Chapter 199: Hydrocarbons and Volatile Substances

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FIGURE 199-1.

INTRODUCTION

Hydrocarbons are a diverse group of organic compounds consisting primarily of carbon and hydrogen atoms. The two basic forms of hydrocarbons are aliphatic (straight- or branched-chain carbon arrangement) or aromatic (carbon arranged in a ring). Hydrocarbons are in many household and occupational products (Table 199-1). While all hydrocarbons can be toxic, aromatic and halogenated hydrocarbons are associated with the most severe systemic toxicity. Volatile agents are associated with the highest aspiration risk. Identification of the specific hydrocarbon or class can help anticipate specific potential toxicity and guide management.
# TABLE 199-1
Common Products That Contain Hydrocarbons

<table>
<thead>
<tr>
<th>Hydrocarbon (state at room temperature)</th>
<th>Commercial Use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aliphatic – linear structure, toxicity varies depending on volatility</strong></td>
<td></td>
</tr>
<tr>
<td>Gasoline (petrol) – liquid</td>
<td>Motor fuel</td>
</tr>
<tr>
<td>Kerosene (paraffin) – liquid</td>
<td>Stove and lamp fuel</td>
</tr>
<tr>
<td>Mineral seal oil – liquid</td>
<td>Furniture polish</td>
</tr>
<tr>
<td>Petroleum ether – liquid</td>
<td>Industrial solvent</td>
</tr>
<tr>
<td>Diesel fuel – liquid</td>
<td>Motor fuel</td>
</tr>
<tr>
<td>( n )-Hexane – liquid</td>
<td>Plastic cement, rubber cement</td>
</tr>
<tr>
<td>Methane, butane, propane, and ethane – gas</td>
<td>Fuel</td>
</tr>
<tr>
<td>Mineral spirits (white spirits) – liquid</td>
<td>Solvent, paint thinner</td>
</tr>
<tr>
<td>Turpentine – liquid</td>
<td>Solvent, paint thinner</td>
</tr>
<tr>
<td>Mineral oil (liquid paraffin) – liquid</td>
<td>Lubricant, laxative</td>
</tr>
<tr>
<td>Paraffin wax – solid</td>
<td>Industrial uses, candles</td>
</tr>
<tr>
<td>Petroleum jelly (petrolatum or soft paraffin) – solid</td>
<td>Skin lotion</td>
</tr>
<tr>
<td><strong>Aromatic – ring structure, high toxicity</strong></td>
<td></td>
</tr>
<tr>
<td>Benzene – liquid</td>
<td>Chemical intermediate, gasoline (small amount, 0.8% on average)</td>
</tr>
<tr>
<td>Toluene – liquid</td>
<td>Airplane glue, plastic cement, acrylic paint</td>
</tr>
<tr>
<td>Xylene – liquid</td>
<td>Solvent, cleaning agent, degreaser</td>
</tr>
<tr>
<td><strong>Halogenated – high toxicity</strong></td>
<td></td>
</tr>
</tbody>
</table>
Chain length and branching determine the phase of the hydrocarbon at room temperature. Short-chain aliphatic compounds (up to 4 carbons), such as methane, ethane, propane, and butane, are gases; intermediate-chain aliphatic compounds (5 to 19 carbons), such as solvents, lamp oil, lighter fluid, and gasoline, are liquid; and long-chain aliphatic compounds (>19 carbons), such as waxes, are solids. Liquid hydrocarbons account for most exposures seen in the ED.1

Most hydrocarbon exposures occur as liquid ingestions or inhalations and usually have a benign clinical course.1,2 Serious toxicity and deaths associated with hydrocarbon exposure are usually due to ingestions rather than inhalation. Symptoms and signs of pulmonary injury develop in up to 50% of the children who ingest hydrocarbons,3,4 and hydrocarbon aspiration can produce acute respiratory distress syndrome.5 Suicidal injection of gasoline or kerosene with severe multiorgan toxicity has been reported.6,7

