Camphor and Mothballs

- Camphor has been used medicinally for centuries, most commonly as a rubefacient.

- Accidental ingestion of 20 %camphorated oil, mistaken for castor oil, has caused numerous cases of toxicity.

- Although camphorated oil is no longer available and the efficacy of camphor itself is not widely accepted, camphor remains a common ingredient in many over-the-counter preparations.

- 20 grams of Vicks VapoRub, 10 mL of Campho-Phenique, and 16 mL of Vicks VapoSteam each contain about 1 g of camphor

- Clinically significant camphor toxicity has not been reported with ingestions of less than 30 mg/kg .
- uncommon when less than 50 mg/kg of camphor is ingested.
- the human lethal dose of camphor is reported to range from 50 to 500 mg/kg.
- In adults, 10 mL of 20 % camphorated oil (2 g) has been reported to cause symptoms,
- In 19-month-old child 5 mL (1 g) resulted in the death

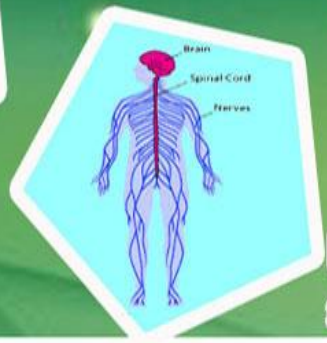
Ayurvedic Health Benefits of Camphor Oil

Relieves severe pain, spasms and inflammation.

Combats microbes and skin infections.

Alleviates cold and other respiratory problems.

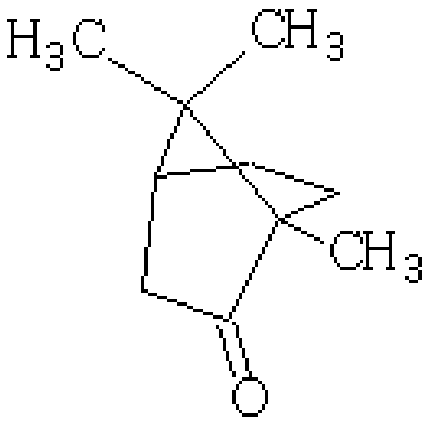
Supports digestive functions.



Pacifies the nervous system.

Toxicokinetics/Pathophysiology

- Camphor is a cyclic terpene with a characteristic, penetrating odor and a pungent, aromatic taste.



(1*S*,4*S*)-1,7,7-trimethylbicyclo[2.2.1]heptan-2-one
Camphor

- Camphor is rapidly absorbed from the mucous membranes and the gastrointestinal tract.

- Its lipophilic nature is responsible for central nervous system (CNS) toxicity, which is believed to involve excitatory mechanisms

- It is also absorbed through inhalation, through dermal application, and by nasal instillation.

- It has been abused as an abortifacient orally and by intrauterine injection and readily crosses the placenta.

- Pulmonary elimination of camphor causes a distinctive odor on the breath that is helpful in diagnosis.

Clinical Presentation

- Symptoms of camphor toxicity usually begin 5 to 90 minutes after ingestion and are often abrupt in onset.
- Spontaneous emesis, with the odor of camphor readily apparent, typically occurs first.
- CNS stimulation ensues with restlessness, confusion, delirium, and increased muscular activity.
- Severe toxicity may include seizures, apnea, and coma.
- Death results from respiratory depression or status epilepticus

Clinical Presentation

- Chronic ingestion of camphor can cause a variety of symptoms clinically similar to Reye's syndrome.
- Gastrointestinal symptoms may include nausea, vomiting, epigastric pain, and hepatic enzymes elevation.
- Pathologic hepatic changes often include such findings as granulomatous hepatitis and fatty metamorphosis.
- With chronic dermal exposure, systemic effects and contact dermatitis can occur as well as significant allergic responses.
- Ocular exposure results primarily in irritation only, although oral intake has been associated with visual problems.

