Exhibit D44

Hydrocarbon Poisoning

Introduction

- Hydrocarbons are diverse group of **organic substances** that contain **carbon** and **hydrogen**. Toxic exposure to hydrocarbons primarily impacts the respiratory and central nervous system.
- Hydrocarbons are found in substances such as ***glues, nail polishes, paints, paint removers, pine oil, gasoline, kerosene, furniture polishes, lamp oil, lighter fluid.***
- Hydrocarbons are often mixed with agents, such as camphor, aniline dyes, heavy metals, and pesticides that have systemic toxicity.

Epidemiology:

- **1-2%** of non-pharmaceutical exposures in children <6 years old reported to US poison control centers.
- In young children the ingestion typically occurs as a result of **exploratory** behavior. Ingestion of large quantities of hydrocarbons alone by children is unusual because hydrocarbons have a bad taste.
- Toxicity in adolescents often arises from intentional behaviors such as inhalant abuse.
- Moderate to major toxic effects are associated most commonly with ingestion of lamp oil, kerosene, lighter fluid, and or naptha.
- Most commonly use ingested substances include gasoline, chlorofluorocarbon propellants, motor oils, lighter fluid/naptha, lamp oil, and mineral spirits.

Classification:

Hydrocarbon toxicity can be classified according to potential for toxicity:

- **Low toxicity** (unless complicated by gross aspiration)
  - Examples include asphalt, tars, mineral oil, petroleum jelly, motor oil, and axle grease.
- **Aspiration hazard:**
  - Examples include kerosene, furniture polish, charcoal lighter fluid, mineral spirits, or outdoor torch and cigarette lighter fluids clinical effects are typically limited to direct pulmonary damage and subsequent inflammation.
  - Determinants of pulmonary aspiration is determined by 3 properties; **volatility, surface tension, viscosity.** Hydrocarbons with decreased viscosity, low surface tension and high volatility are more likely to be aspirated and cause pulmonary injury.
- **Systemic toxicity:**
  - Systemic toxicity occurs after ingestion of compounds including halogenated and aromatic hydrocarbons (e.g. in substances like glues, solvents, nail polishes) or after ingestion of compounds that combine aliphatic hydrocarbons with toxic additives (e.g. organophosphates, heavy metals, camphor).
  - The extent of acute systemic toxicity is determined by their **degree of absorption and volatility** from the GI tract and/or respiratory system.
  - Systemic effects most commonly include **cardiac arrhythmias secondary to myocardial sensitization and CNS depression, hepatic and acute renal tubular necrosis** has also been described. Systemic absorption is not a major contributor to pulmonary injury except in the setting of massive ingestion.

Pathophysiology/ Mechanism of Toxicity:

Primarily impacts the ***respiratory*** and ***central nervous system.*** Respiratory system is primarily affected by direct injury. Low viscosity, low surface tension and solvent properties of aspirated hydrocarbons together determine a compound's ability to cause chemical pneumonitis. The main pathologic finding is **severe necrotizing pneumonia.** Other findings include direct destruction of the airway epithelium, pulmonary capillaries, and alveolar septae, as well as solubilization of the lipid surfactant layer. Secondary changes include atelectasis, interstitial inflammation, and hyaline membrane formation. The **inflammatory response** from chemical irritation generally causes temperature elevation, usually within hours of exposure.

Volatile hydrocarbons are highly **lipid** soluble. They enter the circulation through the lungs and rapidly diffuse throughout the body and into the CNS. **Neurons,** which have a high lipid content, are particularly susceptible to severe pulmonary injury and hypoxia.

Differential diagnosis:

Includes bronchopneumonia, salicylate overdose, and other toxins. The following clinical manifestations and ancillary studies help differentiate between the different diagnoses.

Clinical Manifestations:

- Typically asymptomatic ***initially,*** with history of hydrocarbon exposure
- Those symptomatic at presentation (e.g. hypoxemia, respiratory distress) typically progress to respiratory failure **rapidly.**
- If history of hydrocarbon exposure is lacking, diagnosis is based on clinical features. There are less rapidly available studies to confirm hydrocarbon exposure by detection of urinary metabolites or direct measurements of blood levels.
- Characteristic **odors** can help identify the type of hydrocarbon ingested. Pine oil has a pine scent. Halogenated hydrocarbons have a sweet solvent odor. Kerosene and other aliphatic hydrocarbons have a petroleum distillate odor.

