirrhosis and hyperbaric O2 reduced elevated SGPT & improved survival
- NAC within 8 to 10 hrs. Max. efficacy within 16 hours.
- Loading dose- 140mg/kg orally as 5% solution in cola followed by maintenance dose of 70mg/kg q4hrs 17doses.
- **Prescott protocol: Gastric lavage followed by i.v infusion of NAC at 150mg/kg over 5mins, then 50mg/kg over 4hrs, followed by 100mg/kg for 16hrs.**

- IV NAC maybe used in methyl bromide poisoning as well.
- Treat renal failure with dialysis and hepatic failure with FFP, vitamin K, low protein diet, lactulose & neomycin.
- Hemodialysis is not recommended in the absence of liver and renal failure
- treat the dermal burns (methyl bromide).

KEROSENE POISONING – CASE PRESENTATION

SUBJECTIVE

- Name: Ms. XY
- Age: 56 years
- Admitted to emergency dpt. With a h/o ingestion of kerosene (~40ml) with suicidal intent, 6h before ingestion.
- Past medical history: Type 2 DM x 5 years
- Past medication history: Metformin, Glyburide.
- Family history: Nil
- Allergies: Nil

OBJECTIVE

- O/E partially conscious, smell of kerosene perceived
- RR: 30/min (tachypnic)
- Basal Crepitations
- Other Vitals: normal
- Initial ABG analysis, hemogram, Sr. Electrolytes were normal.
- Chest radiograph: Bilateral lower zone consolidation with right sided mild pleural effusion

ASSESSMENT

CHEMICAL PNEUMONITIS

PLAN

DAY	ON EXAMINATION	THERAPY
1	RR- 30/MIN CHEST X- RAY Bilateral lower zone consolidation Right sided mild pleural effusion	EMPERICAL ANTIBIOTICS Ceftriaxone Azithromycin Metrogyl Steroids, Iv fluids.
2	Dyspnea RR-50/min Increased Crepitations ABG- Po2 : 52 mmHg Pco2: 32 mmHg CHEST XRAY Increased consolidation n right side with moderate pleural effusion.	Oxygen inhalation 6L/min with 100% oxygen mask to maintain oxygen saturation. Continue antibiotics and steroids.

The treatment was continued for 2 weeks , she showed a gradual response to treatment and was discharged after 2 weeks.

ON FOLLOW UP AFTER 2 WEEKS:

On follow up visit after 2 weeks she had complaints of dyspnoea and fresh chest X-ray showed right-sided moderate hydropneumothorax .She was readmitted and intercostal drainage (ICD) tube was put.

DAY	ON EXAMINATION	THERAPY
I	<p>Pleural fluid was purulent and its analysis revealed total leucocyte count of 1840 cells/cumm, differential count—polymorphs 75% and lymphocyte 25%, protein—5.36 g/dl (serum protein—7.32), sugar—60 mg/dl, adenosine deaminase level 75 IU/l, [>36 IU/L] and Gram-positive cocci.</p> <p><i>Staphylococcus aureus</i> grew on culture that was sensitive to piperacillin-tazobactam and amikacin.</p>	<p>Patient was put on antibiotics according to sensitivity pattern</p>

even after 2 weeks of specific therapy there was no significant improvement. Repeat X-ray showed persistent hydropneumothorax, although there was no drainage from ICD tube]. Computed tomography (CT) of chest showed right-sided pyopneumothorax with septations, thickening of overlying pleura, and underlying collapsed consolidated lung with ICD *in situ*. Small cavitary lesions were also seen on left side with peripheral consolidation. Considering the findings of CT scan, decortication was done after which she improved.

INTERVENTIONS

- CULTURE SENSITIVITY NOT DONE AFTER EMPIRICAL TREATMENT WITH ANTIBIOTIC- LEADS TO ESCALATION OF ANTIBIOTICS
- BLOOD GLUCOSE LEVELS- IMPORTANT TO BE MONITORED AS HIGH DOSE STEROIDS CAUSE SIGNIFICANT HYPERGLYCEMIA AND PATIENT HAS TYPE 2 DM.