Treatment

- Activated charcoal should be administered for gastrointestinal decontamination
- Due to prominent CNS effects, the induction of emesis is contraindicated.
- If liquid camphor is ingested, a nasogastric tube can be used to aspirate gastric contents before instillation of activated charcoal.
- Alcohols and oil solutions should be avoided because they have been reported to enhance absorption of camphor.

Treatment

- Benzodiazepines such as lorazepam or diazepam are indicated for symptoms of CNS hyperactivity, such as agitation, tremors, and seizures.
- Phenobarbital can be used for recurrent or prolonged seizures.
- Thorough flushing of exposed eyes and skin is usually sufficient treatment to relieve transient irritation produced by camphor.

MOTHBALLS

- Naphthalene and paradichlorobenzene are sole ingredients in moth repellents in the form of balls, flakes, or crystals.
- Naphthalene can produce serious hematologic and CNS effects, necessitating treatment,


Naphthalene Ball
MADE IN CHINA


POWER
MOTH BALLS
KILLS MOTHS AND HOUSE INSECTS
Global
NEWS

MOTHBALLS

- Differentiating between these two white, crystalline solid compounds is important.

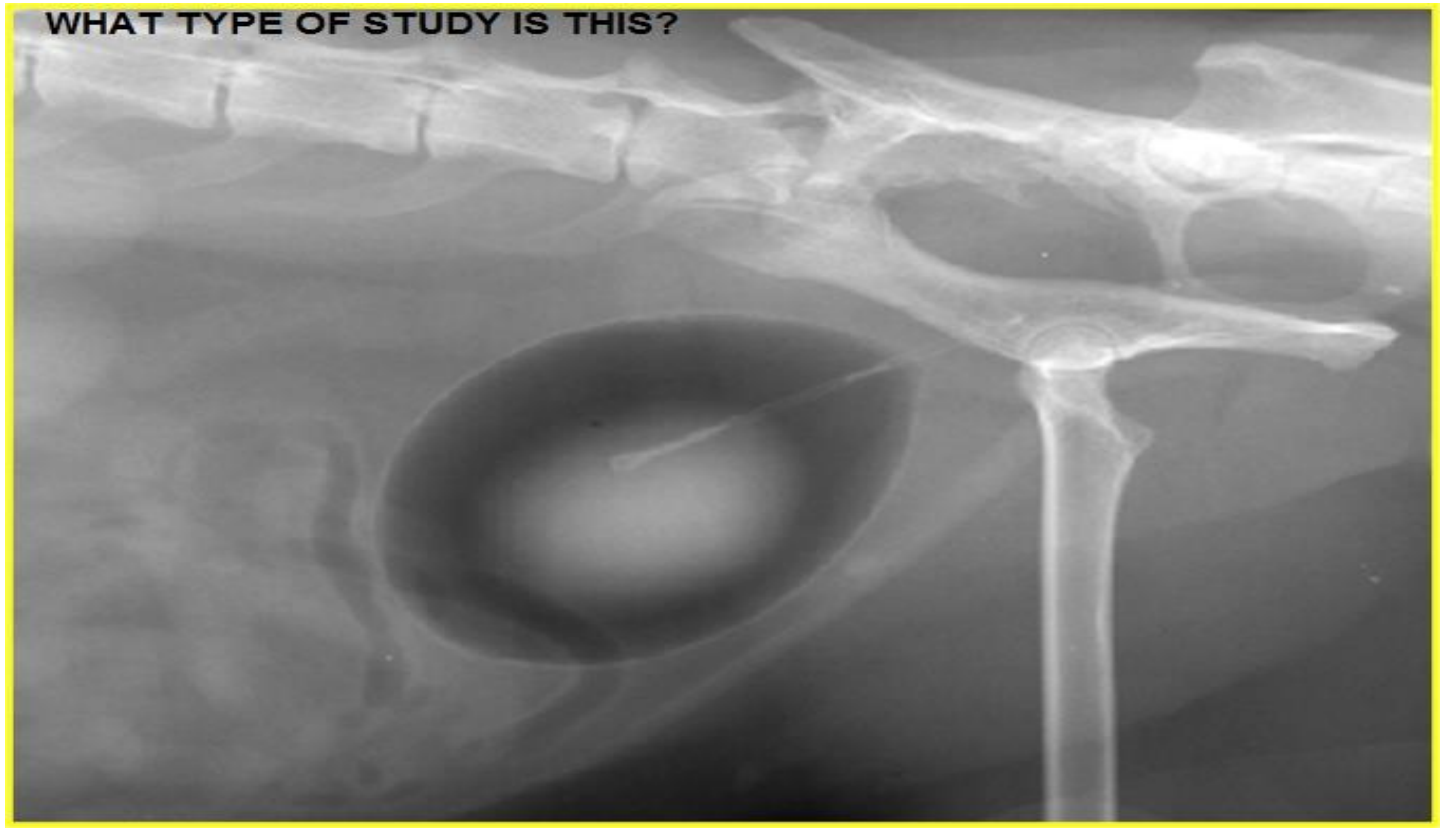
- Differentiation is possible through a variety of methods.