Volatile substances, usually hydrocarbon solvents contained in household or commercial products, can be inhaled for their euphoric effects (Table 199-2).8 Abusers are typically teenagers and younger adults, especially those in lower socioeconomic groups.9 Inhalation occurs by three different methods: (1) in "huffing," the individual soaks a rag with the inhalant and then places it over the mouth and nose; (2) in "bagging," the individual puts the hydrocarbon in a bag (usually a plastic bag) and repeatedly inhales deeply from the bag; and (3) in "sniffing," the hydrocarbon is directly inhaled via the nostrils.10 In addition to causing deaths, abuse of volatile agents is associated with crimes such as homicide, sexual assault, and child abuse.11 The most commonly abused volatile hydrocarbons are paints, solvents, and gasoline.
### TABLE 199-2

**Commonly Abused Volatile Substances**

<table>
<thead>
<tr>
<th>Product</th>
<th>Volatile Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrylic spray paint</td>
<td>Toluene</td>
</tr>
<tr>
<td>Adhesives, glue</td>
<td>Toluene, trichloroethylene</td>
</tr>
<tr>
<td>Aerosol propellants</td>
<td>Propellants and butane</td>
</tr>
<tr>
<td>Cigarette lighter refills</td>
<td>Butane</td>
</tr>
<tr>
<td>Degreasing agents</td>
<td>Trichloroethylene</td>
</tr>
<tr>
<td>Dry cleaning agents</td>
<td>Tetrachloroethylene</td>
</tr>
<tr>
<td>Fire extinguishers</td>
<td>Bromochlorodifluoromethane</td>
</tr>
<tr>
<td>Inhalational anesthetics</td>
<td>Nitrous oxide, halothane</td>
</tr>
<tr>
<td>Lighter fluid</td>
<td>Naphtha</td>
</tr>
<tr>
<td>Motor fuel</td>
<td>Gasoline (petrol)</td>
</tr>
<tr>
<td>Nitrites (&quot;poppers&quot;)</td>
<td>Isobutyl nitrite, amyl nitrite</td>
</tr>
<tr>
<td>Paint stripper</td>
<td>Methylene chloride</td>
</tr>
<tr>
<td>Plastic modeling cement</td>
<td>Methyl ethyl ketone, toluene</td>
</tr>
<tr>
<td>Spot removers</td>
<td>Trichloroethylene, trichloroethane</td>
</tr>
<tr>
<td>Typewriter correction fluid</td>
<td>Trichloroethane, trichloroethylene</td>
</tr>
</tbody>
</table>

**PATHOPHYSIOLOGY**

The toxic potential of hydrocarbons depends on their physical characteristics (viscosity, surface tension, and volatility), chemical characteristics (aliphatic, aromatic, or halogenated), presence of toxic additives
(pesticides or heavy metals), routes of exposure, concentration, and dose. The physical characteristics contribute the most to aspiration risk.

Viscosity refers to the general "thickness" of a liquid; fluids with a lower viscosity flow more easily than ones with high viscosity. Viscosity is measured in Saybolt universal seconds (SUS); fluids such as gasoline, kerosene, mineral seal oil, and turpentine have low viscosity (<60 SUS), whereas diesel fuel, grease, mineral oil, paraffin wax, and petroleum jelly have high viscosity (>100 SUS). Surface tension refers to the property where liquid molecules tend to cohere to each other. Liquids with high surface tension in contact with a solid surface tend to ball up, creating the smallest surface area rather than spreading out. Volatility refers to the ability of the liquid or solid to vaporize and is inversely related to the boiling point; highly volatile liquids have a low boiling point. Ingestion of liquids with low viscosity and surface tension and high volatility increases the risk for aspiration because these substance can flow easily, spreading out widely on the oral mucosa, and vaporize at body temperature. Inhalation of aromatic hydrocarbons or halogenated hydrocarbons can result in systemic absorption and the potential for significant toxicity.

