Clinical toxicology

Chemical and Environmental toxicity

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Environmental toxicity

It is a multidisciplinary field of science concerned with the study of the harmful effects of various chemical, biological and physical agents on living organisms.

Harmful effects of such chemical and biological agents as toxicants include pollutants, insecticides, hydrocarbons, pesticides, fertilizer, antiseptic and disinfectant camphore and moth repellents.

Poisoning by Hydrocarbons

Exposure to hydrocarbons is common in modern society. Hydrocarbons are easily accessible in products such as gasoline, turpentine, furniture polish, household cleansers, propellants, kerosene, and other fuels. Although hydrocarbons include all compounds composed predominantly of carbon and hydrogen, the compounds of interest are derived from petroleum and wood. Most of the dangerous hydrocarbons are derived from petroleum distillates and include aliphatic (straight-chain) hydrocarbons and aromatic (benzene-containing) hydrocarbons. Other hydrocarbons such as pine oil and turpentine are derived from wood.

Mechanism of toxicity

Hydrocarbons may **cause direct injury to the lung** after pulmonary aspiration or systemic intoxication after ingestion, inhalation, or skin absorption. Many hydrocarbons are also irritating to the eyes and skin.

Mechanism of toxicity

A. Pulmonary aspiration (few milliliters). Chemical pneumonitis is caused by direct tissue damage and disruption of surfactant. Aspiration risk is greatest for hydrocarbons with low viscosity and low surface tension (eg, petroleum naphtha, gasoline, turpentine).

B. Ingestion (about 20-25 ml)

1. Aliphatic hydrocarbons and simple petroleum distillates such as lighter fluid, kerosene, furniture polish, and gasoline are poorly absorbed from the GI tract and do not pose a significant risk for systemic toxicity after ingestion as long as they are not aspirated.

2. In contrast, many aromatic and halogenated hydrocarbons, alcohols, ethers, ketones, and other substituted or complex hydrocarbons are capable of causing serious systemic toxicity, such as coma, seizures, and cardiac arrhythmias.

C. Inhalation

Inhalation of hydrocarbon vapors in an enclosed space may cause intoxication as a result of systemic absorption or displacement of oxygen from the atmosphere; in addition, sensitization of the myocardium to catecholamines can cause cardiac dysrhythmias.

D. Injection

Injection of hydrocarbons into skin (less than 1ml), subcutaneous tissue, or muscle may cause a severe local inflammatory reaction and liquefaction necrosis.

E. Skin and eye contact

They can cause local irritation. Dermal absorption can be significant for some agents but is insignificant for most of the simple aliphatic compounds.

Clinical presentation

A. Pulmonary aspiration usually causes immediate onset of coughing or choking.

This may progress to a chemical pneumonitis characterized by respiratory distress, including tachypnea and hypoxia. Death may ensue from respiratory failure, secondary bacterial infection.

B. Ingestion often causes abrupt nausea and vomiting, occasionally with hemorrhagic gastroenteritis.

C. Systemic toxicity caused by hydrocarbon ingestion, inhalation, intravenous injection, or dermal absorption ,often includes confusion, ataxia, lethargy, and headache. With significant exposure, syncope, coma, and respiratory arrest may occur. Cardiac arrhythmias (AF and VF) may occur as a result of myocardial sensitization, especially with halogenated and aromatic compounds. Many agents also may cause hepatic and renal injury.

D. Injection of hydrocarbons can cause local tissue inflammation, pain, and necrosis.

E. Skin or eye contact may cause local irritation, burns, or corneal injury. Chronic skin exposure often causes a defatting dermatitis (resulting from removal of oils from the skin). Some agents are absorbed through the skin and can produce systemic effects.

Diagnosis

1. From history of exposure and smelling the patients mouths and skin or site of injection
2. Presence of respiratory symptoms such as coughing, tachypnea, and wheezing.
3. Signs and symptoms.
4. Take ECG.

Treatment

1. Emergency and supportive measures.
2. Maintain an open airway and assist ventilation if necessary . Administer supplemental oxygen.
3. Use epinephrine and other beta-adrenergic medications with caution in patients with significant hydrocarbon intoxication because arrhythmias may be induced.
4. Using salbutamol (beta 2 stimulant) to treat bronchospasm and hypoxia.
5. Use steroids or prophylactic antibiotics if necessary.