1. Naphthalene feels dry to the touch paradichlorobenzene feels moist and oily
2. A naphthalene will float, whereas the paradichlorobenzene-containing mothball will sink in a saturated salt solution (3 heaping tablespoonfuls of salt and 4 ounces of tepid water stirred vigorously until the salt will not dissolve further.

MOTHBALLS

3-On an abdominal radiograph, paradichlorobenzene is densely radiopaque whereas naphthalene-containing mothballs are radiolucent or faintly radiopaque

4-Odors are characteristic and can be compared with known samples.



Naphthalene

- Naphthalene is well absorbed after oral, dermal, and inhalational exposure. It is soluble in fats and oils, and the administration of milk or food after ingestion may aid absorption.

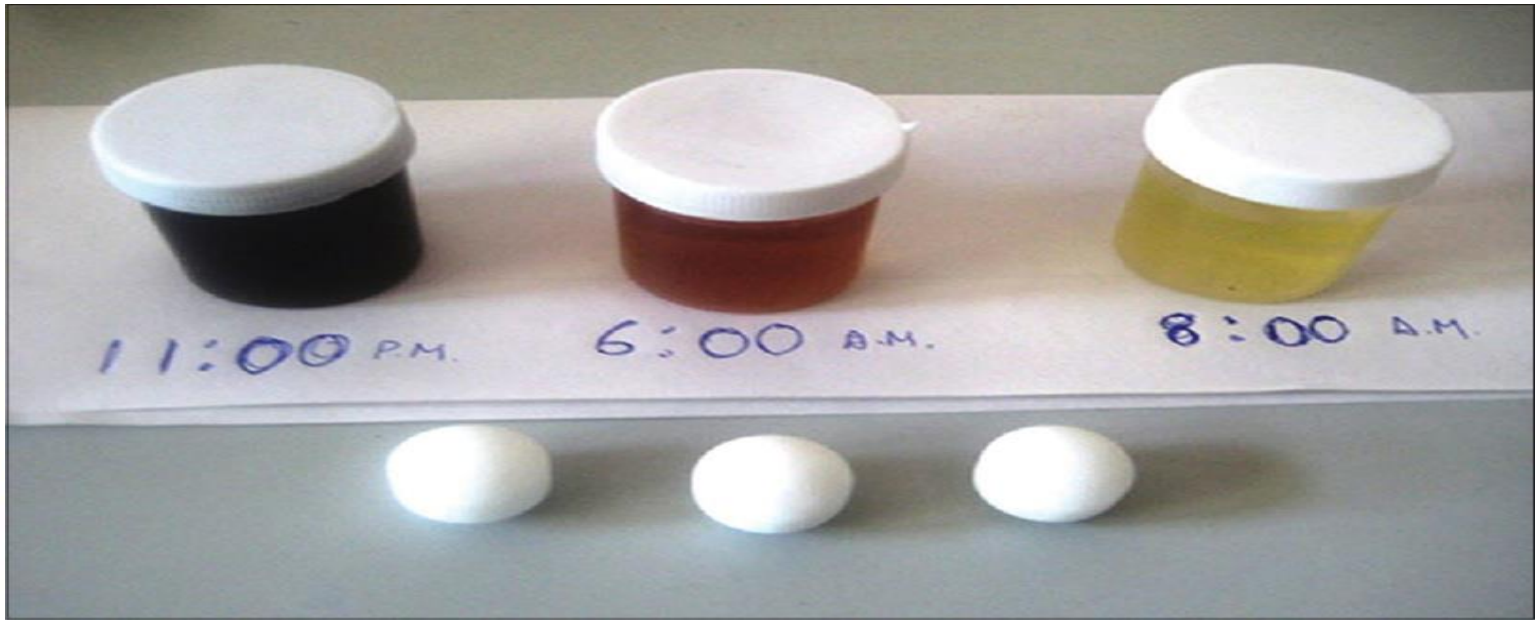
- Hepatic metabolism generates α-naphthol, β-naphthol, α-naphtholquinone, and β-naphtholquinone metabolites.