CLINICAL FEATURES

Ingestion or aspiration of hydrocarbons mainly impairs the pulmonary system, but depending on the specific compound, the central nervous, peripheral nervous, GI, cardiovascular, renal, hepatic, dermal, and/or hematologic systems may be affected (Table 199-3).
<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary</td>
<td>Tachypnea, grunting respirations, wheezing, retractions</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Ventricular dysrhythmias (may occur after exposure to halogenated hydrocarbons and aromatic hydrocarbons)</td>
</tr>
<tr>
<td>Central nervous</td>
<td>Slurred speech, ataxia, lethargy, coma</td>
</tr>
<tr>
<td>Peripheral nervous</td>
<td>Numbness and paresthesias in the extremities</td>
</tr>
<tr>
<td>GI and hepatic</td>
<td>Nausea, vomiting, abdominal pain, loss of appetite (mostly with halogenated hydrocarbons)</td>
</tr>
<tr>
<td>Renal and metabolic</td>
<td>Muscle weakness or paralysis secondary to hypokalemia in patients who abuse toluene</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Lethargy (anemia), shortness of breath (anemia), neurologic depression/syncope (carbon monoxide from methylene chloride), cyanosis (methemoglobinemia from amine-containing hydrocarbons)</td>
</tr>
<tr>
<td>Dermal</td>
<td>Local erythema, papules, vesicles, generalized scarlatiniform eruption, exfoliative dermatitis, &quot;huffer's rash,&quot; cellulitis</td>
</tr>
</tbody>
</table>

**PULMONARY TOXICITY**

Hydrocarbon aspiration causes chemical pneumonitis by direct toxicity to the pulmonary parenchyma and alteration of surfactant function. Destruction of alveolar and capillary membranes results in increased vascular permeability and edema. The clinical manifestations of pulmonary aspiration are usually apparent soon after exposure from irritation of the oral mucosa and tracheobronchial tree. Symptoms include coughing, choking, gasping, dyspnea, and burning of the mouth. Patients with these symptoms should be assumed to have aspirated.

Signs include tachypnea, grunting respirations, wheezing, or retractions depending on the severity of aspiration. An odor of the hydrocarbon may be noted on the patient's breath. Hyperthermia of ≥39°C
(≥102.2°F) is likely and may occur initially or 6 to 8 hours after exposure. The fever is usually an inflammatory response due to pneumonitis. Necrotizing pneumonitis and hemorrhagic pulmonary edema may develop within minutes to hours in patients with severe aspiration. In most fatalities, these complications occur rapidly. With less severe damage, symptoms usually subside within 2 to 5 days, except in the case of pneumatoceles and lipid pneumonias, the symptoms of which may persist for weeks to months.

Although in most patients with clinically significant aspiration chest radiographic results eventually are abnormal, the time course of radiographic changes varies, and correlation with physical examination findings may be poor. Changes may be seen as early as 30 minutes after aspiration, but the initial radiograph in a symptomatic patient may be deceptively clear. Conversely, an asymptomatic patient can still have abnormal chest radiographic findings later during the clinical course. Radiographic changes usually appear by 2 to 6 hours and are almost always present by 24 hours, if they are to occur (Figure 199-1). The most common radiologic finding is bilateral infiltrates at the bases with multilobar involvement more common than single-lobe involvement and right-sided involvement more common than left-sided involvement.\textsuperscript{14,15,16} Hydrocarbon-induced aspiration pneumonitis can lead to lung necrosis and the creation of a pneumatocele.\textsuperscript{17}

**FIGURE 199-1.**
Two chest radiographs of a child who aspirated lamp oil and developed aspiration pneumonitis. A. Day 1: intubated, left lower lobe infiltrates. B. Day 3: intubated, worsening perihilar and bibasilar patchy infiltrates and new right small pleural effusion.
CARDIAC TOXICITY