Physical findings after hydrocarbon exposure:

SYSTEM	MANIFESTATIONS
Vital Signs	**Fever** (38 to 40 degrees Celsius), persistence >48 hours suggests bacterial superinfection, decreased **oxygen** saturation on pulse oximetry.
Pulmonary	Generally signs and symptoms occur within 30 minutes, although onset might be **delayed for 12 to 24 hours.** Immediate signs of aspiration include coughing, choking, vomiting and gagging. Other signs depend on degree of pulmonary injury and include tachypnea, wheezing, and dullness on percussion. Major complications include asphyxia, necrotizing chemical pneumonitis, hemorrhagic pulmonary edema, lipoid pneumonia that quickly progresses to shock and respiratory arrest. ***Secondary*** bacterial or viral infection may exacerbate the chemical pneumonitis.
Central Nervous System	Ingested or inhalation causes **rapid effects** including somnolence, dizziness, weakness, ataxia, fatigue, lethargy, seizures, and coma, depending on the amount of hydrocarbons exposure. Hypoxia secondary to hydrocarbon aspiration may have detrimental CNS effects including drowsiness, tremors or seizures.
Cardiovascular	Cardiac **dysrhythmias** and **dysfunction** (particularly after exposure to solvent hydrocarbons) because the myocardium is sensitized to endogenous and exogenous catecholamine's.
Hematologic	**Leukocytosis** can occur early after exposure and last up to 1 week. Hemolysis, hemoglobinuria and consumptive coagulopathy rarely occurs after large ingestions.
Gastrointestinal	Ingestion can cause **direct local injury** (eg. edema and mucosal ulceration) to the upper GI system (eg. esophagus, stomach, etc.)which may causes nausea and hematemesis. Halogenated hydrocarbons may cause hepatic and/or renal tubular necrosis 1-2 days after ingestion.

Ancillary studies:

- Children who show signs of pulmonary aspiration should receive a **chest radiograph** within 4-6 hours exposure.
- Evidence of hydrocarbon aspiration is evident within 2 hours of exposure in 88% and in 98% by 12 hours. Radiographic abnormality es usually **peak between 2-8 hours** after aspiration.
- Initial findings on radiograph consist of multiple, small patchy densities with ill -defined margins. As the injury progresses, the lesions become larger and coalesce.

- Radiograph findings typically lag behind clinical improvement, which occurs 3-5 days after aspiration.
- **Symptomatic patients** should also receive the following laboratory tests: complete blood count, blood gas (typically have a mild respiratory alkalosis with hypoxemia), serum electrolytes, serum glucose, urinalysis.

Management:

It is most important to recognize pulmonary toxicity and rapidly initiate appropriate supportive care.

- **Stabilization**: depends on the degree of symptoms including respiratory distress and altered mental status. Asymptomatic patients should be observed with serial examination and be placed on **NPO** status. Patients in respiratory distress should receive **oxygen**, serial chest **radiographs** and **beta-2 agonists** (for treatment of bronchospasms). If their respiratory status continues to decline, they require endotracheal **intubation**. Patients with seizures should receive IV benzodiazepines.
- **External decontamination:** remove all contaminated clothing, clean affected hair and skin to reduce risk of additional irritation and inhalation. Dermal and ocular exposure can be treated with copious water irrigation.
- GI decontamination: Gastric lavage, ipecac administration and activated charcoal is **NOT** recommended because of increased risk of vomiting and additional pulmonary aspiration.
- Avoid administration of exogenous catecholamine's, prophylactic antibiotic or corticosteroids.

Poison control is an excellent resource for management and information for providers and families alike. CLICK ON THE LINK BELOW for more information.

Prognosis:

Depends on amount of hydrocarbon ingested or aspirated, the type of hydrocarbon exposure and the adequacy of medical care. The typical course lasts for **2-5 days on average**. ***Most*** children survive without serious complications, but some may progress to respiratory failure and death.

Prevention:

- Poisoning in young children typically results from an exploratory occurrence that can be prevented through safe packaging and storage.
- Since 2001, US consumer product safety commission has required child-resistant packaging for products that have low viscosity and contain greater than 10 percent hydrocarbon by weight.

References

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