- The α-naphthol oxidative metabolite is responsible for the severe hemolytic activity, acute tubular necrosis, and methemoglobinemia.

- Less than one mothball containing 200 to 500 mg of naphthalene can cause hemolysis in patients with
- glucose-6-phosphate dehydrogenase (G6PD) deficiency.
- Infants due to inadequate conjugation of toxic metabolites.

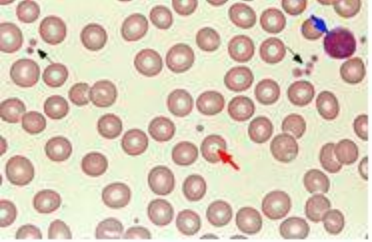
Clinical Presentation

- Nausea, vomiting, abdominal pain, diarrhea, and fever may develop as early as 1 day after exposure.
- Development of clinically recognized hemolysis may be delayed 1 to 5 days and is typically preceded by gastrointestinal symptoms.
- Signs of hemolysis, including pallor, tachypnea, weakness, jaundice, and dark urine, may also be present or evolve over the next several days.

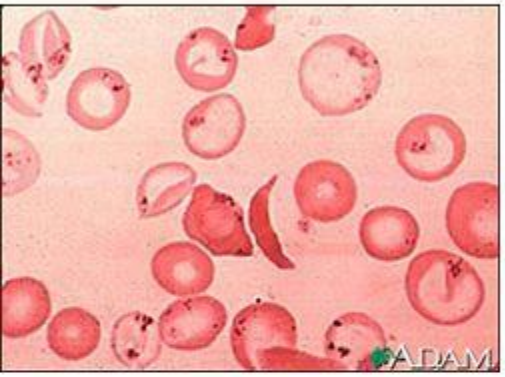


Clinical Presentation

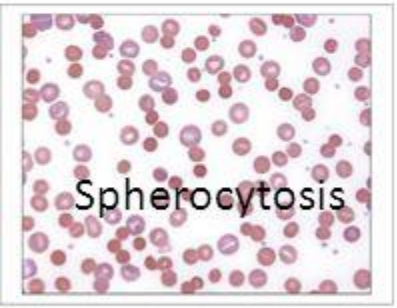
- Cyanosis may indicate methemoglobinemia.
- Lethargy and seizures are other possible effects.
- Hemoglobin frequently is decreased with an increase in red cell fragmentation.
- Anisocytosis and poikilocytosis with Heinz body formation may occur in severe cases.
- Chronic exposure has resulted in nausea, vomiting, fever, jaundice, lethargy, aplastic anemia, and hepatic centrilobular necrosis.