Life-threatening dysrhythmias, such as ventricular tachycardia and ventricular fibrillation, may occur with systemic absorption. Dysrhythmias occur most commonly after exposure to halogenated hydrocarbons and aromatic hydrocarbons. Exposure to short-chain aliphatic hydrocarbons occasionally causes dysrhythmias (ventricular fibrillation).10 The most worrisome acute complication found in solvent abusers is "sudden sniffing death syndrome" and occurs within minutes of exposure.8 The mechanism of toxicity is believed to be catecholamine sensitization of the heart by hydrocarbons (especially halogenated hydrocarbons), resulting in ventricular dysrhythmias.8,10,18 Other mechanisms for sudden death include simple asphyxia, respiratory depression, and vagal inhibition. Ventricular akinesia and polymorphic ventricular dysrhythmias have also been described after overdose of chloral hydrate (a halogenated aliphatic hydrocarbon).18

CNS TOXICITY

CNS effects, primarily depression of consciousness, result from: (1) a direct toxic response to the systemic absorption of the hydrocarbon, (2) an indirect result of severe hypoxia secondary to aspiration, (3) simple asphyxiation due to the displacement of oxygen by the volatile hydrocarbon, and/or (4) volatile substance abuse with a plastic bag that prevents adequate oxygenation. Systemic effects occur through GI absorption, the inhalation of highly volatile petroleum distillates, or direct dermal penetration.

Signs of neurologic toxicity include slurred speech, ataxia, lethargy, and coma.13 Although hydrocarbons are central neurologic depressants, they often have an initial excitatory effect manifested as hallucinations, tremor, agitation, and convulsions. Individuals who abuse volatile solvents or workers who experience long-term hydrocarbon exposure may present to the ED complaining of recurrent headaches, ataxia, emotional lability, cognitive impairment, or psychomotor impairment.

PERIPHERAL NERVOUS SYSTEM TOXICITY

Exposure to n-hexane, methyl n-butyl ketone, and other six-carbon aliphatic hydrocarbons is associated with the development of a characteristic peripheral polyneuropathy caused by demyelination and retrograde axonal degeneration.19 Onset of symptoms may be delayed for weeks, and toxicity is attributed to a metabolite, 2,5-hexanedione, produced by the cytochrome P-450–mediated biotransformation of the parent compounds. This neurotoxic metabolite is thought to inhibit glutaraldehyde-3-phosphate dehydrogenase, which supplies energy for axonal transport.

Clinically, the patient may complain of chronic numbness and paresthesias in the extremities. The key component in making the diagnosis is a history of exposure to solvents, usually through occupations and hobbies. The compound n-hexane is found in solvents used in the printing, shoemaking, textile, and furniture industries, as well as in gasoline, quick-drying glues, and rubber cement.20

GI AND HEPATIC TOXICITIES
Most hydrocarbons are GI irritants. Vomiting, which occurs in many patients with aliphatic hydrocarbon ingestions, increases the risk of pulmonary aspiration. Gastric perforation has been reported after accidental ingestion of chlorofluorocarbons.\(^{21}\)

Hepatic damage resulting from ingestion of halogenated hydrocarbons is well described.\(^{22,23}\) Chlorinated hydrocarbons, such as carbon tetrachloride, methylene chloride, trichloroethylene, and tetrachloroethylene, are especially hepatotoxic. For example, carbon tetrachloride causes centrilobular liver necrosis similar to acetaminophen toxicity. Free radical metabolites of these agents that cause lipid peroxidation are apparently responsible for hepatocellular destruction. The time course of hepatic dysfunction with acute exposures appears similar to that of acetaminophen hepatotoxicity—within 24 to 48 hours after ingestion.

Clinically, patients may come to the ED complaining of nausea, vomiting, abdominal pain, or loss of appetite. Depending on the severity, the physical examination may reveal a patient with jaundice, lethargy, and/or abdominal tenderness, especially in the right upper quadrant. Results of serum transaminase tests and other hepatic synthetic function tests may be abnormal within 24 hours after ingestion.