- **B. Specific drugs and antidotes**
- 1. There is no specific antidote for aspiration pneumonitis; corticosteroids are of no proven value.
- 2. Specific drugs or antidotes may be available for systemic toxicity of some hydrocarbons (eg, acetylcysteine for carbon tetrachloride and methylene blue for methemoglobin formers) or their solutes (eg, chelation therapy for leaded gasoline and antidotes for pesticides).

Decontamination

- 1. Inhalation. Move the victim to fresh air and administer oxygen if available.
- 2. Skin and eyes. Remove contaminated clothing and wash exposed skin with water and soap. Irrigate exposed eyes with copious water or saline and perform fluorescein examination for corneal injury.
- 3. Ingestion toxicity. Gut decontamination is neither necessary nor desirable because it increases the risk for aspiration. For systemic toxins, consider aspiration of the liquid via nasogastric tube and administration of activated charcoal. Take precautions to prevent pulmonary aspiration if the patient is obtunded.

ANTISEPTICS AND DISINFECTANTS

- **Antiseptics** are applied to living tissue to kill or prevent the growth of microorganisms.
- **Disinfectants** are applied to inanimate objects to destroy pathogenic microorganisms.

Examples

- Phenol, cresol, chlorohexyleol (dettol); Oxidizig agent: Hydrogen peroxide, Iodine, chlorine, Chlorohexidine (mouth wash) ; glutaraldehyde, hexylresorcinol (Strepsils losenges) ;Cetrimide; Ethanol and isopropanol; potassium permanganate.

Mechanism of toxicity and clinical presentation

Antiseptics	Mechanism of toxicity	Clinical presentation
Dettol (Chlorohexyleol, pine oil and isopropy alcohol).	It is derivative of phenol denatures protein, disrupts the cell wall, and produces a coagulative tissue necrosis. It may cause corrosive injury to the eyes, skin, and respiratory tract. Systemic absorption may result in cardiac arrhythmias and CNS stimulation.	respiratory tract irritation, coughing and chemical pneumonia. vomiting and diarrhea. Systemic absorption may cause agitation, confusion, seizures, coma, hypotension, arrhythmias, and respiratory arrest
Chlorohexidine (mouth wash)	Ingestion of higher concentrations and/ or large volume (>100 ml) have caused esophageal damage and hepatic injury.	bitter aftertaste, Nausea and vomiting and stomach pain. Rarely headache, euphoria, giddiness and blurred vision
Glutaraldehyde (used to disinfect medical equipment, as a tissue preservative, and topically as an antifungal and is found in some x-ray solutions) .	Irritating to the skin and respiratory tract. Rhinitis, epithelial changes, mild atrophy of olfactory mucosa	Nasal discharge, mouth breathing. Allergic contact dermatitis. Glutaraldehyde has been widely implicated as the cause of colitis and diarrhea following endoscopy.

Mechanism of toxicity and clinical presentation

Antiseptics	Mechanism of toxicity	Clinical presentation
Hydrogen peroxide	Generation of oxygen gas in closed body cavities can potentially cause mechanical distension that results in gastric or intestinal perforation, as well as venous or arterial gas embolization	Gastric distension
Potassium permanganate	Concentrated solutions (> 1:5000) are corrosive to skin, mucous membranes, and oropharyngeal, esophageal, or gastric injury may occur. due to the release of potassium hydroxide.	Glottic edema and methemoglobinemia

Diagnosis

Diagnosis is based on a history of exposure and the presence of mild GI upset or frank corrosive injury. Solutions of potassium permanganate are dark purple, and skin and mucous membranes are often stained brown-black.

Other useful laboratory studies include electrolytes, glucose, methemoglobin level (for potassium permanganate exposure), and upright chest radiography (for suspected gastric perforation).

Treatment of poisoning by Antiseptic and disinfectant:

1. Emergency and supportive measures
2. In patients who have ingested concentrated solutions, monitor the airway for swelling and intubate if necessary.
3. Consult a gastroenterologist for possible endoscopy after ingestions of corrosive agents such as concentrated hydrogen peroxide and potassium permanganate.