Normal

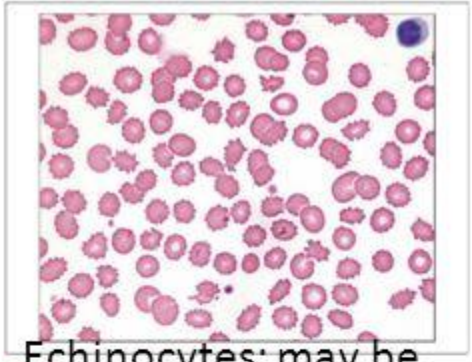


Anisocytosis (size)
Poikilocytosis (Shape)

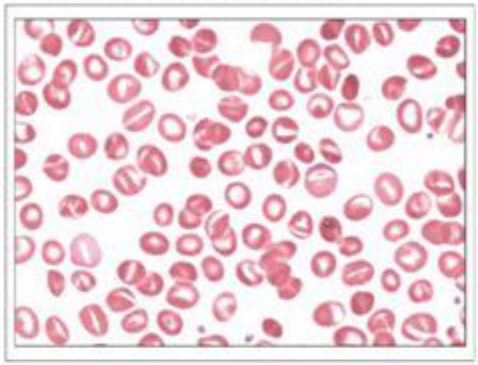


Spherocytosis

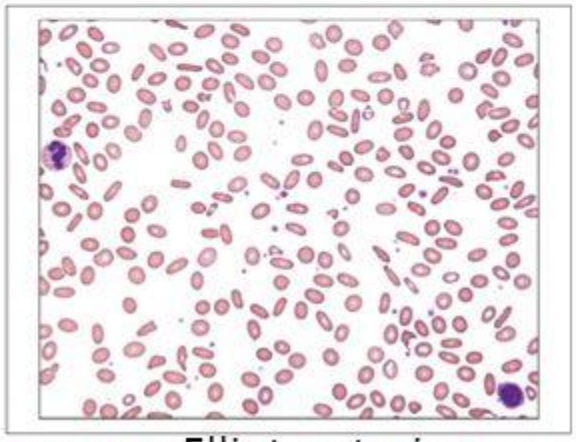
Teardrop



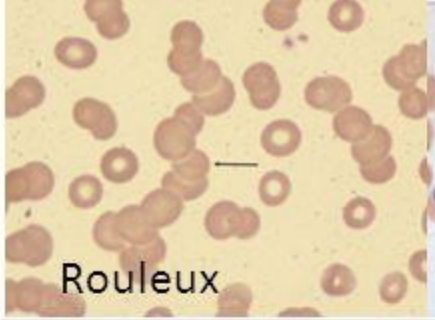
Echinocytes: may be artifact from storing



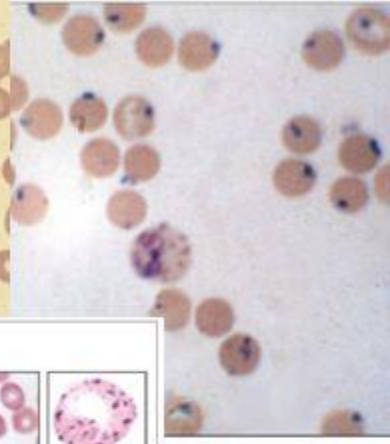
Stomacytosis



Elliptocytosis



Rouleux



Heinz body
Denatured hemoglobi

Basophilic Stippling: RNA

Pappenheimer body: Iron

Howell-Jolly: DNA

Treatment

- Treatment of naphthalene toxicity is primarily supportive.
- Treatment should be initiated for ingestions of one half or more of a naphthalene-containing mothball.
- In early therapy, ipecac syrup–induced emesis, in the first 2 hours after ingestion, is preferred to lavage, owing to the size of the mothballs.
- activated charcoal should also be administered.
- Milk or fatty meals that increase absorption should be avoided for several hours.
- Transfusion may be indicated in cases of severe hemolysis.
- Intravenous fluids to maintain urinary flow may prevent renal deposition of cell breakdown products.
- oxygen and methylene blue to patients symptomatic from methemoglobinemia

Treatment

- Exposed skin should be thoroughly washed with soap and water.
- Naphthalene is not easily removed from clothing, and contaminated clothing should be discarded to prevent further exposure.