**RENA L AND METABOLIC TOXICITIES**

Solvent abuse and occupational exposure to hydrocarbons may result in renal dysfunction. Chlorinated hydrocarbons, such as chloroform, carbon tetrachloride, and trichloroethylene, are also nephrotoxic. Toluene, an aromatic hydrocarbon that is commonly abused, may cause renal tubular acidosis in patients who inhale toluene-containing substances.\(^{8,24}\) The mechanism of toluene-induced renal tubular acidosis is not clear. The typical metabolic profile of renal tubular acidosis is a normal anion gap hyperchloremic acidosis with hypokalemia and a urine pH of >5.5. The metabolites of toluene (hippuric acid and benzoic acid) can be the cause of an elevated anion gap metabolic acidosis.\(^{25}\)

Clinically, habitual toluene abusers may complain of muscle weakness caused by hypokalemia.\(^{26}\) The serum potassium level may be so low (<2 mEq/L) that severe weakness develops, occasionally resulting in muscle paralysis. Significant rhabdomyolysis may also result.\(^{27}\) **Toluene abuse should be considered in individuals (especially young patients) who come to the ED with symptoms similar to hypokalemic periodic paralysis.**\(^{24,26}\)

**HEMATOLOGIC TOXICITY**

Hydrocarbon-induced hemolysis rarely occurs after the acute ingestion of gasoline, kerosene, and tetrachloroethylene, and after inhalation of mineral spirits. Exposure to benzene (an aromatic hydrocarbon) is associated with an increased incidence of hematologic disorders, including aplastic anemia, acute myelogenous leukemia, and multiple myeloma.\(^{28}\) Naphthalene exposure is associated with hemolytic anemia. Delayed methemoglobinemia is associated with occupational exposure to hydrocarbons containing amine functional groups such as aniline (see chapter 207, "Dyshemoglobinemias").\(^{29}\) Delayed carboxyhemoglobinemia is associated with methylene chloride exposure due to its metabolism to carbon
monoxide, which takes a few hours. This is unlike ordinary carbon monoxide exposure from exogenous sources in which the maximum carboxyhemoglobin level occurs at the time of the exposure. Clinically, patients may come to the ED with malaise, headache, dyspnea, or cyanosis depending on the exposure and the severity of the toxicity.

DERMAL TOXICITY

Dermal toxicity from exposure to hydrocarbons is most often associated with the short-chain aliphatic, aromatic, and halogenated hydrocarbons. These agents act as primary irritants and as sensitizers. Occasionally, highly permeable hydrocarbons can penetrate the skin, resulting in systemic toxicity. Skin findings can range from local erythema, papules, and vesicles to a generalized scarlatiniform eruption and an exfoliative dermatitis (Table 199-3). A "huffer's rash" may be noted over the face of patients who habitually abuse the volatile hydrocarbons. Frostbite of the face may develop during the inhalational abuse of fluorinated agents. A defatting dermatitis, similar to chronic eczematoid dermatitis, may occur.

Cellulitis and sterile abscesses have been associated with the injection of hydrocarbons, and even a small amount of injected hydrocarbon can cause significant injury. Hydrocarbon-induced soft tissue necrosis has recently been seen in patients using "krokodil," desomorphine synthesized from codeine with the use of hydrocarbon solvents. Dermal exposure to heated high-viscosity, long-chain aliphatics, such as tar, asphalt, or bitumen, presents a particularly challenging problem because of their association with thermal burns, hyperthermia, and difficulty with decontamination. Tar burns are discussed in the chapter 217, "Chemical Burns."