Most ingestions are benign, and mild irritation is self-limited.

4. Consider hyperbaric oxygen treatment for gas emboli associated with concentrated peroxide ingestion.

5.No specific antidotes are available for irritant or corrosive effects. But, if methemoglobinemia occurs, administer methylene blue

6. In case of ingestion of concentrated corrosive agents:

a. Dilute immediately with water or milk.

b. Do not induce vomiting because of the risk for corrosive injury. Perform gastric lavage cautiously.

c. Activated charcoal and cathartics are probably not effective and interfere with endoscopy procedure.

7.Eyes and skin contamination can be treated first by remove contaminated clothing and then doing irrigation with large amount of tepid water.

Camphor and moth repellents

- Camphor is one of several essential oils (volatile oils) derived from natural plant products that have been used for centuries as topical rubefacients for analgesic and antipruritic purposes. Camphor and other essential oils are found in overthecounter remedies such as Vicks VapoRub. In addition, camphor is used for religious, spiritual, aromatic, folk medicinal, and insecticidal purposes, often in powder or cube form. Toxic effects have occurred primarily when essential oils have been largely administered orally for therapeutic effects and in accidental pediatric ingestions.

Mechanism of toxicity:

After topical application, essential oils produce dermal hyperemia followed by a feeling of comfort, but if ingested, they can cause systemic toxicity. Most essential oils cause CNS stimulation or depression. Camphor is a CNS stimulant that causes seizures soon after ingestion. The underlying mechanism is unknown; however, a transient decrease in hyperpolarization-activated conductance has been noted in human poisoning.

Clinical sings and symptoms:

A. Oral. Manifestations of acute oral overdose usually occur within 5–30 minutes after ingestion. Burning in the mouth and throat occurs immediately, followed by nausea and vomiting. Camphor typically causes abrupt onset of seizures within 20–30 minutes after ingestion. Ataxia, drowsiness, dizziness, confusion, restlessness, delirium, muscle twitching, and coma may occur. Aspiration may result in pneumonitis. Death may result from CNS depression and respiratory arrest or may be secondary to status epilepticus. Myocarditis, granulomatous hepatitis, and death following chronic camphor intoxication are reported.

B. Dermal exposure may result in allergic reactions. Prolonged skin contact may result in a burn.

Clove oil

- **Clove oil** Contains 80–90% eugenol.
- ***Mechanism of toxicity***: Metabolic acidosis, CNS depression, seizures, coagulopathy, and hepatotoxicity after acute ingestion. Fulminant hepatic failure has been seen in a 15-month-old boy after a 10-mL ingestion. Smoking clove cigarettes may cause irritant tracheobronchitis, hemoptysis.
- ***Treatment***: N-Acetylcysteine may be beneficial in preventing or treating the hepatotoxicity.

Peppermint oil:

- Contains 50% menthol. Oral mucosal irritation, burning, and rarely mouth ulcers reported. Intravenous injection resulted in coma, cyanosis, pulmonary edema, and acute respiratory distress syndrome. Allergic contact dermatitis with dermal exposure. Nasal instillation in 2-month-old resulted in dyspnea, stridor, hyperextension, coma, and metabolic acidosis.

Diagnosis:

Diagnosis usually is based on a history of exposure. The pungent odor of camphor and other volatile oils is usually apparent.

Other useful laboratory studies include electrolytes, glucose, liver aminotransferases,

and arterial blood gases (if the patient is comatose or in status epilepticus).

Treatment

1. Emergency and supportive measures

2. Maintain an open airway and assist ventilation if necessary.

3. Treat seizures (Diazepam) and coma (Cocktil coma) if they occur. N-acetylcysteine may be effective for preventing hepatic injury after pennyroyal and clove oil ingestion.

4. Decontamination by administer activated charcoal orally if conditions are appropriate

5. Enhanced elimination. The volumes of distribution of camphor and other essential oils are extremely large, and it is unlikely that any enhanced removal procedure will remove significant amounts of camphor.

Questions & Discussion