Paradichlorobenzene

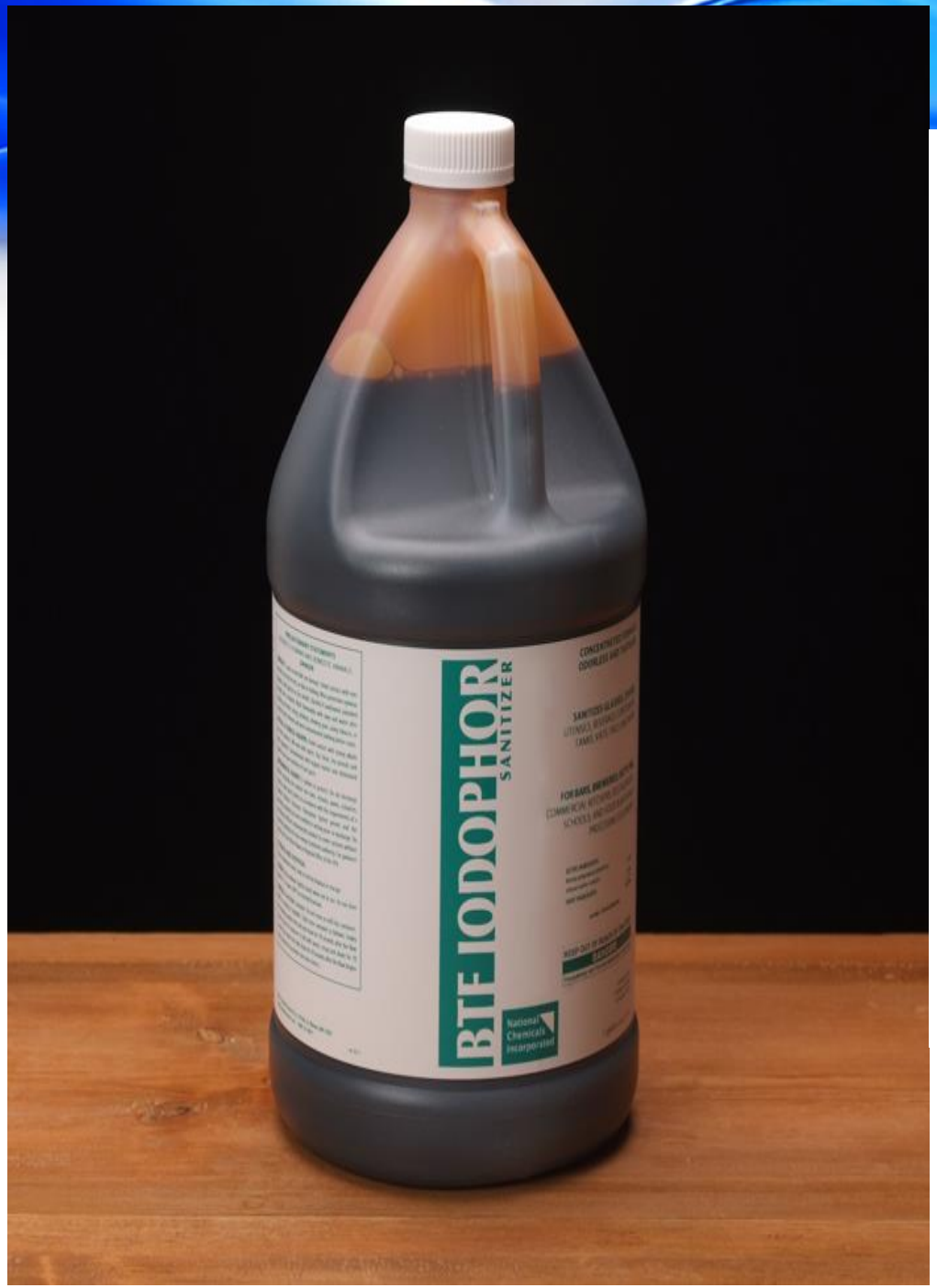
- The toxicity of mothballs containing paradichlorobenzene is low, with ingestion causing possible nausea and vomiting only.
- After chronic exposure, hepatotoxicity, anemia, and pulmonary granulomatosis have been reported.
- Emesis is indicated when large amounts have been ingested.
- Activated charcoal can also be administered.
- Milk and fatty foods should be withheld for several hours after ingestion to minimize absorption.
- If the composition of a mothball is unknown, gastric decontamination and treatment should proceed as outlined for naphthalene.