DIAGNOSIS

Diagnosis of hydrocarbon toxicity incorporates the findings of the history, physical examination, bedside cardiac and pulmonary monitoring, laboratory tests, and chest radiography. Determine the specific hydrocarbon-containing product, because identification can help anticipate specific potential toxicity and guide management. Pulse oximetry is useful to evaluate oxygenation status, and arterial blood gas analysis can be used to assess ventilation and acid-base status. Cardiac rhythm monitoring and an ECG are indicated in symptomatic patients and patients who ingest halogenated hydrocarbons. A chest radiograph is indicated in a symptomatic patient after hydrocarbon aspiration.

There are no specific quantitative hydrocarbon tests in standard use when evaluating suspected hydrocarbon intoxication. A basic metabolic panel is indicated in patients with a history of toluene abuse or in whom electrolyte abnormalities and renal insufficiency are suspected. Obtain hepatic function studies, serum ammonia, and prothrombin time in patients who ingest or inhale halogenated hydrocarbons. A CBC is indicated if anemia, bleeding disorder, hemolysis, or leukemia is considered. Measure carboxyhemoglobin level in patients with exposure to methylene chloride; repeat measurements may be necessary. Determination of methemoglobin level is indicated in patients with exposure to hydrocarbons containing amine functional groups. Abdominal radiographs may show evidence of ingestion of chlorinated...
hydrocarbons such as carbon tetrachloride or chloroform because of the radiopaque nature of polyhalogenated substances.\textsuperscript{22}

An outpatient nerve conduction study and electromyography can be considered in patients who present with chronic numbness and paresthesias in the extremities and who have a history of \textit{n}-hexane exposure.

**TREATMENT**

Securing the airway and maintaining ventilation are the critical maneuvers in patients who present with respiratory depression and/or significant neurologic depression (\textbf{Table 199-4}). Swelling of the lips and tongue due to irritant effect or freeze injuries can complicate airway management. Administer \textit{oxygen} to correct hypoxia. Inhaled \textit{\textbeta}_2-agonists may also be useful, especially in the setting of bronchospasm, but their role in the treatment of hydrocarbon pneumonitis has not been studied. Positive end-expiratory pressure or continuous positive-pressure airway ventilation may sometimes be required to maintain oxygenation, but may increase the potential for further injury from barotrauma, such as the development of pneumatoceles or pneumothorax. In cases of severe pulmonary aspiration resulting in refractory hypoxemia, treatment with high-frequency jet ventilation or extracorporeal membrane oxygenation has proved successful according to case reports.\textsuperscript{34,35} Surfactant therapy has been used to treat acute lung injury from hydrocarbon aspiration.\textsuperscript{36,37,38}
| Airway and breathing | Secure airway.  
Antidotes: Administer oxygen for carboxyhemoglobinemia and methylene blue for methemoglobinemia.  
Provide supplemental oxygen.  
Administer inhaled β₂-agonists.  
Ventilatory support: Provide positive end-expiratory pressure or continuous positive airway pressure as needed to achieve adequate oxygenation. |
|---|---|
| Cardiac | Circulation: Administer IV crystalloid fluid for initial volume resuscitation of hypotensive patients.  
Do not use catecholamines in cases of halogenated hydrocarbon exposure.  
Consider propranolol, esmolol, or lidocaine for ventricular dysrhythmias induced by halogenated hydrocarbon exposure.  
Consult the poison control center, toxicologist, and other appropriate specialists as needed. |
| Decontamination | Dermal: Remove hydrocarbon-soaked clothes, decontaminate skin with soap and water, and decontaminate eyes with saline irrigation.  
GI: Not indicated. See discussion below. |
| Other | Laboratory tests: Order CBC, basic metabolic panel, liver function tests (serum transaminase, bilirubin, albumin levels), prothrombin time, partial thromboplastin time, carboxyhemoglobin level, methemoglobin level, and/or radiologic studies as indicated (see text).  
Correct electrolyte abnormalities.  
Do not give steroids  
Administer blood products as needed. |