HYDROCARBONS MCQS

I. A 3yr old is playing in the garage while her father is working on the car. Soon, she starts vomiting and gagging, and smells of gasoline. A few hrs later she develops a cough, subcostal retractions and tachypnea. Select appropriate management.

- A. Insert a NG tube and start activated charcoal
- B. Arterial blood gas and continuous pulse oximetry
- C. Bronchoscopy
- D. Insert an NG tube and perform lavage.

2. A 15-year-old male with autism is brought to the emergency department by emergency medical services (EMS) after his parents found him with a bottle of kerosene in his hand. Parents report multiple episodes of vomiting. EMS reports the patient has been tachypneic and hypoxic in route to the hospital. Upon arrival, vitals are heart rate 160 beats/min, respirations 35, pulse oximetry 93% on 15 L of oxygen, afebrile, and normotensive. Diffuse wheezes are present on the lung exam. The patient appears dyspneic and anxious. He is started on BiPaP with only mild improvement of oxygenation. The heart rate improves. A chest x-ray and labs are obtained. The chest x-ray shows bilateral lower lobe infiltrates consistent with chemical pneumonitis. An ECG shows mild sinus tachycardia. What is the best next step in management?

- A. Start ceftriaxone for suspected pneumonia
- B. Add nebulized albuterol
- C. Obtain a urine drug screen
- D. Start on esmolol for suspected arrhythmia

3. Which chemical when ingested causes a strong predisposition to calcium oxalate kidney stones?

- A. Ethanol
- B. Methanol
- C. Ethylene glycol
- D. Isopropyl glycol

4. Which of the following substances and their antidotes do not match?

- A. Midazolam- flumazenil
- B. Cyanide- thiosulfate
- C. Isoniazid- pyridoxine
- D. Ethylene glycol- methanol

5. A patient who has ingested ethylene glycol will have what type of electrolyte imbalance?

- A. Anion gap metabolic alkalosis
- B. Non-anion gap metabolic alkalosis
- C. Anion gap metabolic acidosis
- D. Non-anion gap metabolic acidosis

6. A 4-year-old child drinks a sweet liquid from a water bottle he finds in his family's garage. Several hours later his mental status becomes altered and his parents bring him to the emergency room. Given this scenario, which is the least appropriate treatment for this case of poisoning?

- A. Hemodialysis
- B. Sodium bicarbonate
- C. Activated charcoal followed by gastric lavage
- D. Fomepizole

7. An obtunded adult male is brought to the emergency department by emergency medical services (EMS). He appears to be in his twenties, is disheveled, and has what appears to be paint around his nose and mouth. Hydrocarbon toxicity is suspected. What is the best initial step in management?

- A. Obtain chest x ray
- B. Obtain ECG c
- C. to check for an arrhythmia
- D. Secure the airway
- E. CT scan without contact

8. A 3-year-old male presents to the emergency department unresponsive. He is obtunded on physical exam. His babysitter admitted that the patient had been playing in the garage earlier and his shirt was covered in a liquid. Lab results reveal severe metabolic acidosis and oxalate crystals in the urine. What is the most likely toxin involved?

- A. Insecticide
- B. Gasoline
- C. Antifreeze
- D. Paint thinner

9. Which one of the following is not a type of hydrocarbon?

- A. Ketones
- B. Carboxylic acid
- C. Waxes
- D. Ethylene glycol

10. Acetylcysteine is given as an antidote for which of the following :

- A. Carbon tetrachloride
- B. Ethylene glycol
- C. Paraffins
- D. Carboxylic acids

ANSWERS

1. B
2. B
3. C
4. D
5. C
6. C
7. C
8. C
9. D
10. A