PARADICHLOROBENZENE

Antiseptics, Disinfectants, and Sterilizing Agents

- **Iodophors, Iodine, and Iodide**

1. iodide (I^-),
2. free iodine (I_2), and
3. iodine bound to povidone, a carrier molecule that limits the availability of free iodine.


BTF IODOPHOR
SANITIZER
National Chemicals Incorporated


NDC 0869-0088-10
SWAN
IODINE
TINCTURE U.S.P.
FIRST AID ANTISEPTIC
FOR EXTERNAL USE ONLY
CAUTION POISON
Centerland Swan
Smyrna, TN 37167 U.S.A.
1 FL OZ (30 mL)
Drug Facts
5310336

The iodophors are used as

- ✓ antiseptics and irrigants.
- ✓ Iodine-containing solutions are also used as expectorants
- ✓ as vaginal irrigants
- ✓ for contrast studies in radiology
- ✓ as therapy for acute hyperthyroidism. Lugol's iodine solution, commonly used in the acute treatment of hyperthyroidism, contains 5 % I_2 and 10 % potassium iodide (I^-).

- The antibacterial property of iodide is facilitated by a covalent linkage to bacterial cell surface glycoproteins, which enhances the bactericidal properties of polymorphonucleocytes

- The mechanism of iodine cellular toxicity has been attributed to interference with cellular oxidative metabolism and ATP production.
- Chronic exposure results in disturbances of thyroid structure and function and may be manifest as hyperthyroidism, hypothyroidism, goiter, or thyroiditis.
- These paradoxical responses are due to the stimulation of increased thyroid hormone production in some individuals (Jod basedow reaction)
- the blockade of thyroid hormone production in others (Wolff-Chaikoff effect).
- Iodine toxicity occurs through ingestion, through absorption during wound irrigation with iodophor-containing solutions, through mucosal exposure, and during topical application to dermal burns.

Clinical Presentation

- Acute ingestion of iodine (I_2)-containing solutions results in gastrointestinal irritation, gastrointestinal ulceration, and chemical pneumonitis .

- These effects are concentration dependent, so tincture of iodine or Lugol's iodine solution results in far more tissue injury than does ingestion of povidone-iodine.

- Any acute exposure may lead to acute tubular necrosis, hemolytic anemia, and hyperthyroidism.

- Early toxic symptoms are related to stimulation of increased secretions, leading to rhinorrhea, conjunctivitis, and cough productive of serous fluid.

Clinical Presentation

- Acute respiratory distress
- severe lactic acidosis
- renal dysfunction
- cardiogenic shock, and death occur within hours.

- Anaphylactic and anaphylactoid reactions also occur

- Chronic ingestion of iodine leads to thyroid disorders in some individuals.
-
- Large or chronic exposure to iodides results in sensitivity reactions manifest as painful salivary gland enlargement and a variety of dermal eruptions, so-called ioderma.

Treatment

- Good supportive care is critical in the management of all cases and is all that is required in most cases of povidone-iodine ingestion.
- After ingestion of significant amounts, gastric decontamination may be done by aspiration with a small nasogastric tube.
- Iodine may be converted to less toxic iodide by the administration of starch or milk.
- Sodium thiosulfate will also convert iodine to iodide.
- Activated charcoal is indicated unless significant symptoms suggest the need for endoscopic evaluation of caustic injury.
- Iodine is removed by hemodialysis.