Treat hypotension with aggressive fluid resuscitation. **Avoid administration of catecholamines such as dopamine, norepinephrine, and epinephrine.** Catecholamines may cause dysrhythmias, especially after exposure to halogenated hydrocarbons and aromatic hydrocarbons. Hydrocarbon-induced dysrhythmias are generally seen shortly after the exposure, especially with inhalational use. Continuous cardiac monitoring should be initiated, and an ECG should be obtained. For hydrocarbon-induced ventricular dysrhythmias, class IA (procainamide) or class III (amiodarone, bretylium, and sotalol) antiarrhythmics should be avoided because of the risk of QT-interval prolongation. Propranolol, esmolol, and lidocaine have been reported to treat these ventricular dysrhythmias successfully.¹⁰,¹⁸,³⁹
There is no benefit to gastric lavage because risks of aspiration far outweigh any theoretical benefits. Activated charcoal does not adsorb hydrocarbons well and poses a risk for vomiting and aspiration, so charcoal is not recommended either. In the rare case where the hydrocarbon was combined with a highly toxic substance, such as a pesticide, or is highly toxic itself, such as an aromatic or halogenated chemical, and ingestion has occurred in the last hour or less, consult with the poison center first, as even in that situation, benefit from gastric lavage is unproven.

There is no clear evidence that corticosteroids are helpful in hydrocarbon-induced pneumonitis. One meta-analysis advocating low-dose steroids for acute lung injury and respiratory distress has been criticized because the patients were not generally treated with current lung-protective ventilation strategies. Antibiotics are not indicated unless there is clinical suspicion of superimposed bacterial pneumonitis.

In dermal exposures, decontamination is preferably done at the scene or before entering the ED to avoid spreading fumes to patient treatment areas. The patient needs to be fully undressed to prevent ongoing contamination from hydrocarbon-soaked clothes. Make sure staff wear protective gloves and aprons to prevent secondary exposure, especially to organophosphate-containing mixtures. Dermal decontamination with soap and cold water, and eye decontamination with saline irrigation should be performed.

**DISPOSITION AND FOLLOW-UP**

Consult a medical toxicologist or regional poison control center for all symptomatic or asymptomatic exposures to aromatic hydrocarbons or hydrocarbons with toxic additives. In cases of inhalation or aspiration of nonhalogenated aliphatic hydrocarbons, asymptomatic patients may be discharged home after about 6 to 8 hours of observation with instructions to return if delayed symptoms develop.

Further observation or hospitalization is required for patients who are symptomatic after hydrocarbon exposure. For pediatric hydrocarbon ingestions, the presence of wheezing, altered consciousness, or tachypnea within 2 hours predicts the need for further treatment. Hospitalization is also recommended for those who ingest hydrocarbons capable of producing delayed complications (e.g., halogenated hydrocarbons causing hepatic toxicity) or hydrocarbons with toxic additives (organophosphates and organic metal compounds). Patients with suicidal intent or with complications of solvent abuse need behavioral health evaluation.

**REFERENCES**


[PubMed: 19091298]

[PubMed: 15687517]

[PubMed: 19650952]

[PubMed: 11696523]

[PubMed: 18392064]

[PubMed: 3561457]

[PubMed: 19347673]

[PubMed: 2222600]

[PubMed: 24106534]

[PubMed: 24650492]


[PubMed: 8020296]


[PubMed: 8699548]


[PubMed: 19421666]


[PubMed: 21624880]


[PubMed: 25285387]


[PubMed: 10784325]


[PubMed: 23418938]


[PubMed: 19325471]


[PubMed: 23957905]


[PubMed: 18344104]
USEFUL WEB RESOURCES

American Academy of Clinical Toxicology—http://www.clintox.org/index.cfm

American Association of Poison Control Centers—http://www.aapcc.org/DNN/

Asia Pacific Association of Medical Toxicology—http://www.asiatox.org/

European Association of Poisons Centres and Clinical Toxicologists—http://www.eapcct.org/


South Asian Clinical Toxicology Research Collaboration—http://www.sactrc.org/

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