Boric Acid

- Boric acid had significant medical uses in the past as a topical antiseptic and irrigant.
- At one time it was mixed with honey and glycerine and sold to treat topical mucosal sores in children.
- Because of its weak antiseptic properties and significant toxicity it is no longer used in medicinal preparations.
- A common cause of exposure today is related to accidental ingestion of powdered boric acid used to kill cockroaches.
- Suicidal ingestions of large amounts (280 g in one case) may be fatal.

Pathophysiology

- Boric acid is easily absorbed across

1. mucosal surfaces
2. abraded skin, and I
3. gastrointestinal tract.

- It is eliminated unchanged in the kidneys with a half-life of 21 hours in patients with normal renal function.

- Excretion is markedly prolonged in seriously poisoned patients.

Clinical Presentation

- The characteristic presentation of boric acid poisoning is the development of blue-green vomiting and diarrhea
- followed by intense erythroderma that has a boiled lobster appearance.
- This progresses to desquamation and erosion of mucosal surfaces.
- Renal, bone marrow, and hepatic injuries occur.
- CNS symptoms include seizures, delirium, and coma.

Treatment

- Treatment in most cases is supportive.
- Activated charcoal does not absorb boric acid effectively.
- Ipecac-induced emesis for 200 to 400 mg/kg,
- lavage and emergency department evaluation for more than 400 mg/kg. Lavage was recommended for a 12-g ingestion in larger patients.
- Hemodialysis may be indicated for severely poisoned patients.
- Exchange transfusions have been used in severely poisoned neonates.

Pathophysiology

- Formaldehyde is a protoplasmic poison, precipitating proteins and causing coagulation necrosis.
- It promotes allergic reactions by binding to and altering tissue proteins.
- Repeated exposure is believed to result in allergic contact dermatitis (type IV hypersensitivity reaction).
- Type I hypersensitivity reactions are thought to precipitate the bronchospastic disease associated with formaldehyde
- Formaldehyde is readily absorbed from the gut and lungs and rapidly metabolized through formate to carbon dioxide and water by the liver and erythrocytes.
- Formaldehyde is a naturally occurring product of tetrafolate reactions with endogenous tissue levels of 3 to 12 ng/g.

Clinical Presentation

- **Inhalational Exposure**

- Formaldehyde has an easily detectable odor at 0.5 ppm
- frankly irritating to upper airways and mucous membranes at 1 ppm.

- These unpleasant attributes serve as an early warning and tend to limit inhalational exposure.
- Severe respiratory irritation develops at higher concentrations, producing dyspnea at 10 ppm. Inhalation of 100 ppm may be life threatening.
- Formaldehyde is believed to be a potent sensitizer in immune-mediated bronchospasm.
- Chronic low-level exposure to up to 1.6 ppm produced fatigue, headache, and mucous membrane irritation but did not produce significant changes in pulmonary function tests.
- The incidence of dermal sensitization may be as high as 5 per cent in the general population.

- Chronic off-gassing of formaldehyde from urea-formaldehyde foam insulation (UFFI) has been suggested as the cause of a myriad of complaints such as headache, nausea, upper airway irritation, reactive airway disease, dermatitis, and insomnia.

Oral Exposure

- Ingestion of formaldehyde results in gastrointestinal injury with nausea, vomiting, diarrhea, abdominal pain, and lethargy.
- Serious ingestions may be complicated by hemorrhage, shock, coagulation necrosis of stomach and distal esophagus, severe metabolic acidosis, and death.
- Multiple factors contribute to the acidosis, including the metabolism of formaldehyde to formate, increased tissue lactate, and concomitant methanol poisoning.
- Methanol levels were elevated for over 13 hours in a 58-year-old man who ingested 4 ounces of formalin. This prolonged course was attributed to delayed absorption due to “fixation” of the gastric mucosa.
- Perforation and strictures of the gastrointestinal tract may limit recovery.
- Inadvertent parenteral exposure to 2 per cent formalin from incompletely purged hemodialysis machines has resulted in intravascular hemolysis.

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